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# MANUAL OF EQUINE LAMENESS

GARY M. BAXTER

WILEY-BLACKWELL

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DVD included featuring additional anatomical images and video clips demonstrating key procedures and examples of conditions in motion.

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# COMMON TERMINOLOGIES AND ABBREVIATIONS

| Terminology | Abbreviations |
|---|---|
| Distal or third phalanx | P3, coffin bone |
| Middle or second phalanx | P2 |
| Proximal or first phalanx | P1 |
| Distal interphalangeal joint | DIP joint or coffin joint |
| Proximal interphalangeal joint | PIP joint or pastern joint |
| Metacarpo/metatarsophalangeal joint | MCP/MTP joint or fetlock joint |
| Distal sesamoidean ligaments | DSL |
| Distal sesamoidean impar ligament | DSIL |
| Collateral suspensory ligaments of navicular bone | CSL |
| Collateral ligaments of coffin joint | CLs of DIP joint |
| Deep digital flexor tendon | DDFT or DDF tendon |
| Superficial digital flexor tendon | SDFT or SDF tendon |
| Metacarpus/metatarsus | MC/MT or MC3/MT3 or MCIII/MTIII, cannon bone |
| Second and fourth metacarpal/metatarsal bones | MC2 or MCII, MC4 or MCIV, MT2 or MTII, MT4 or MTIV; splint bones |
| Digital flexor tendon sheath | DFTS or digital sheath |
| Common digital extensor tendon | CDET |
| Long digital extensor tendon | LDET |
| Tarsometatarsal joint | TMT joint |
| Distal intertarsal joint | DIT joint |
| Proximal intertarsal joint | PIT joint |
| Tarsocrural joint | TC joint |
| Medial femorotibial joint | MFT joint |
| Lateral femorotibial joint | LFT joint |
| Femoropatellar joint | FP joint |
| Scapulohumeral joint | SHJ or shoulder joint |
| Sacroiliac joint | SI joint |
| Computed tomography | CT |
| Magnetic resonance imaging | MRI or MR |
| Ultrasonography | US |
| Osteochondrosis | OC/OCD |
| Osteochondritis dissecans | OCD |

| | |
|---|---|
| Subchondral cystic lesion | SCL |
| Angular limb deformity | ALD |
| Osteoarthritis | OA |
| Accessory ligament of deep digital flexor tendon | ALDDFT, ICL, or inferior check |
| Accessory ligament of superficial digital flexor tendon | ALSDFT, SCL, or superior check |
| Developmental orthopedic disease | DOD |
| Proximal suspensory desmitis | PSD |
| Suspensory ligament | SL |
| Nonsteroidal anti-inflammatory drug | NSAID |
| Hyaluronan or hyaluronic acid | HA |
| Polysulfated glycosaminoglycans | PSGAG, Adequan |
| Platelet-rich plasma | PRP |
| Interleukin receptor antagonist protein or conditioned serum | IRAP |
| Extracorporeal shockwave treatment | ESWT or shock wave |
| Intra-articular | IA |
| Dorsopalmar/plantar | DP |
| Mediolateral | ML |
| Triamcinolone | TA |
| Methyl prednisolone acetate | MPA or Depo-Medrol® |
| Dimethyl sulfoxide | DMSO |
| Diclofenac cream | Surpass® |
| Mesenchymal stem cell | MSC |
| Proximal sesamoid bone | PSB |

# MANUAL OF EQUINE LAMENESS

1

CHAPTER

# Functional Anatomy of the Equine Musculoskeletal System

## ANATOMIC NOMENCLATURE AND USAGE

Informative and logical names for parts of the horse's body, as well as positional and directional terms, have evolved through the efforts of nomenclature committees. *Nomina Anatomica Veterinaria* is the standard reference in veterinary science. Some older terminology is still widely used. For example, the following are acceptable synonyms: navicular bone for distal sesamoid bone, coffin joint for distal interphalangeal joint, pastern joint for proximal interphalangeal joint, and fetlock joint for metacarpophalangeal joint. It is useful to be familiar with the older terms because many times they are used interchangeably.

Figure 1.1 provides the appropriate directional terms for veterinary anatomy. With the exception of the eye, the terms anterior and posterior are not applicable to quadrupeds. Cranial and caudal apply to the limbs proximal to the antebrachiocarpal (radiocarpal) joint and the tarsocrural (tibiotarsal) joint. Distal to these joints, dorsal and palmar (on the forelimb) or plantar (on the hindlimb) are the correct terms. The term "solar" is used to designate structures on the palmar (plantar) surface of the distal phalanx and the ground surface of the hoof.

## THORACIC LIMB

### *Digit and Fetlock*

The foot and pastern comprise the equine digit, a region including the distal (third), middle (second), and proximal (first) phalanges and associated structures (Figure 1.2). The fetlock consists of the metacarpophalangeal (fetlock) joint and the structures surrounding it. The digits and fetlocks of the thoracic limb and the pelvic limbs are similar in most respects. The term "palmar" is used when referring to structures of the forelimb, whereas "plantar" is used when referring to the hindlimb.

#### Foot

The foot consists of the epidermal hoof and all it encloses: the connective tissue corium (dermis), digital cushion, distal phalanx (coffin bone), most of the cartilages of the distal phalanx, distal interphalangeal (coffin) joint, distal extremity of the middle phalanx (short pastern bone), distal sesamoid (navicular) bone, podotrochlear bursa (navicular bursa), several ligaments, tendons of insertion of the common digital extensor and deep digital flexor muscles, blood vessels, and nerves. Skin between the heels is also part of the foot.

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

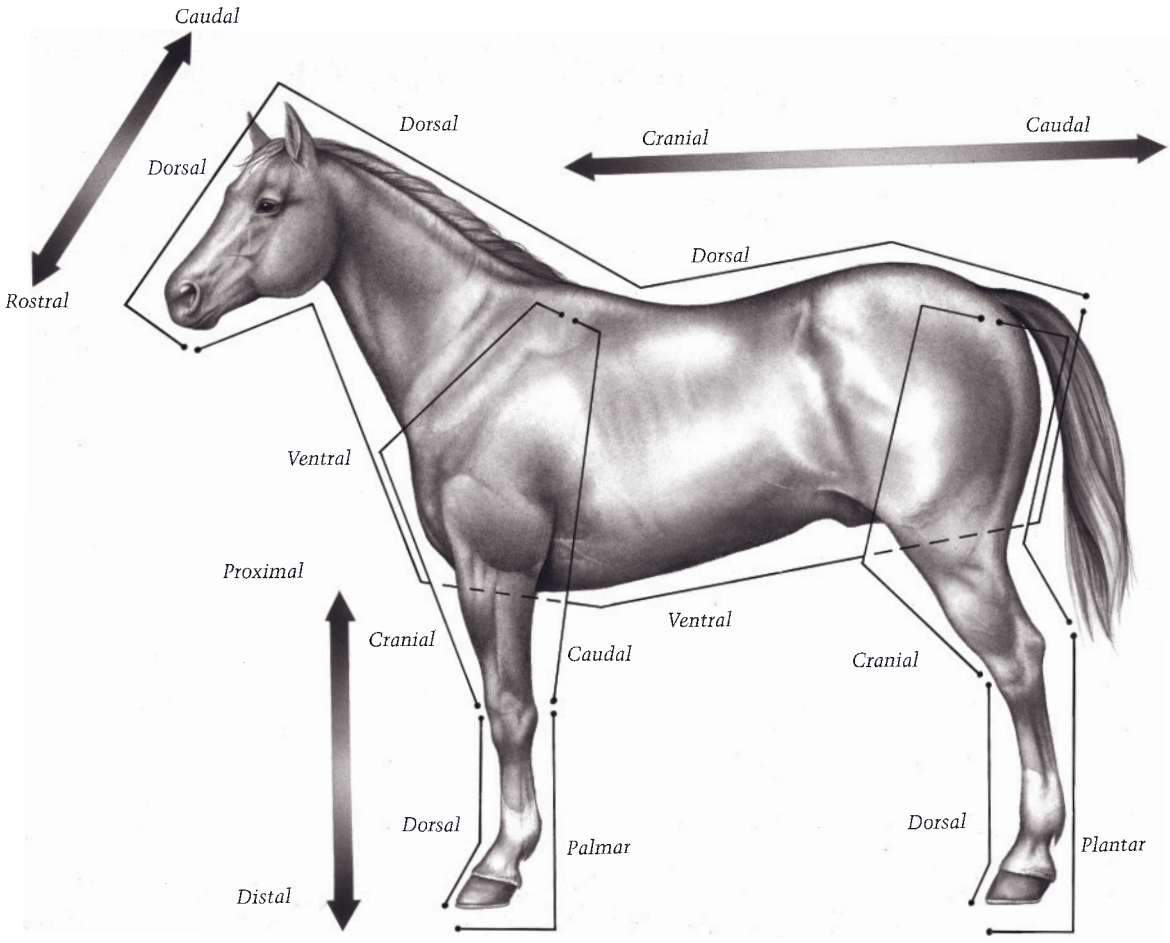


**Figure 1.1.** Positional and directional terms.

## Hoof Wall, Sole, and Frog

The hoof is continuous with the epidermis at the coronet. Here the dermis of the skin is continuous with the dermis (corium) deep to the hoof. Regions of the corium correspond to the parts of the hoof under which they are located: perioplic corium, coronary corium, laminar (lamellar) corium, corium of the frog, and corium of the sole. Examination of the ground surface of the hoof reveals the sole, frog, heels, bars, and ground surface of the wall (Figure 1.3). The ground surface of the forefoot is normally larger than that of the hind foot, reflecting the shape of the distal surface of the enclosed distal phalanx.

The hoof wall extends from the ground proximad to the coronary border where the soft white horn of the periople joins the epidermis of the skin at the coronet. The regions of the wall are the toe, medial and lateral quarters, and heels (Figures 1.3, 1.4). From the thick toe, the wall becomes progressively thinner and more elastic toward the heels, where it thickens again where it reflects dorsad as the bars. Ranges for the angle of the toe between the dorsal surface of the hoof wall and the ground surface of the hoof vary widely. In the ideal digit, the dorsal surface of the hoof wall and the dorsal surface of the pastern should be parallel, reflecting the axial alignment of the phalanges.

The highly vascular and densely innervated collagenous connective tissue of the coronary corium (dermis) gives rise to elongated, distally directed papillae. Laminar (lamellar) corium

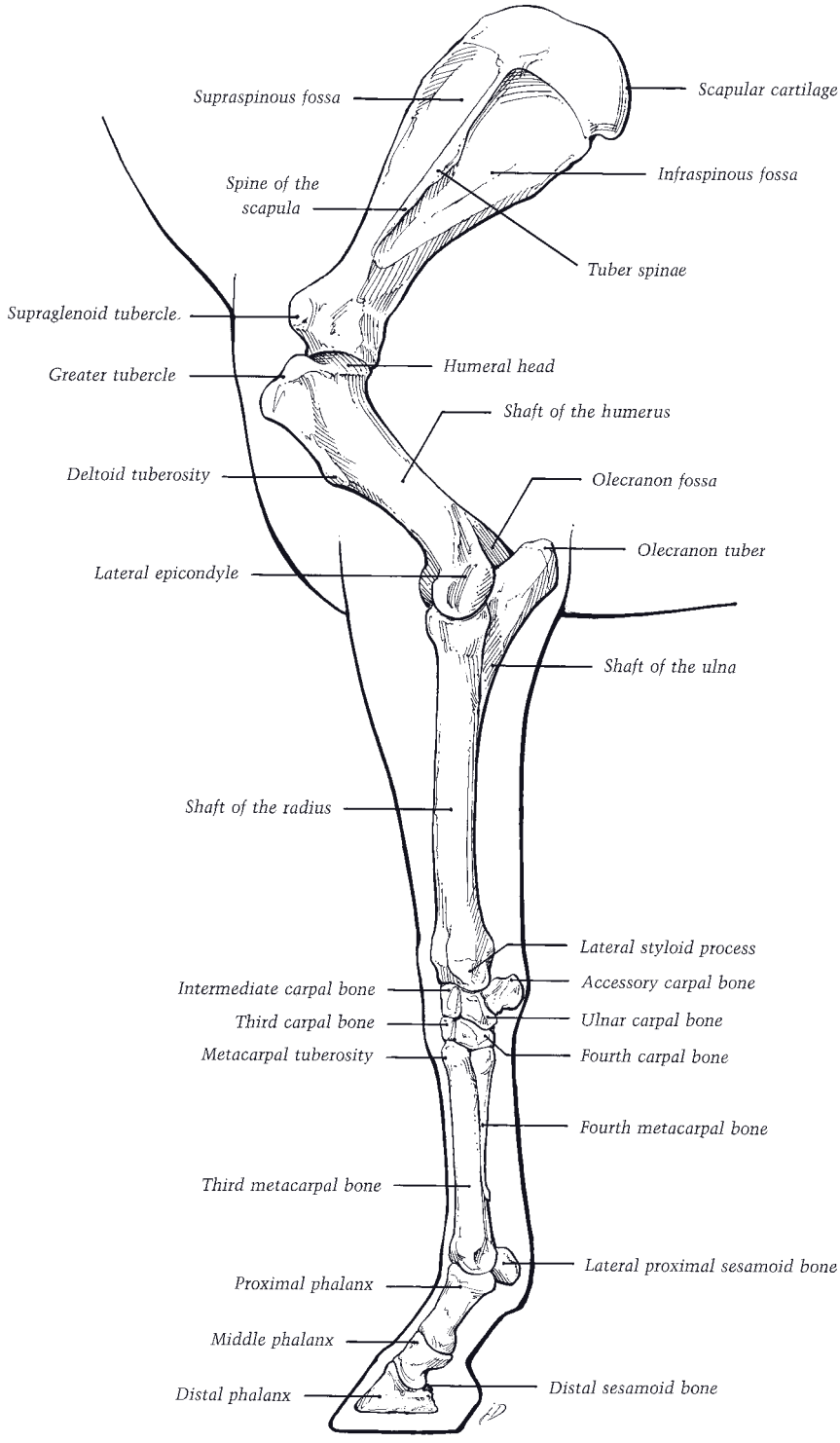


**Figure 1.2.** Bones of the left equine thoracic limb (lateral view).

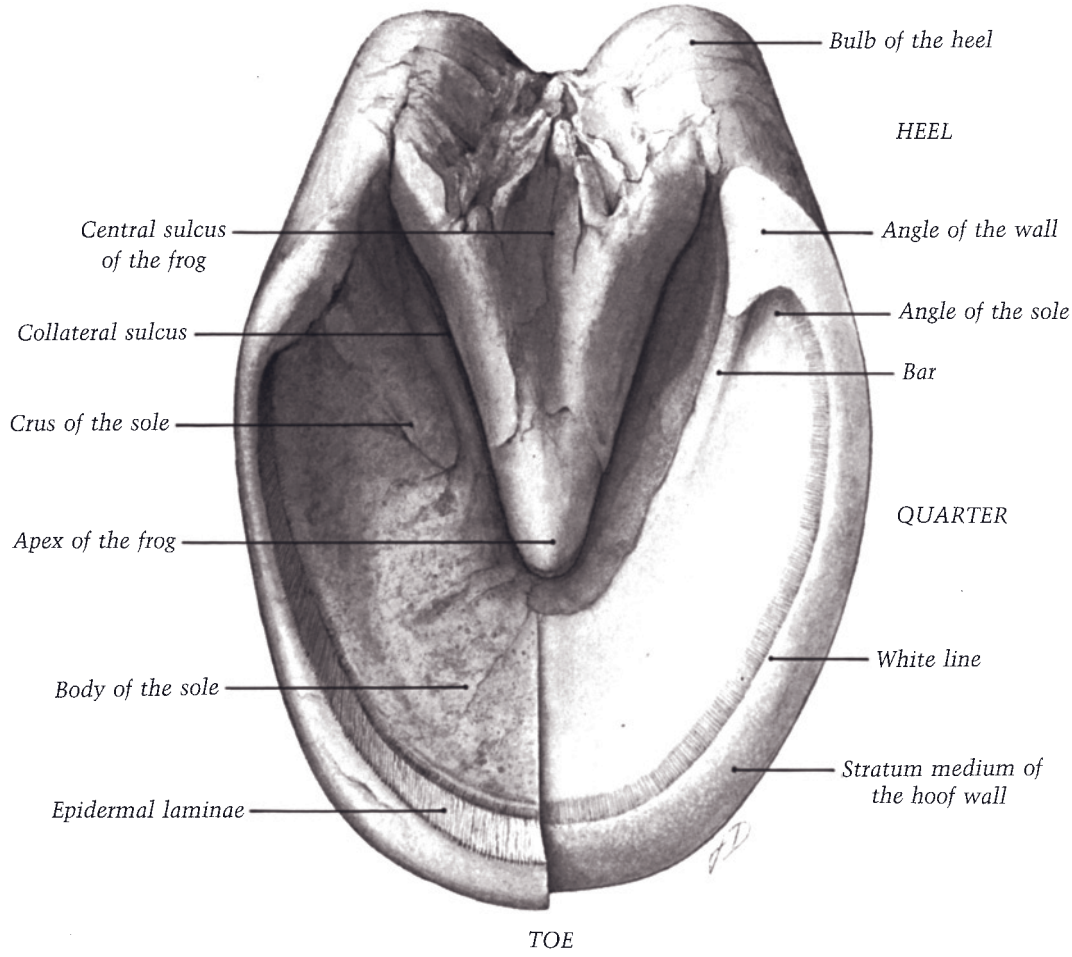


**Figure 1.3.** Topography of the solar surface of the hoof. The right half has been trimmed to emphasize the region of the white line.

forms a series of laminae that interdigitate with epidermal laminae of the stratum internum of the hoof wall. Shorter papillae extend from the perioplic, solar, and cuneate (frog) coria. The corium provides sensation as well as nourishment and attachment for the overlying stratified squamous epithelium comprising the ungual epidermis (hoof).

Three layers comprise the hoof wall: the stratum externum, stratum medium, and stratum internum (Figure 1.5). The superficial stratum externum is a thin layer of horn extending distad from the coronet a variable distance; this thin, soft layer, commonly called the periople, wears from the surface of the hoof wall so that it is present only on the bulbs of the heels and the proximal parts of the hoof wall. The bulk of the wall is a stratum medium consisting of horn tubules and intertubular horn. Horn tubules are generated by the stratum basale of the coronary epidermis covering the long papillae of the coronary corium. Intertubular horn is formed in between the projections.

Distal to the coronary groove, about 600 primary epidermal laminae of the stratum internum interweave with the primary dermal laminae of the laminar corium (Figures 1.6, 1.7). Approximately 100 microscopic secondary laminae branch at an angle from each primary lamina, further binding the hoof and corium together (Figure 1.6). There is some confusion concerning the terms "insensitive" and "sensitive" laminae. In the strictest sense the keratinized parts of the primary epidermal laminae are

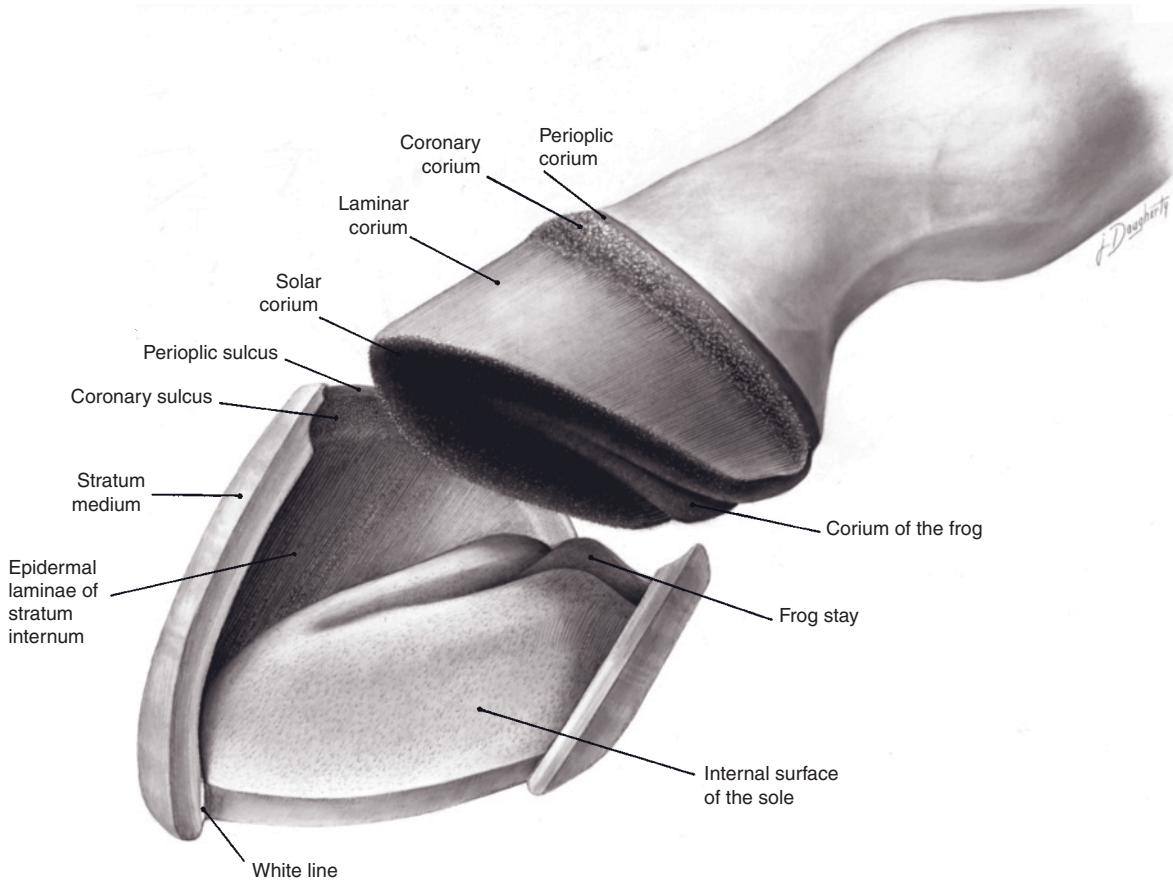


**Figure 1.4.** Dissected view of the relationship of the hoof to underlying regions of the corium (dermis).

insensitive; the stratum basale, which includes all of the secondary epidermal laminae, and the laminar corium are "sensitive." The terms "epidermal" and "dermal" (or corial) are more accurate adjectives to describe the laminae.

Growth of the hoof wall is primarily from the basal layer of the coronary epidermis toward the ground. The growth of the wall progresses at the rate of approximately 6 mm/month, taking nine to 12 months for the toe to grow out. The wall grows more slowly in a cold environment. Growth also is slower in a dry environment when adequate moisture is not present in the wall. The hoof wall grows evenly distal to the coronary epidermis so that the youngest portion of the wall is at the heel (where it is shortest). Because this is the youngest part of the wall, it is also the most elastic, aiding in heel expansion during concussion. Contrary to popular belief, there is no difference in the stress-strain behavior or ultimate strength properties of pigmented and nonpigmented equine hooves. Water content of the hoof significantly affects its mechanical properties. A very dry or extremely hydrated hoof wall is more likely to crack than a normally hydrated hoof wall.

The frog (cuneus ungulae) is a wedge-shaped mass of keratinized stratified squamous epithelium rendered softer than other parts of the hoof by an increased water content. Apocrine glands, spherical masses of tubules in the corium of the frog, extend ducts that deliver secretions to the surface of the frog. The ground surface of the frog presents a pointed apex and central sulcus enclosed by two crura. Paracuneal (collateral) sulci separate the crura of the frog from the bars and the sole. The palmar aspect of the frog blends into the bulbs of the heels.

The coronary and perioplic coria and the stratum basale of the coronary and perioplic epidermis constitute the coronary band. Deep to the coronary band the subcutis is modified into the

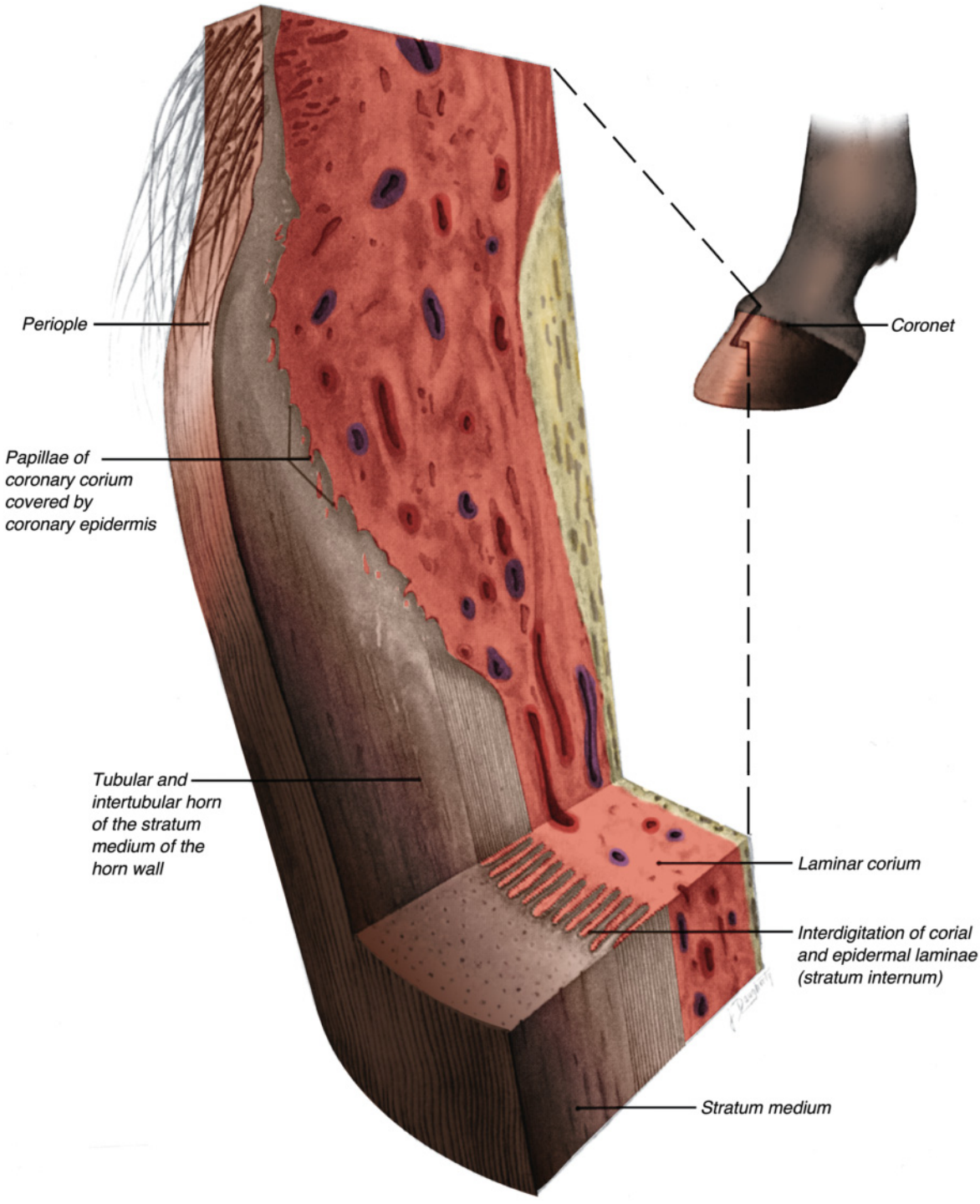


**Figure 1.5.** Three-dimensional dissection of the coronary region of the hoof wall.

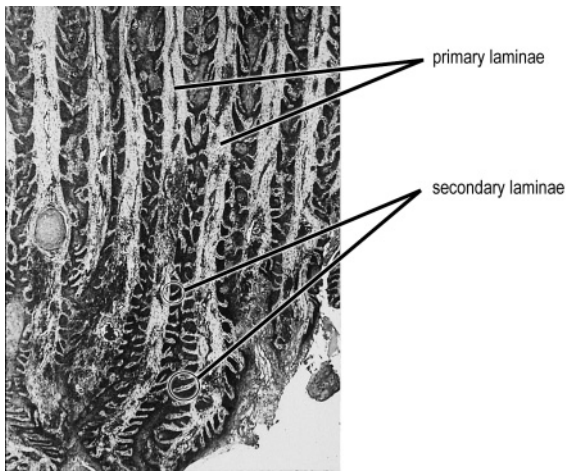


**Figure 1.6.** Photomicrograph of a cross section of equine hoof. Interdigitations of primary epidermal laminae and dermal (corial) laminae can be appreciated. Notice the small secondary laminae.

highly elastic coronary cushion. The coronary band and cushion form the bulging mass that fits into the coronary groove of the hoof. Part of the coronary venous plexus is within the coronary cushion. The plexus receives blood from the dorsal venous plexus in the laminar corium. Where the corium is adjacent to the distal phalanx, it blends with the bone's periosteum, serving (particularly in the laminar region) to connect the hoof to the bone (Figure 1.7).

### Internal Structures of the Foot

The medial and lateral cartilages of the distal phalanx (ungual cartilages) lie under the corium of the hoof and the skin, covered on their abaxial surfaces by the coronary venous plexus. They extend from each palmar process of the bone proximal to the coronary border of the hoof, where they may be palpated. The cartilages are concave on their axial surfaces, convex on their abaxial surfaces, and thicker distally where they attach to the bone. Toward the heels they curve toward one another. Each cartilage is perforated in its palmar half by several foramina for the passage of veins connecting the palmar venous plexus with the coronary venous plexus.

Five ligaments stabilize each cartilage of the distal phalanx (Figures 1.8, 1.9). Filling in between the cartilages is the digital cushion, a highly modified subcutis consisting of a meshwork of collagenous and elastic fibers, adipose tissue, and small masses of fibrocartilage (Figure

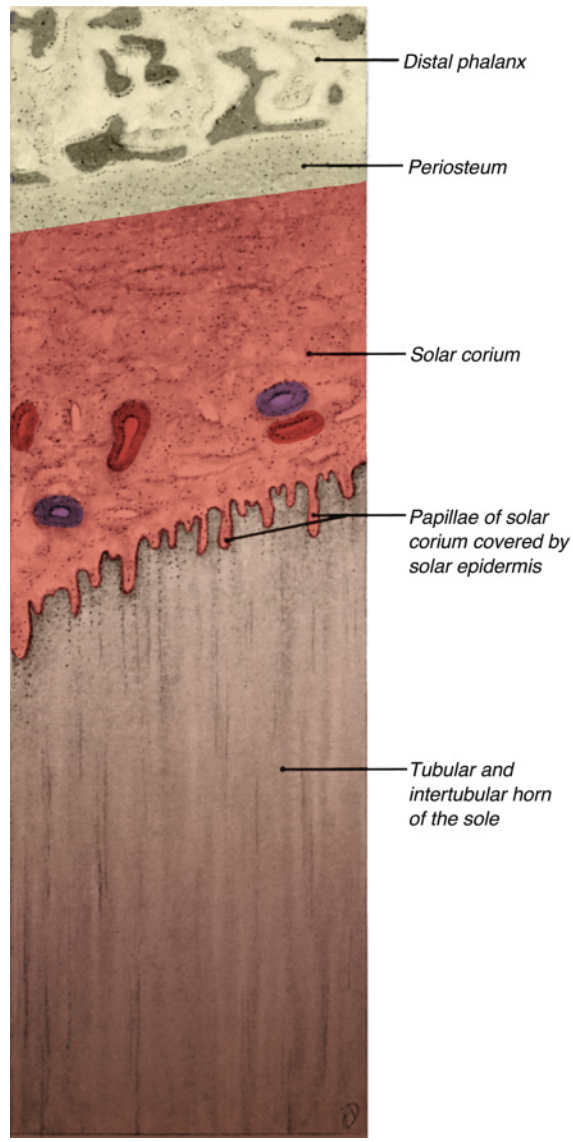


**Figure 1.7.** Histological relationship of the periosteum, corium, and horn of the sole.

1.9). Only a few blood vessels ramify in the digital cushion. Dorsoproximally the digital cushion connects with the distal digital anular ligament. The apex of the wedge-shaped digital cushion is attached to the deep digital flexor tendon (DDFT) as the latter inserts on the solar surface of the distal phalanx. The base of the digital cushion bulges into the bulbs of the heels, which are separated superficially by a central shallow groove. The structure and relationships of the digital cushion indicate its anticoncussive function.

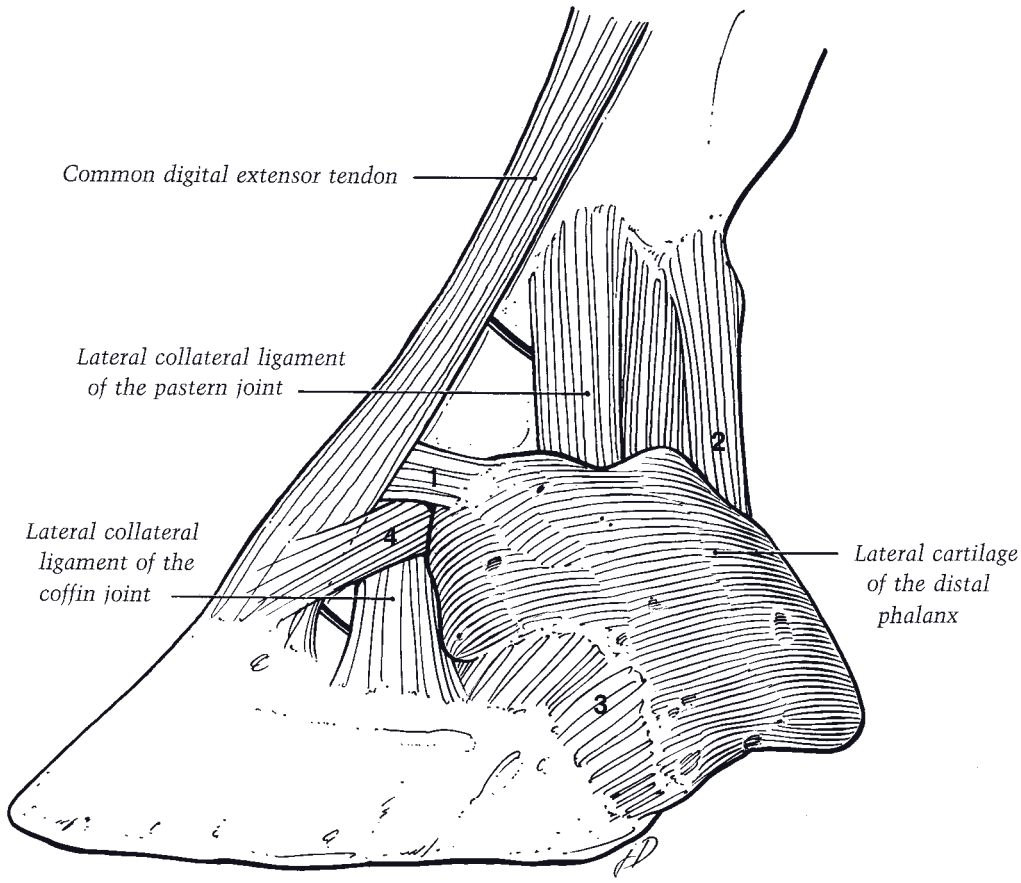


**Figure 1.8.** Four of the ligaments (1, 2, 3, and 4) that stabilize the cartilage of the distal phalanx.

As the DDFT courses to its insertion on the distal phalanx, it is bound down by the distal digital anular ligament (Figure 1.10) and passes over the complementary fibrocartilage, a fibrocartilaginous plate extending from the proximal extremity of the palmar surface of the middle phalanx. Then the tendon gives off two secondary attachments to the distal aspect of the palmar surface of the bone (Figure 1.11). Continuing distad toward its primary attachment on the flexor surface of the distal phalanx, the DDFT passes over the navicular bursa (bursa podotrochlearis) interposed between the tendon and the fibrocartilaginous distal scutum covering the flexor surface of the navicular bone.

The proximal border of the navicular bone presents a groove containing foramina for passage of small vessels and nerves. The distal border of the bone has a small, elongated facet that articulates with the distal phalanx. Several variously enlarged, foramina-containing fossae lie in an elongated depression palmar to that facet (Figure 1.12). Two concave areas on the main articular surface of the navicular bone contact the distal articular surface of the middle phalanx. The navicular bone is supported in its position by three ligaments comprising the navicular suspensory apparatus. A collateral sesamoidean (suspensory navicular) ligament arises from the distal end of the proximal phalanx (Figures 1.10, 1.11). These collateral sesamoidean ligaments sweep obliquely distad, each ligament crossing the pastern joint, and then giving off a branch that joins the end of the navicular bone to the cartilage of the distal phalanx. Each collateral sesamoidean ligament terminates by attaching to the proximal border of the navicular bone and joining with the contralateral ligament. Distally, the navicular bone is stabilized by the distal sesamoidean impar ligament, a fibrous

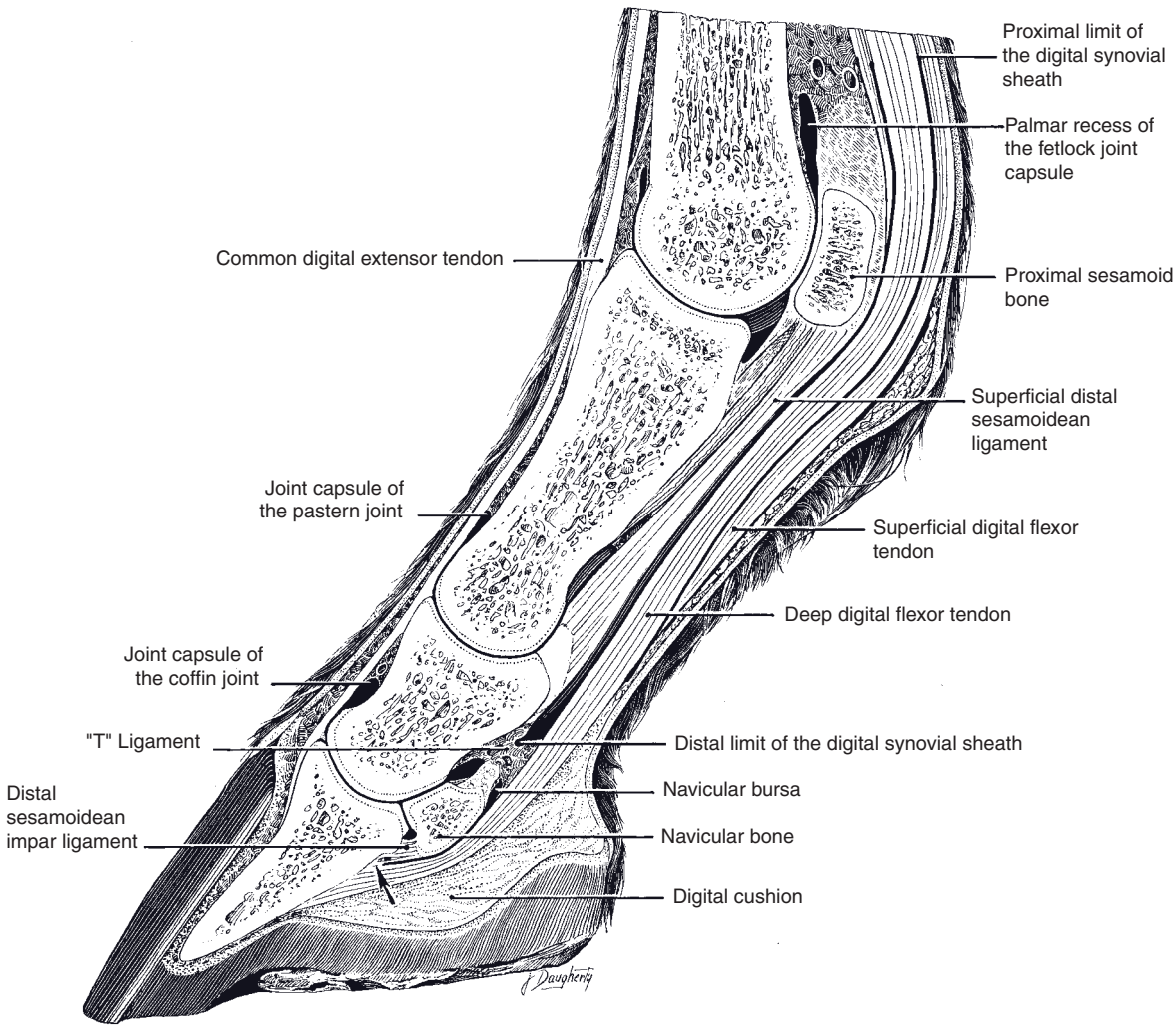


**Figure 1.9.** Sagittal section of the equine fetlock and digit.

sheet extending from the distal border of the bone to intersect with the DDFT (Figure 1.9).

The distal articular surface of the middle phalanx, the articular surface of the distal phalanx, and the two articular surfaces of the navicular bone form the coffin joint, a ginglymus of limited range of motion. Short collateral ligaments arise from the distal end of the middle phalanx, pass distad deep to the cartilages of the distal phalanx, and terminate on either side of the extensor process and the dorsal part of each cartilage.

The synovial membrane of the coffin joint has a dorsal pouch that extends proximad on the dorsal surface of the middle phalanx under the common digital extensor tendon nearly to the pastern joint. The synovium has a complex relationship on its palmar side to the ligaments and tendons that are found there. The proximal portions wrap around the distal ends of the collateral sesamoidean ligament and the distal palmar pouch forms a thin extension between the articulation of the navicular bone and the distal phalanx. Distally, this pouch's synovial membrane surrounds the distal sesamoidean impar ligament on each side where the DIP joint is closely associated with the neurovascular bundle that will enter the distal phalanx. Although a direct connection between the DIP joint and the navicular bursa is rare, passive diffusion of injected dye, anesthetics, and medications is thought to occur.

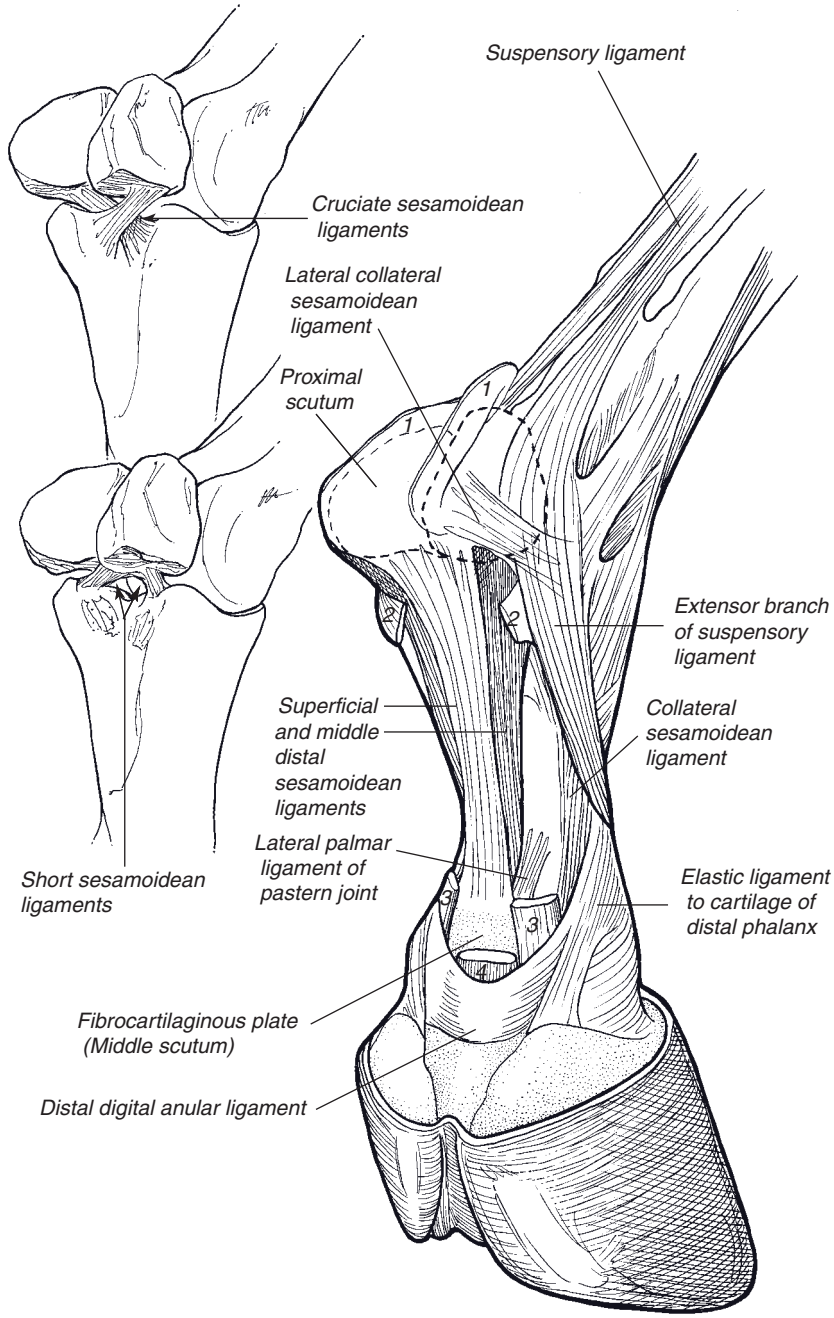


**Figure 1.10.** Sesamoidean ligaments. Dashed lines indicate positions of the proximal sesamoid bones embedded in the metacarpointersesamoidean ligament. Numbers indicate cut stumps of (1) palmar anular ligament, (2) proximal digital anular ligament, (3) superficial digital flexor, and (4) deep digital flexor tendon.

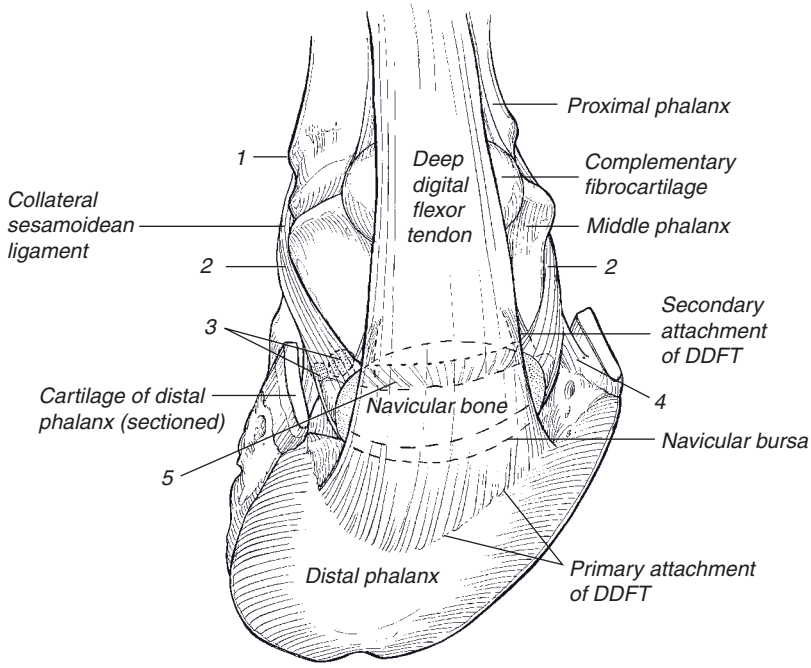


**Figure 1.11.** Attachments of the deep digital flexor tendon and collateral sesamoidean ligaments (CSL). (1) Attachment of the CSL to the proximal phalanx, (2) attachment of the CSL to the middle phalanx, (3) abaxial outpocketings of the palmar pouch of the synovial cavity of the distal interphalangeal joint, (4) attachment of the CSL to cartilage of the distal phalanx, (5) attachment of the medial and lateral CSLs to the navicular bone.

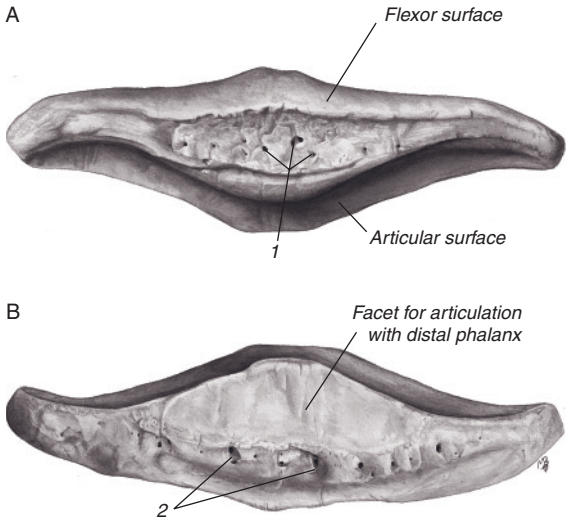


**Figure 1.12.** Distal sesamoid (navicular) bone. (A) Proximal view. (B) Distal view. (1) Foramina, (2) fossae.

## Pastern

Deep to the skin and superficial fascia on the palmar aspect of the pastern, the proximal digital anular ligament adheres to the superficial digital flexor tendon (SDFT) and extends to the medial and lateral borders of the proximal phalanx (long pastern bone). This fibrous band of deep fascia covers the SDFT as it bifurcates into two branches that insert on the proximal extremity of the middle phalanx just palmar to the collateral ligaments of the proximal interphalangeal (PIP; pastern) joint. The DDFT descends between the two branches of the SDFT and the digital flexor tendon sheath (DFTS) enfolds both tendons as far distally as the so-called "T ligament" (Figure 1.9). The latter is a fibrous partition attaching to the middle of the palmar surface of the middle phalanx.

Deep to the digital flexor tendons a series of ligaments referred to as distal sesamoidean ligaments (DSLs) extends distad from the bases of the two proximal sesamoid bones. The superficial straight sesamoidean ligament attaches distally to the fibrocartilaginous plate on the

proximal extremity of the palmar surface of the middle phalanx, the triangular middle (oblique) sesamoidean ligament attaches distally to a rough area on the palmar surface of the proximal phalanx, and the deep pair of cruciate ligaments cross, each attaching distally to the contralateral eminence on the proximal extremity of the proximal phalanx (Figure 1.10). A short sesamoidean ligament extends from the dorsal aspect of the base of each proximal sesamoid bone to the palmar edge of the articular surface of the proximal phalanx (Figure 1.10).

The pastern joint is formed by two convex areas on the distal extremity of the proximal phalanx and two corresponding concave areas expanded by a palmar fibrocartilaginous plate on the proximal extremity of the middle phalanx. Bones of the pastern joint are held together by two short collateral ligaments and four palmar ligaments. A central pair of palmar ligaments extends from the triangular rough surface on the proximal phalanx to the palmar margin of the proximal extremity of the middle phalanx; medial and lateral palmar ligaments pass from the proximal phalanx to the palmar surface of the proximal extremity of the middle phalanx. The joint capsule of the pastern joint blends with the deep surface of the common digital extensor tendon dorsally (Figure 1.9) and with the collateral ligaments of the joint. The palmar aspect of the capsule extends slightly proximad, compressed between the middle phalanx and the terminal branches of the SDFT and the straight sesamoidean ligament. These taut, overlying structures subdivide the capsule into medial and lateral pouches that are accessible for arthrocentesis.

### *Fetlock*

The fetlock of the thoracic limb is the region around the metacarpophalangeal (MCP) joint. Deep to the skin and superficial fascia, the palmar anular ligament of the fetlock binds the digital flexor tendons and their enclosing digital sheath in the sesamoid groove. The smooth depression between the proximal sesamoid bones through which the digital flexor tendons pass is formed by the fibrocartilage of the metacarpointersesamoidean ligament which covers the flexor surfaces of the proximal sesamoid bones. Immediately distal to the canal formed by the palmar anular ligament of the fetlock, the DDFT perforates through a circular opening in the SDFT, the manaca flexoria.

The distal extremity of the cannon bone, the proximal extremity of the proximal phalanx, the two proximal sesamoid bones, and the fibrocartilaginous metacarpointersesamoidean ligament in which the proximal sesamoids are embedded form the MCP joint. A somewhat cylindrical articular surface on MCIII/MTIII is divided by a sagittal ridge, and this surface fits into an accommodating depression formed by the proximal phalanx, the proximal sesamoid bones, and the metacarpointersesamoidean ligament. Collateral ligaments of the fetlock joint extend distad from the eminence and depression on each side of the third metacarpal bone. The superficial part of each ligament attaches distally to the edge of the articular surface of the proximal phalanx; the shorter, stouter deep part of the ligament attaches to the abaxial surface of the adjacent proximal sesamoid and the proximal phalanx.

The palmar part of the fetlock joint capsule is thicker and more voluminous than the dorsal part. A palmar recess (pouch) of the fetlock joint capsule extends proximad between the third metacarpal bone and the suspensory ligament. This pouch is palpable and even visible when the joint is inflamed, distending the palmar recess with synovial fluid. The joint capsule is reinforced on each side by the collateral ligaments and dorsally by fascia attaching to the common digital extensor tendon.

In the standing position, the fetlock and digit are prevented from non-physiologic hyperextension by the suspensory apparatus of the fetlock (interosseus muscle, intersesamoidean ligament, and distal sesamoidean ligaments), the digital flexor tendons, and the collateral ligaments of the joints. During flexion of the fetlock and digit, most of the movement is in the fetlock, the least amount of movement is in the pastern joint, and movement in the coffin joint is intermediate. Contraction of the common and lateral digital extensor muscles brings the bones and joints of the digit into alignment just before the hoof strikes the ground. The neurovascular supply to the digit and fetlock are illustrated in Figures 1.13 to 1.15.

### *Metacarpus*

The equine metacarpus consists of the large third metacarpal (cannon) bone, the second (medial) and fourth (lateral) small metacarpal bones (splint bones), and the structures associated with them. The shaft of each small metacarpal

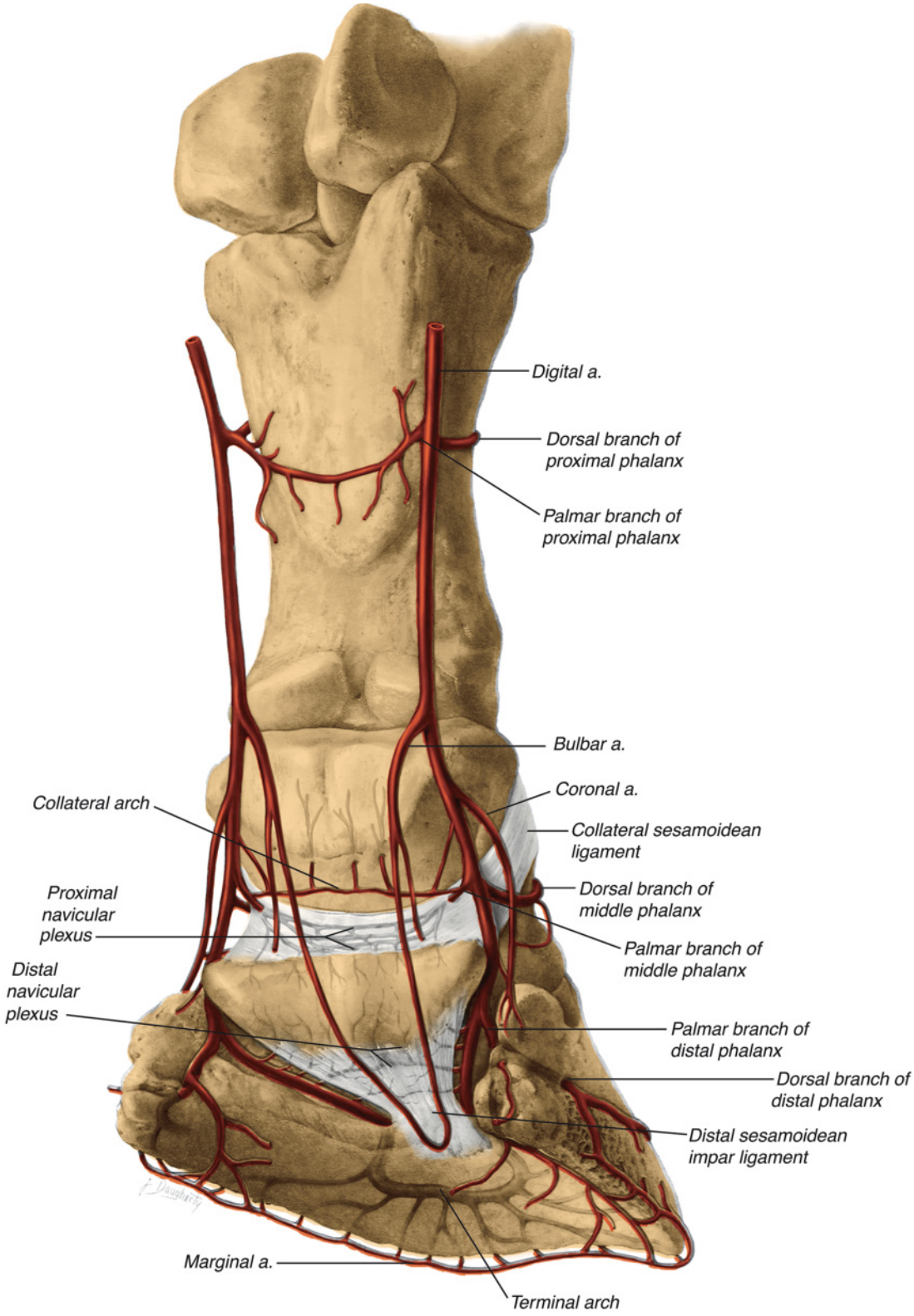


**Figure 1.13.** Arterial supply to the digit of the forelimb.

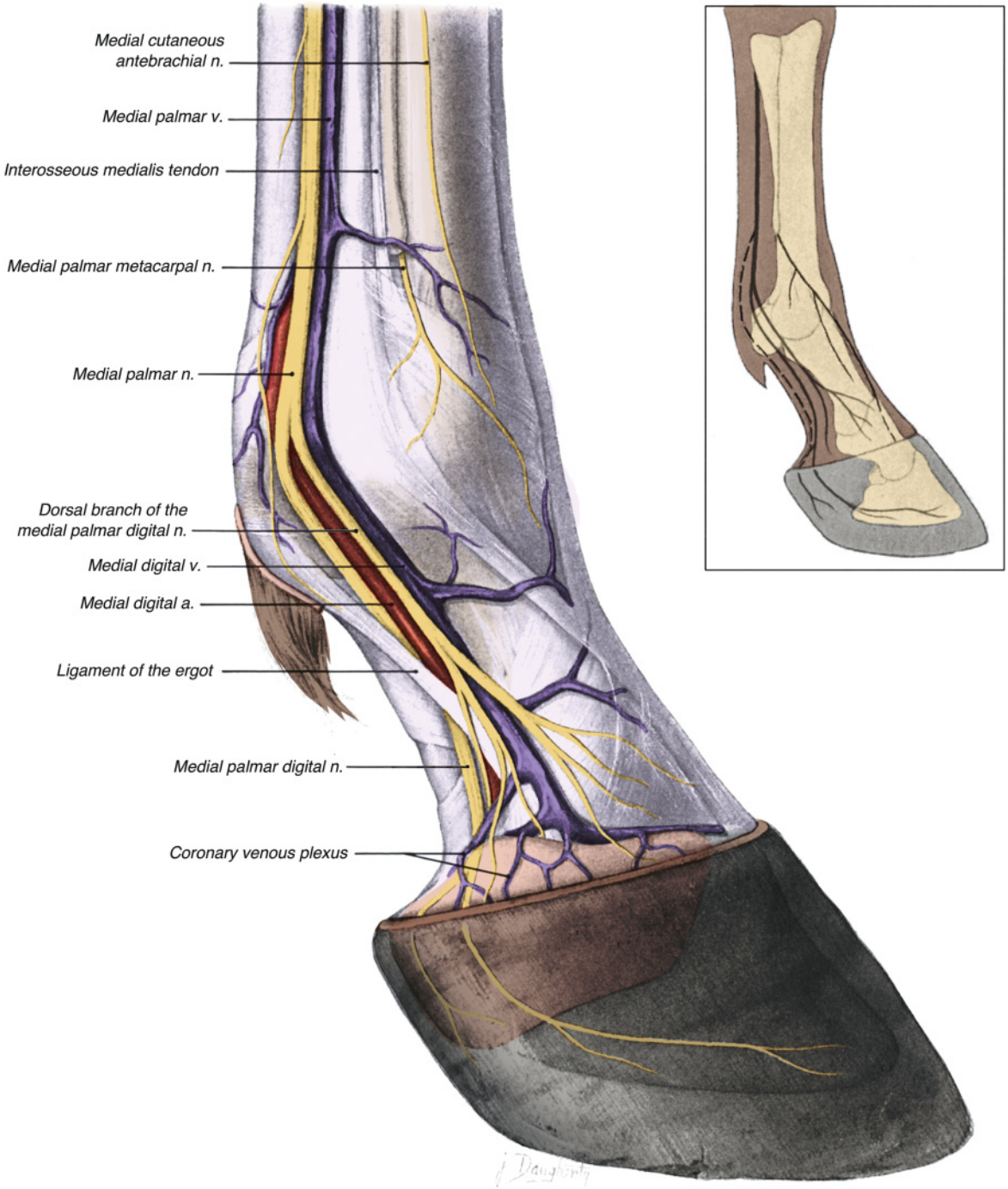


**Figure 1.14.** Medial aspect of the distal metacarpus, fetlock, and digit with skin and superficial fascia removed. Inset: schematic of the distribution of major nerves; dashed lines indicate variant branches.

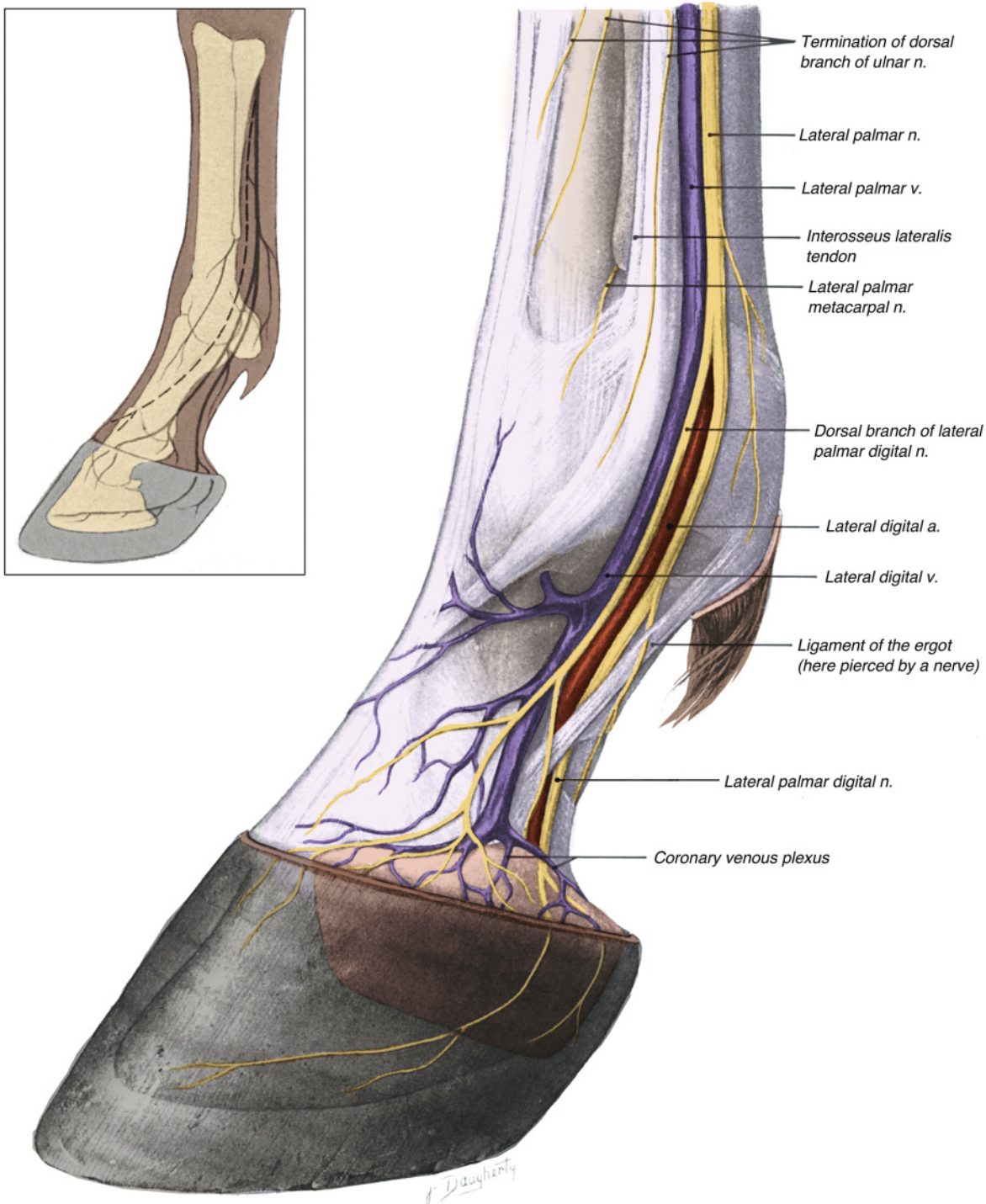


**Figure 1.15.** Lateral aspect of the distal metacarpus, fetlock, and digit with skin and superficial fascia removed. Inset: schematic of the distribution of major nerves; dashed lines indicate variant branches.

bone is united by an interosseous ligament to the large metacarpal bone. The cortex under the rounded dorsal surfaces of the metacarpal bones is thicker than the cortex under their concave palmar surfaces. The length and curvature of the shafts and the prominence of the free distal extremities ("buttons") of the small metacarpal bones are variable. The proximal extremities of the metacarpal bones articulate with the distal row of carpal bones; MCII articulating with the second and third carpals; MCIII articulating with the second, third, and fourth carpals; and MCIV with the fourth carpal bone.

### Dorsal Aspect

Deep to the skin, the main tendon of the common digital extensor muscle inclines proximolaterad from its central position at the fetlock across the dorsal surface of the third metacarpal bone. Proximally, the main tendon and the accompanying tendon of the radial head of the common digital extensor lie lateral to the insertional tendon of the extensor carpi radialis muscle on the prominent metacarpal tuberosity of MCIII. The tendon of the lateral digital extensor muscle is lateral to the common extensor tendon, and the small radial tendon of the latter usually joins the lateral digital extensor tendon. Occasionally the radial tendon pursues an independent course to the fetlock. A strong fibrous band from the accessory carpal bone reinforces the lateral digital extensor tendon as it angles dorsad in its descent from the lateral aspect of the carpus.

### Palmar Aspect

The SDFT is deep to the skin and subcutaneous fascia throughout the length of the metacarpus. Dorsally, it is intimately related to the fascial covering of the DDFT. The latter, in turn, lies against the palmar surface of the suspensory ligament (m. interosseus medius; middle or third interosseous muscle). The carpal synovial sheath, enclosing both digital flexor tendons, extends distad as far as the middle of the metacarpus. At this level, the DDFT is joined by its accessory ligament (carpal check ligament or "inferior" check ligament), the distal continuation of the palmar carpal ligament. The DFTS around the digital flexor tendons extends proximad into the distal fourth of the metacarpus (Figure 1.9).

The metacarpal groove, formed by the palmar surface of the third metacarpal bone and the axial surfaces of the second and fourth metacarpal bones, contains the suspensory ligament. The suspensory ligament arises from the distal row of carpal bones and the proximal end of the third metacarpal bone (Figures 1.16, 1.17). It is broad, relatively flat, and shorter than the suspensory ligament of the hindlimb. Variable amounts of striated muscle fibers within the mainly collagenous suspensory ligament are organized into two longitudinal bundles within the proximal part and body of the ligament (hence, interosseus medius "muscle"). In the distal fourth of the metacarpus, the suspensory ligament bifurcates to become associated with the two proximal sesamoid bones (Figure 1.10). Each side crosses the abaxial surface of proximal sesamoid bone and extends across the abaxial aspect of the proximal phalanx, where it contacts the origin of the ipsilateral collateral sesamoidean ligament. An extensor branch continues on to join the tendon of the common digital extensor muscle on the dorsal surface of the proximal phalanx (Figure 1.10).

The nerves and vessels of the metacarpus are illustrated in Figures 1.16 and 1.17.

## *Carpus*

The carpal region includes the carpal bones (radial, intermediate, ulnar, and accessory in the proximal row; first, second, third, and fourth in the distal row), the distal extremity of the radius, the proximal extremities of the three metacarpal bones, and the structures adjacent to these osseous components. The joints between the radius and proximal carpal bones (radiocarpal joint), ulna, and proximal carpal bones (ulnocarpal joint) together constitute the antebrachiocarpal joint. It and the middle carpal joint between the proximal and distal rows of carpal bones act as hinge joints, whereas the carpometacarpal joint between the distal row of carpal bones and the three metacarpal bones is a plane joint with minimal movement. An extensive antebrachiocarpal synovial sac sends extensions between the carpal bones of the proximal row and also encompasses the joints formed by the accessory carpal bone. A palmarolateral pouch extends from the radiocarpal sac out between the long tendon of the extensor carpi ulnaris muscle and the lateral styloid process of the radius. The middle carpal synovial sac communicates with the small carpometacarpal sac between the third and fourth carpal bones.

On the dorsal carpus, the tendon sheaths of the extensor carpi radialis, extensor carpi

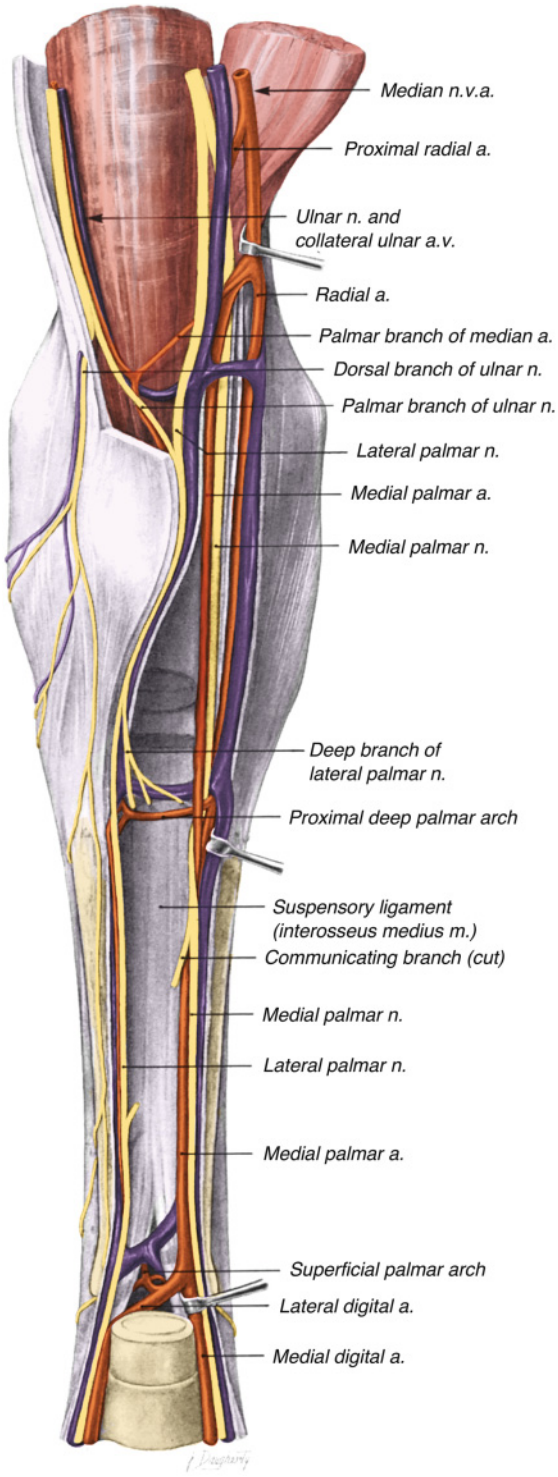


**Figure 1.16.** Deep dissection of the caudal aspects of the left carpus and metacarpus, with the medial palmar artery removed.

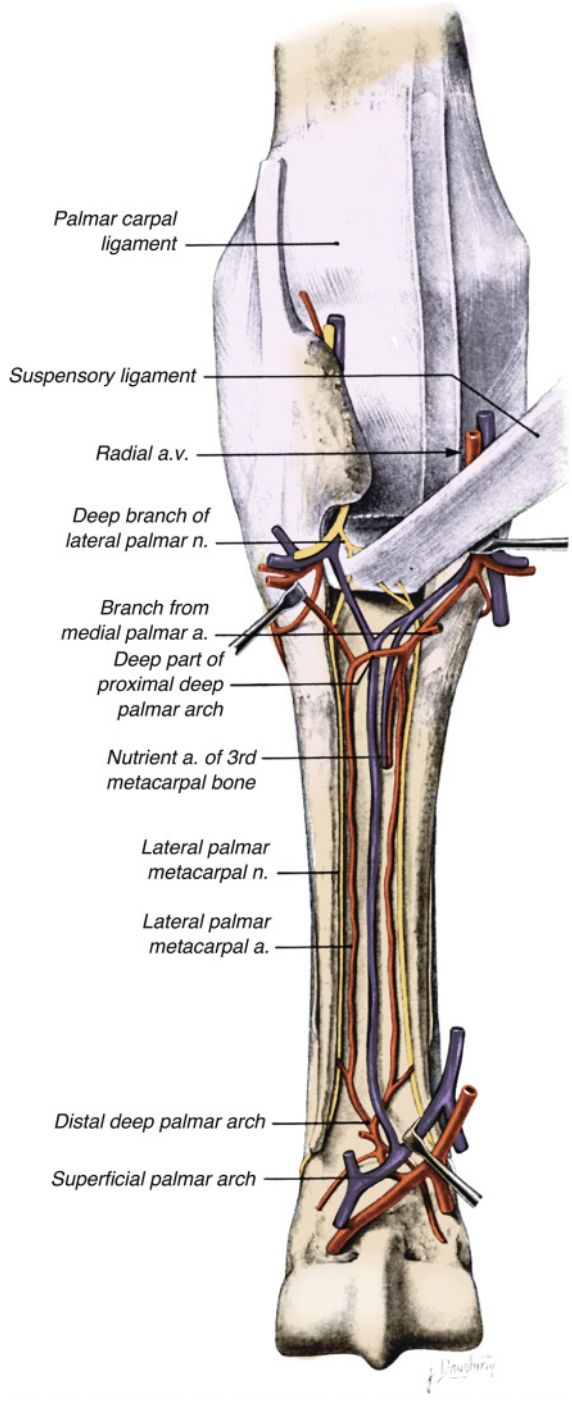


**Figure 1.17.** Caudal view of the left carpus and metacarpus; most of the digital flexor tendons are removed.

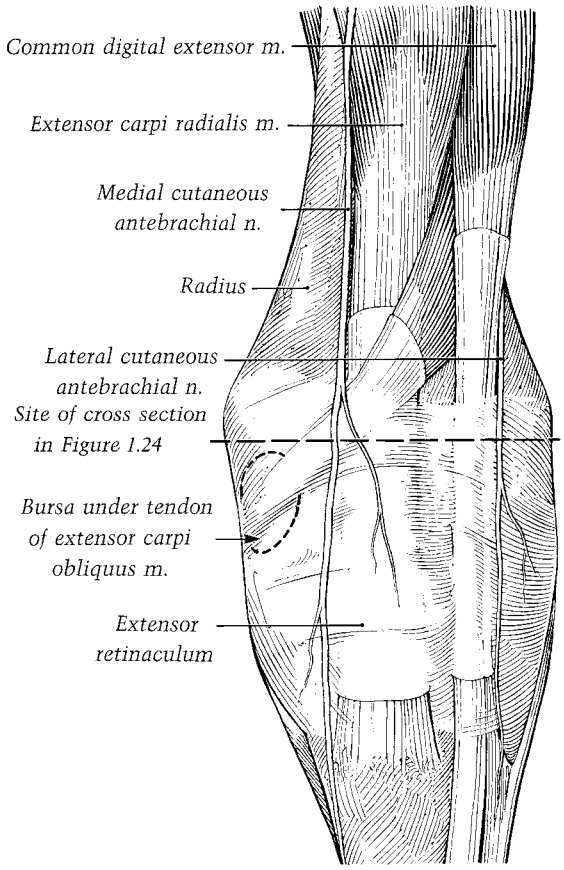


**Figure 1.18.** Dorsal view of the left carpus.

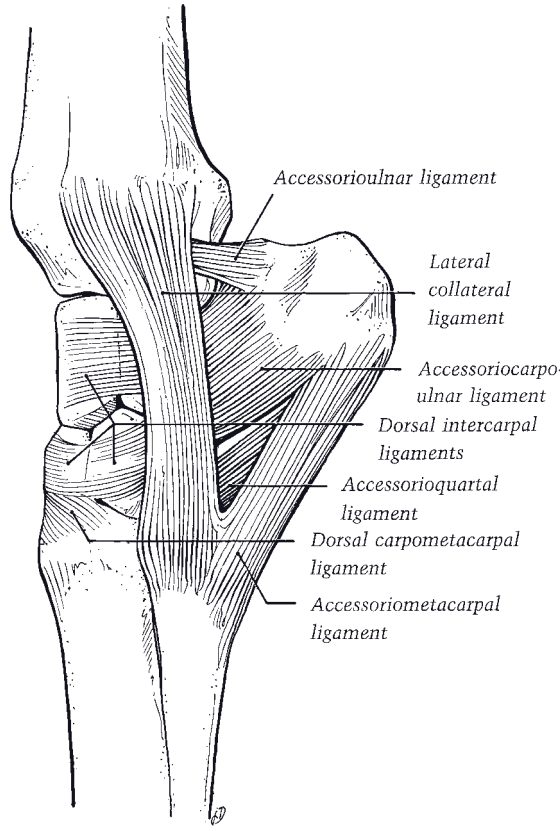


**Figure 1.19.** Carpal ligaments, lateral view.

obliquus (abductor digiti I longus), and the common digital extensor muscles are enclosed in fibrous passages through the deep fascia and then through the extensor retinaculum. The tendon sheaths of the common digital and extensor carpi obliquus tendons extend from the carpometacarpal articulation proximad to 6 to 8 cm above the carpus (Figure 1.18). The tendon sheath of the extensor carpi radialis muscle terminates at the middle of the carpus, and then the tendon becomes adherent to the retinaculum as it extends to its insertion on the metacarpal tuberosity (Figure 1.18).

## Lateral Aspect

The lateral collateral carpal ligament extends distad from its attachment on the styloid process of the radius (Figure 1.19). The superficial part of the ligament attaches distally on the fourth metacarpal bone and partly on the third metacarpal bone. The deep part of the ligament attaches on the ulnar carpal bone. Palmar to the lateral collateral carpal ligament, four ligaments support the accessory carpal bone. These ligaments, named according to their attachments, are from proximal to distal, the accessorioulnar, accessoriocarpoulnar, accessorioquartal, and accessoriometacarpal ligaments (Figure 1.19). Tendons of two muscles are associated with the accessory carpal bone. The short tendon of the extensor carpi ulnaris muscle (formerly ulnaris lateralis m.) attaches to the proximal border and lateral surface of the bone; the muscle's long tendon, enclosed in a synovial sheath, passes through a groove on the bone's lateral surface and then continues distad to insert on the proximal extremity of the fourth metacarpal bone (Figure 1.20). The single tendon of the flexor carpi ulnaris muscle attaches to the proximal border of the accessory carpal bone, blending with the flexor retinaculum.

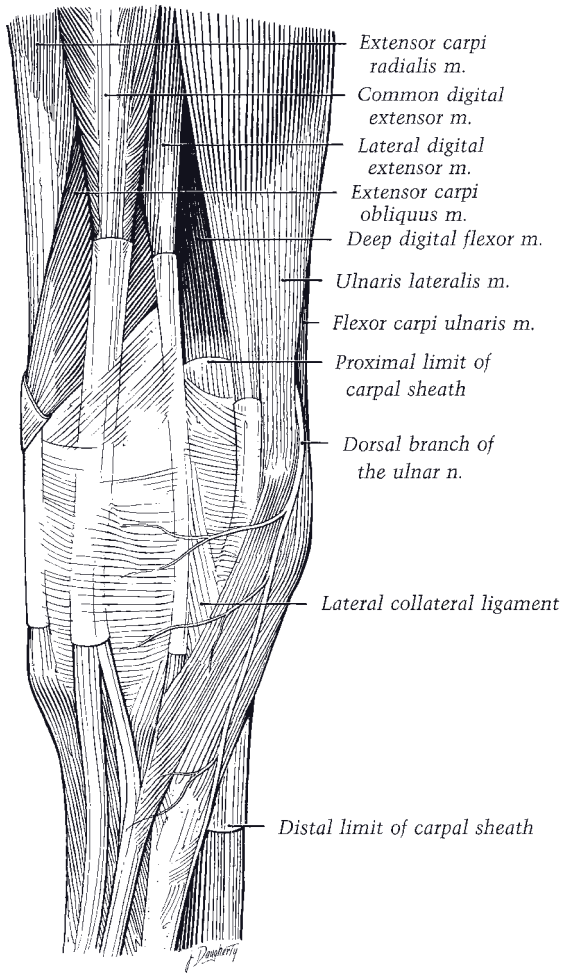


**Figure 1.20.** Lateral view of left distal forearm, carpus, and proximal metacarpus. Note that the ulnaris lateralis is now called extensor carpi ulnaris.

## Medial Aspect

The medial collateral carpal ligament extends from the medial styloid process of the radius and widens distally to attach to the proximal ends of the second and third metacarpal bones. Bundles of fibers also attach to the radial, second, and third carpal bones (Figure 1.21). Palmarly, the ligament joins the flexor retinaculum. The inconstant first carpal bone may be embedded in the palmar part of the medial collateral carpal ligament adjacent to the second carpal bone.

## Palmar Aspect

The flexor retinaculum is a fibrous band extending from the medial collateral ligament,

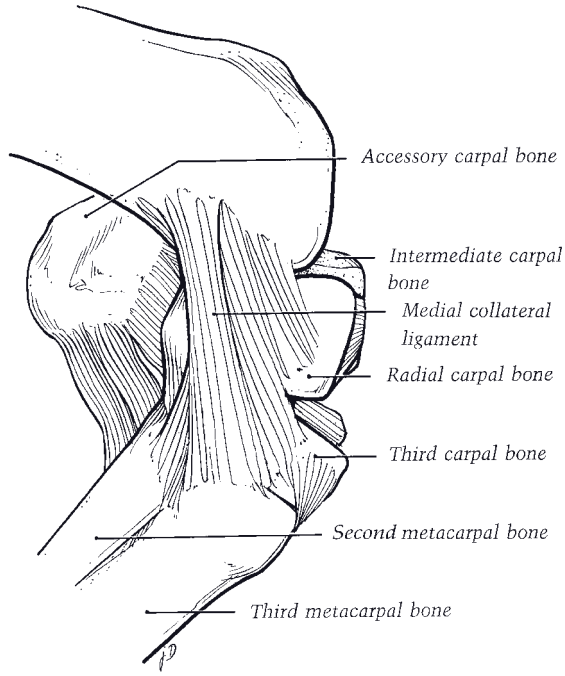


**Figure 1.21.** Carpal ligaments, medial view.

distal end of the radius, radial and second carpal bones, and proximal end of the second metacarpal bone laterad to the accessory carpal bone and the accessorioquartal and accessoriometacarpal ligaments. By bridging the carpal groove, the flexor retinaculum forms the mediopalmar wall of the carpal canal. It blends proximally with the caudal antebrachial fascia; distally, with the palmar metacarpal fascia. Proximally, the fan-shaped accessory ligament of the SDFT (radial check ligament) completes the medial wall of the carpal canal. The lateral wall is formed by the accessory carpal bone and its two distal ligaments. The palmar carpal ligament forms the smooth dorsal wall, its deep surface serving as the palmar part of the common fibrous capsule of the carpal joints. It attaches to the three palmar radiocarpal, three palmar intercarpal, and four carpometacarpal ligaments as well as the carpal bones. Distally, the palmar carpal ligament gives origin to the accessory ligament (carpal check) of the DDFT, which joins the tendon at approximately the middle of the metacarpus.

The carpal canal (Figure 1.22) contains the following structures: the SDFT and DDFT enclosed in the carpal synovial sheath; the medial palmar nerve and artery; and the lateral palmar

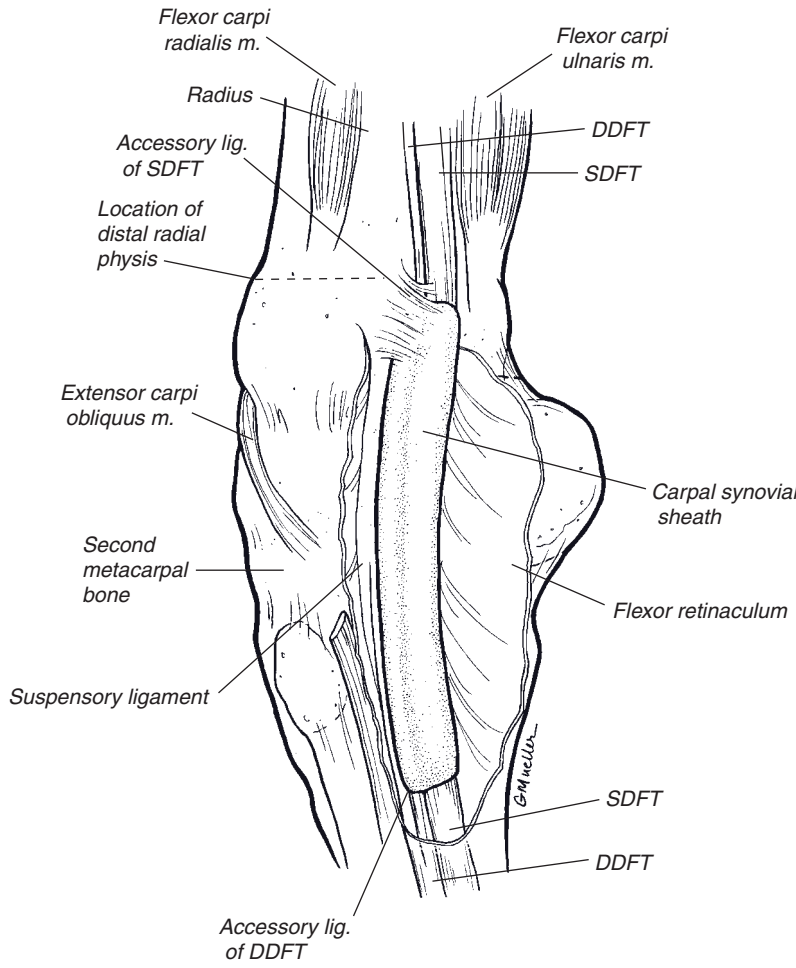


**Figure 1.22.** Palmaromedial view of carpus with flexor retinaculum cut and reflected. SDFT = superficial digital flexor tendon; DDFT = deep digital flexor tendon.

nerve, artery, and vein. Medial to the carpal canal, the tendon of the flexor carpi radialis, enclosed in its tendon sheath, descends to its attachment on the proximal part of the second metacarpal bone. The carpal synovial sheath enclosing the digital flexor tendons extends from a level 8 to 10 cm proximal to the antebrachiocarpal joint distad to near the middle of the metacarpus (Figure 1.22). An intertendinous membrane attaches to the palmaromedial surface of the DDFT and the dorsomedial surface of the SDFT, dividing the carpal synovial sheath into lateral and medial compartments. The neurovascular supply to the carpus is illustrated in Figures 1.16. 1.17, 1.20).

## *Antebrachium*

The antebrachium (forearm) includes the radius and ulna and the muscles, vessels, nerves, and skin surrounding the bones. The prominent muscle belly of the extensor carpi radialis muscle bulges under the skin on the cranial aspect. A horny cutaneous structure, the chestnut, is present on the medial skin of the distal one-third of the forearm. The chestnut is considered to be a vestige of the first digit. Beneath the superficial antebrachial fascia, the thick, deep antebrachial fascia invests all of the muscles of the forearm and provides for insertion of the tensor fasciae antebrachii muscle medially, the cleidobrachialis muscle laterally, and

the biceps brachii muscle cranially by means of the lacertus fibrosus. The deep fascia merges with the periosteum on the medial surface of the radius and attaches to the collateral ligaments and bony prominences at the elbow. Extensor muscles are invested more tightly than the flexor muscles. Intermuscular septa extend from the deep fascia between the common and lateral digital extensors, between the common digital extensor and extensor carpi radialis muscles, and between the radial and ulnar carpal flexors.

### Extensor Muscles

The extensor carpi radialis is the largest of the extensor muscles of the antebrachium. It attaches proximally to the lateral epicondyle and radial fossa of the humerus (along with the tendon of origin of the common digital extensor); it also attaches to the elbow joint capsule, the deep fascia, and the septum between the two muscles. The extensive tendon traversing the extensor carpi radialis blends with the deep fascia of the forearm after the fascia receives the lacertus fibrosus from the biceps brachii muscle. A tendon lying obliquely across the tendon of insertion of the extensor carpi radialis is that of the smallest muscle of the extensor group, the extensor carpi obliquus muscle (abductor digiti I longus), which originates on the lateral surface of the distal half of the radius. In its oblique course the muscle is at first deep to the common digital extensor, then its tendon crosses that of the extensor carpi radialis superficially. Its tendon sheath is adherent to the extensor retinaculum as the tendon angles over the carpus toward its insertion on the head of the second metacarpal bone (Figure 1.18).

The common digital extensor muscle (humeral head) takes common origin along with the extensor carpi radialis on the lateral epicondyle and radial fossa of the humerus, with additional attachments to the ulna, deep fascia, lateral aspect of the radius, and the lateral collateral ligament of the elbow. Its tendon of insertion, enclosed in its tendon sheath, occupies its respective groove on the distal extremity of the radius. The lateral digital extensor muscle lies under the deep fascia against the radius and ulna between the extensor carpi ulnaris caudally and the larger common digital extensor muscle belly cranially. The lateral digital extensor originates from the radius, ulna, lateral collateral ligament of the elbow joint, and the intermuscular septum from the deep fascia.

### Flexor Muscles

The flexor carpi radialis muscle is related to the mediocaudal surface of the radius (Figure 1.23), extending distad from the medial epicondyle of the humerus to the proximal extremity of the second metacarpal bone. Caudal and partially deep to the preceding muscle, the flexor carpi ulnaris muscle is formed by an ulnar head from the olecranon and a humeral head from the medial epicondyle and extends to the accessory carpal bone. The next muscle belly caudal to the flexor carpi ulnaris is that of the extensor carpi ulnaris muscle (formerly ulnaris lateralis) that originates on the lateral epicondyle of the humerus caudal to the lateral collateral ligament of elbow joint. The muscle extends distad to insert on the proximal and lateral aspects of the accessory carpal bone and, by means of a longer, sheathed tendon, to the proximal end of the fourth metacarpal bone. The preceding three muscles flex the carpal joint and extend the elbow joint, even though the extensor carpi ulnaris is morphologically an extensor of the carpal joint and supplied by the radial nerve.

The humeral head of the superficial digital flexor muscle originates from the medial epicondyle of the humerus and then lies deep to the ulnar head of the deep digital flexor (which is quite superficial as it originates from the medial surface of the olecranon) and the flexor carpi ulnaris. Under the proximal part of the flexor retinaculum the tendon of the humeral head of the superficial digital flexor is joined by a flat, wide fibrous band, its accessory ligament (really a radial head of the muscle), which comes from its attachment on a ridge on the mediocaudal surface of the distal half of the radius. The long, distinct tendon of the ulnar head of the deep digital flexor muscle joins the main tendon of the large humeral head proximal to the antebrachiocarpal joint just before the combined tendon becomes enclosed with the tendon of the superficial digital flexor in the carpal synovial sheath.

The neurovascular supply to the antebrachium is illustrated in Figures 1.16 and 1.23.

## *Cubital (Elbow) Joint*

Muscles adjacent to the equine cubital joint include two principal flexors, the biceps brachii and the brachialis (aided by the extensor carpi radialis and common digital extensor muscles), and three principal extensors, the tensor fasciae

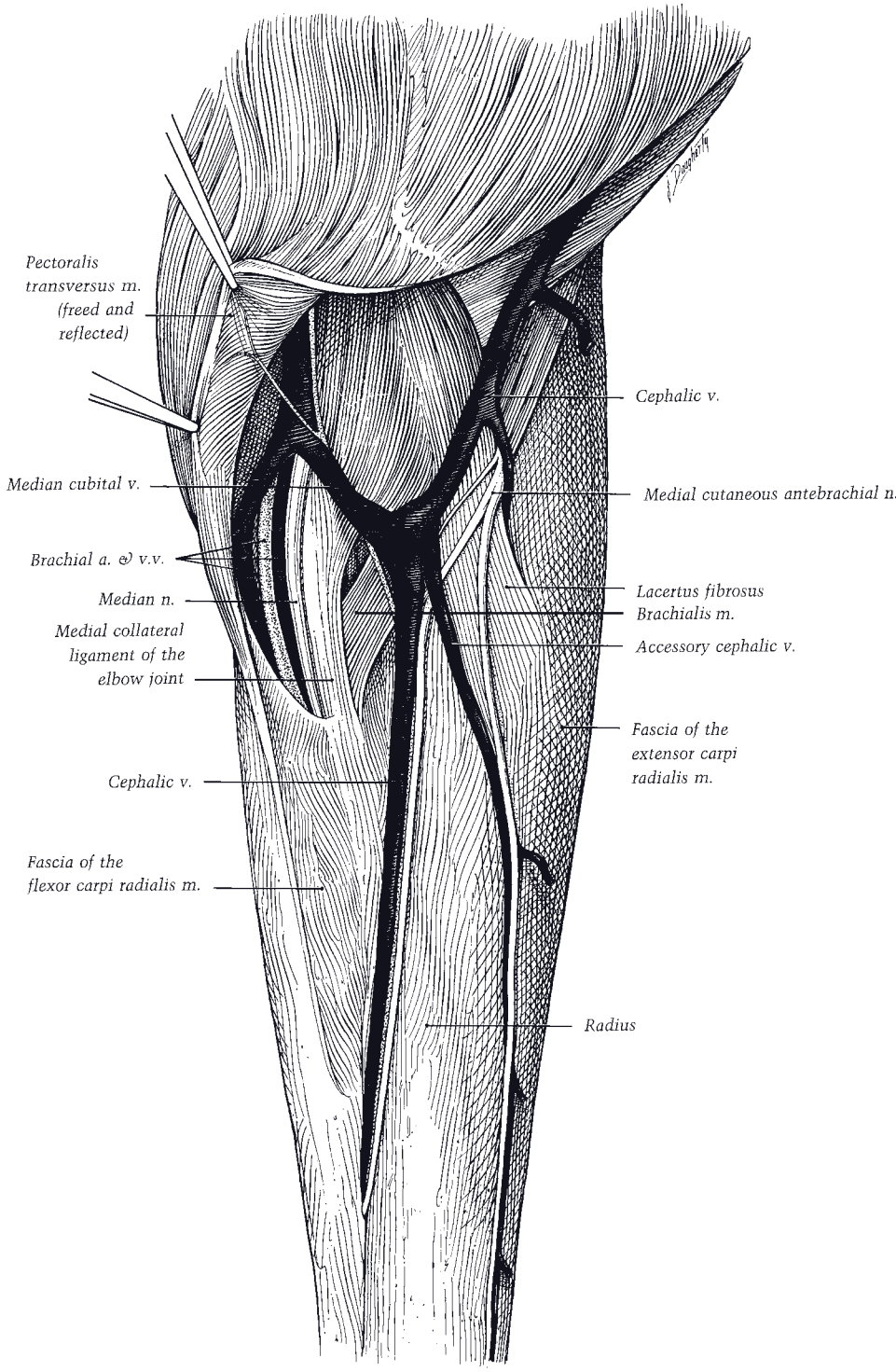


**Figure 1.23.** Caudomedial view of a superficial dissection of left elbow and forearm.

antebrachii, triceps brachii, and the anconeus (assisted by the flexors of the carpus and digit).

Cranially, the terminal part of the biceps brachii muscle crosses the joint, its tendon of insertion branching into the lacertus fibrosus, which joins the deep fascia of the extensor carpi radialis, and a short tendon attaching to the radial tuberosity and medial collateral ligament of the cubital joint (Figure 1.24). The terminal part of the brachialis muscle, curving around from its location in the musculospiral groove of the humerus, passes between the biceps brachii and extensor carpi radialis muscles to attach to the medial border of the radius under the long part of the medial collateral ligament of the elbow joint (Figure 1.23). The medial collateral ligament represents the pronator teres muscle in the horse.

Over the medial aspect of the elbow joint deep to the cranial part of the pectoralis transversus muscle, the median nerve, cranial brachial vein, brachial artery, and caudal brachial vein lie caudal to the medial collateral ligament of the elbow joint (Figure 1.23). The short part of the collateral ligament is deep and attaches to the radial tuberosity. Proximocaudal to the joint the collateral ulnar artery and vein, the ulnar nerve, and its cutaneous branch (caudal cutaneous antebrachial nerve) cross obliquely between the medial head of the triceps brachii and tensor fasciae antebrachii muscles. All three principal extensors of the cubital joint insert on the olecranon tuberosity of the ulna. A subcutaneous bursa may cover the caudal aspect of the olecranon tuberosity; deeply a subtendinous bursa lies under the tendon of insertion of the long head of the massive triceps brachii muscle (Figure 1.25).

The medially located tensor fasciae antebrachii also inserts on and acts to tense the deep antebrachial fascia. Deep to the triceps brachii, the small anconeus muscle originates from the caudal surface of the humerus, covers the olecranon fossa, and attaches to the elbow joint capsule, acting to elevate it when the joint is extended.

A fovea on the head and a ridge on the proximal extremity of the radius and the trochlear notch of the ulna articulate with the trochlea of the humerus, forming a ginglymus. The cranial articular angle is approximately 150° with a range of movement up to 60°. In flexion, the forearm is carried laterad due to the slightly oblique axis of movement of the elbow joint. Laterally, the cubital joint is covered by the distal part of the cutaneous omobrachialis muscle. A short, stout lateral collateral ligament extends from the lateral tuberosity of the radius to the lateral epicondyle of the humerus. Bands of fascia blend with the cranial part of the joint capsule. Caudally, the joint capsule becomes thinner as it extends into the olecranon fossa deep to the anconeus muscle. The joint capsule is adherent to the anconeus muscle and tendons of surrounding muscles. Extensions of the synovial lining project under the origins of the extensor carpi ulnaris and the digital flexor muscles and into the radioulnar articulation.

## *Arm and Shoulder*

The arm is the region around the humerus. The shoulder includes the shoulder joint (scapulohumeral joint) and the region around the scapula that blends dorsally into the withers. The heavy, deep fascia of the shoulder closely invests the underlying muscles and sends intermuscular septa in to attach to the spine and borders of the scapula. Within the superficial fascia over the lateral aspect of the shoulder and arm, the cutaneous omobrachialis muscle covers the deep fascia and extends as far distad as the cubital joint (Figure 1.25). The cutaneous muscle is innervated by the intercostobrachial nerve. Cutaneous sensation in this region is also mediated by brachial branches of the axillary and radial nerves. Superficial blood vessels are branches of the caudal circumflex humeral vessels.

The cleidobrachialis muscle (of the brachiocephalicus) covers the craniolateral aspect of the shoulder joint on the way to its insertion on the deltoid tuberosity, humeral crest, and the fascia of the arm (Figure 1.26). When the head and neck are fixed, this muscle acts as an extensor of the shoulder joint, drawing the forelimb craniad.

### Muscles Substituting for Shoulder Joint Ligaments

Cranially, the heavy, partly cartilaginous tendon of the biceps brachii muscle originates on the supraglenoid tubercle of the scapula and occupies the intertuberal groove of the humerus. A tendinous band from the pectoralis ascendens muscle extends from the lesser tubercle to the greater tubercle, serving to bind down the tendon of the biceps brachii. An intertuberal bursa lies under the tendon and extends around its sides. A tendinous intersection (an "internal tendon") extends distad through the muscle. In addition to flexing the elbow, the biceps brachii fixes the

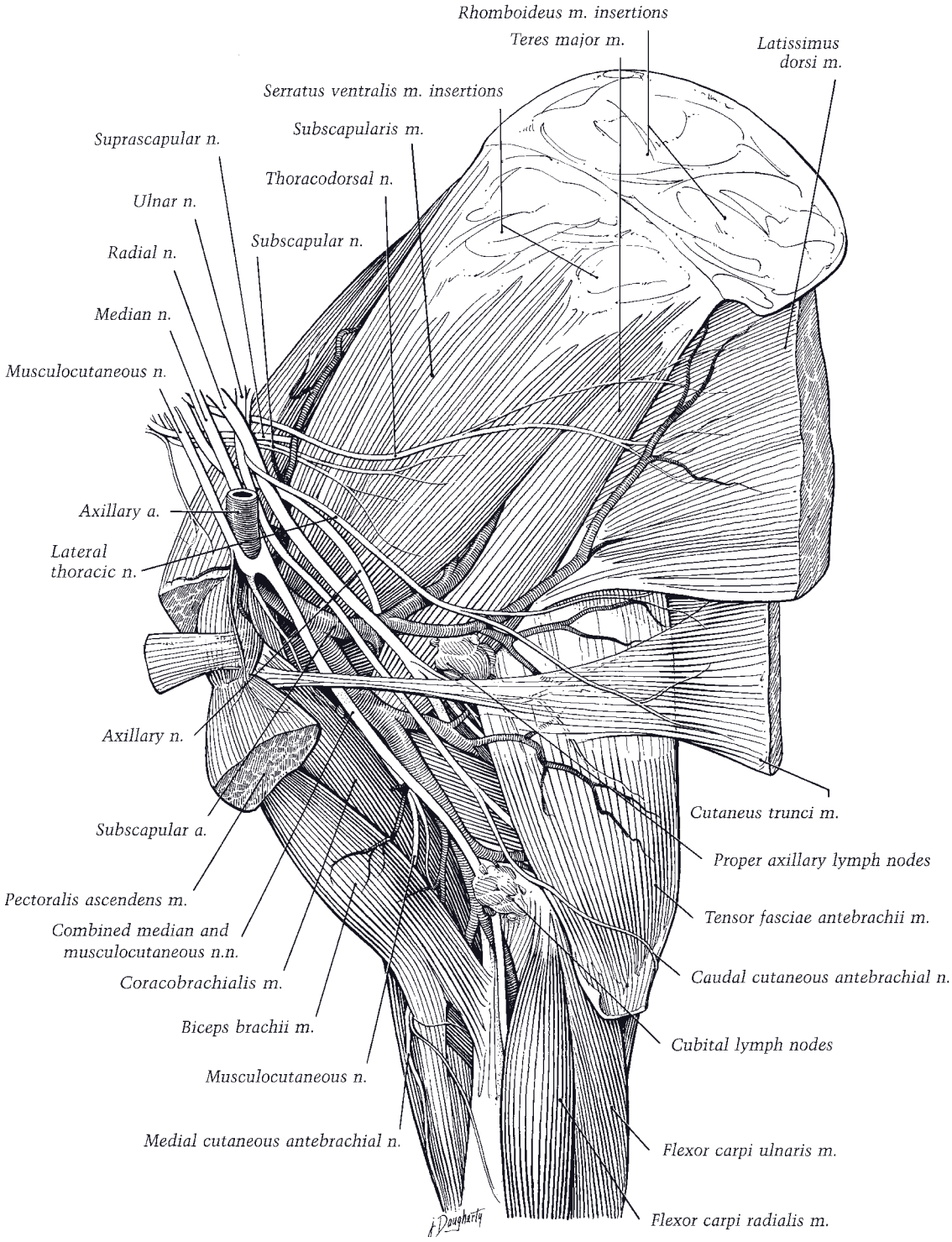


**Figure 1.24.** Medial view of the left shoulder, arm, and proximal forearm. Veins are not depicted.

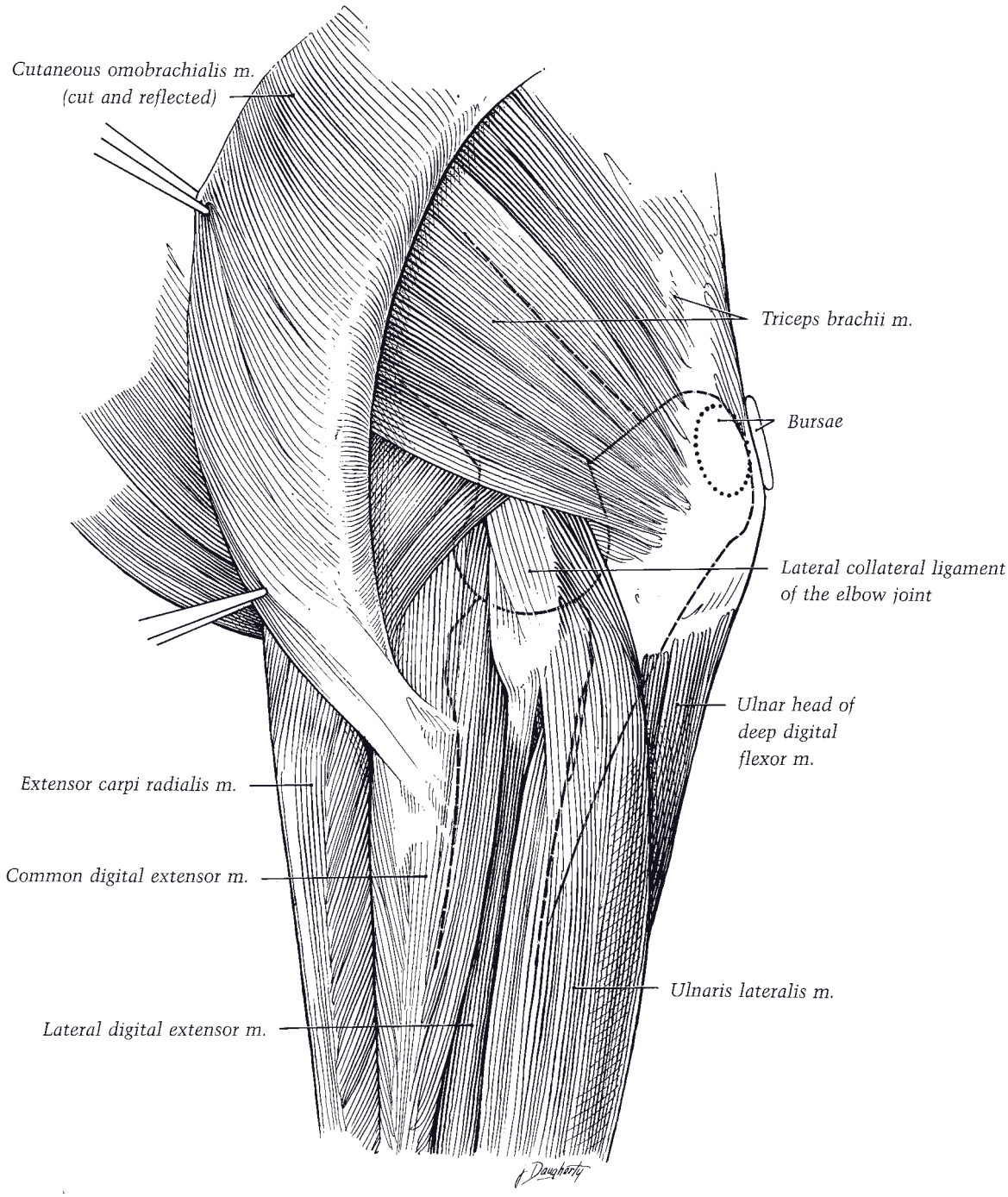


**Figure 1.25.** Lateral view of the left elbow. Dashed lines represent the locations of bony elements. Note that the ulnaris lateralis is now called extensor carpi ulnaris.

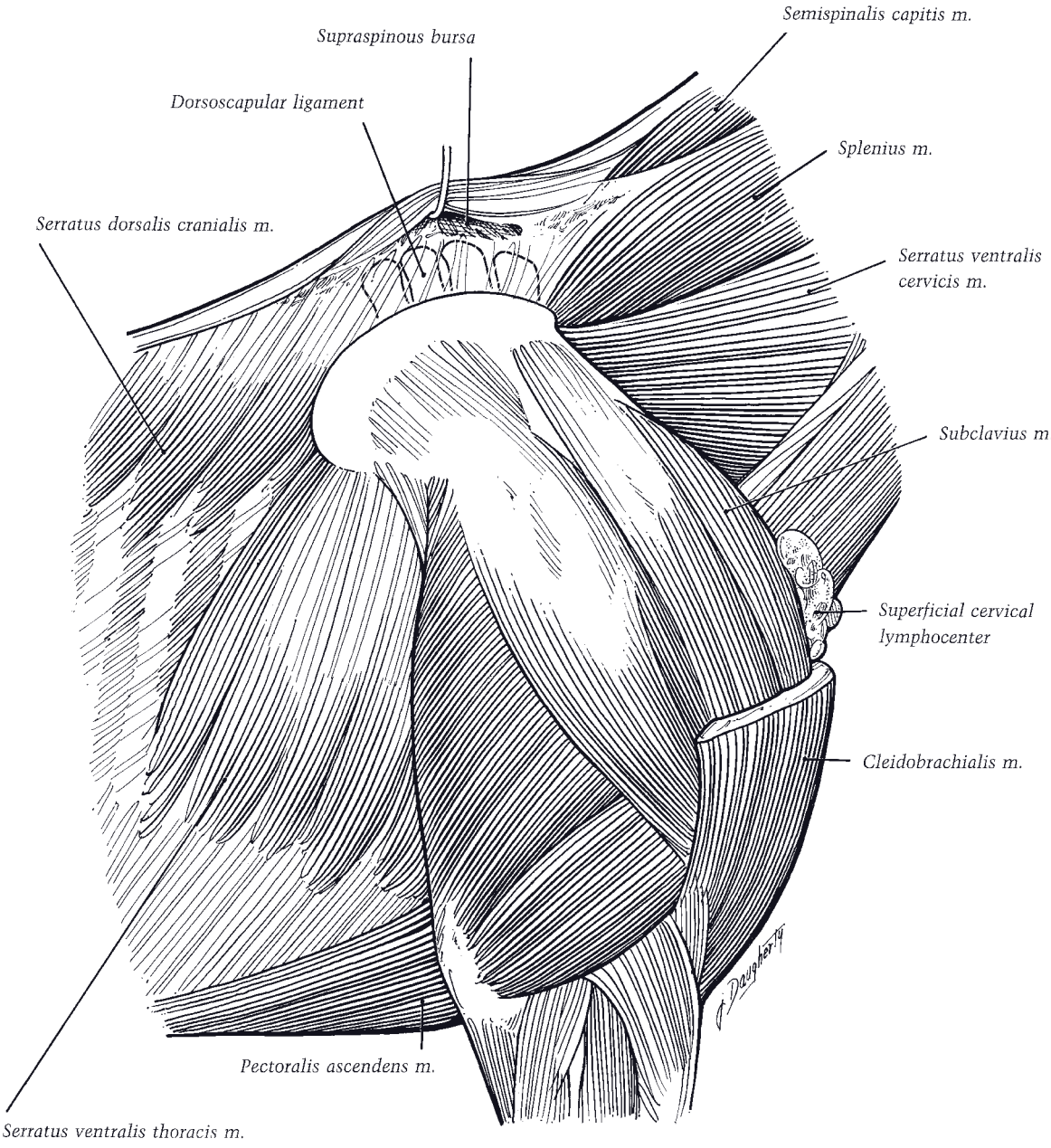


**Figure 1.26.** Right shoulder and dorsoscapular ligament. The spines of thoracic vertebrae 2 through 5 are outlined by dashed lines.

elbow and shoulder in the standing position. The musculocutaneous nerve supplies the biceps brachii. The supraspinatus muscle, which arises from the supraspinous fossa, the spine, and cartilage of the scapula, divides distally to attach to the greater and lesser tubercles of the humerus, serving with the bicipital tendon to stabilize the shoulder joint cranially (Figure 1.27).

Laterally, the infraspinatus muscle extends from the scapular cartilage and infraspinous fossa to insert on the caudal eminence of the greater tubercle and a triangular area on the

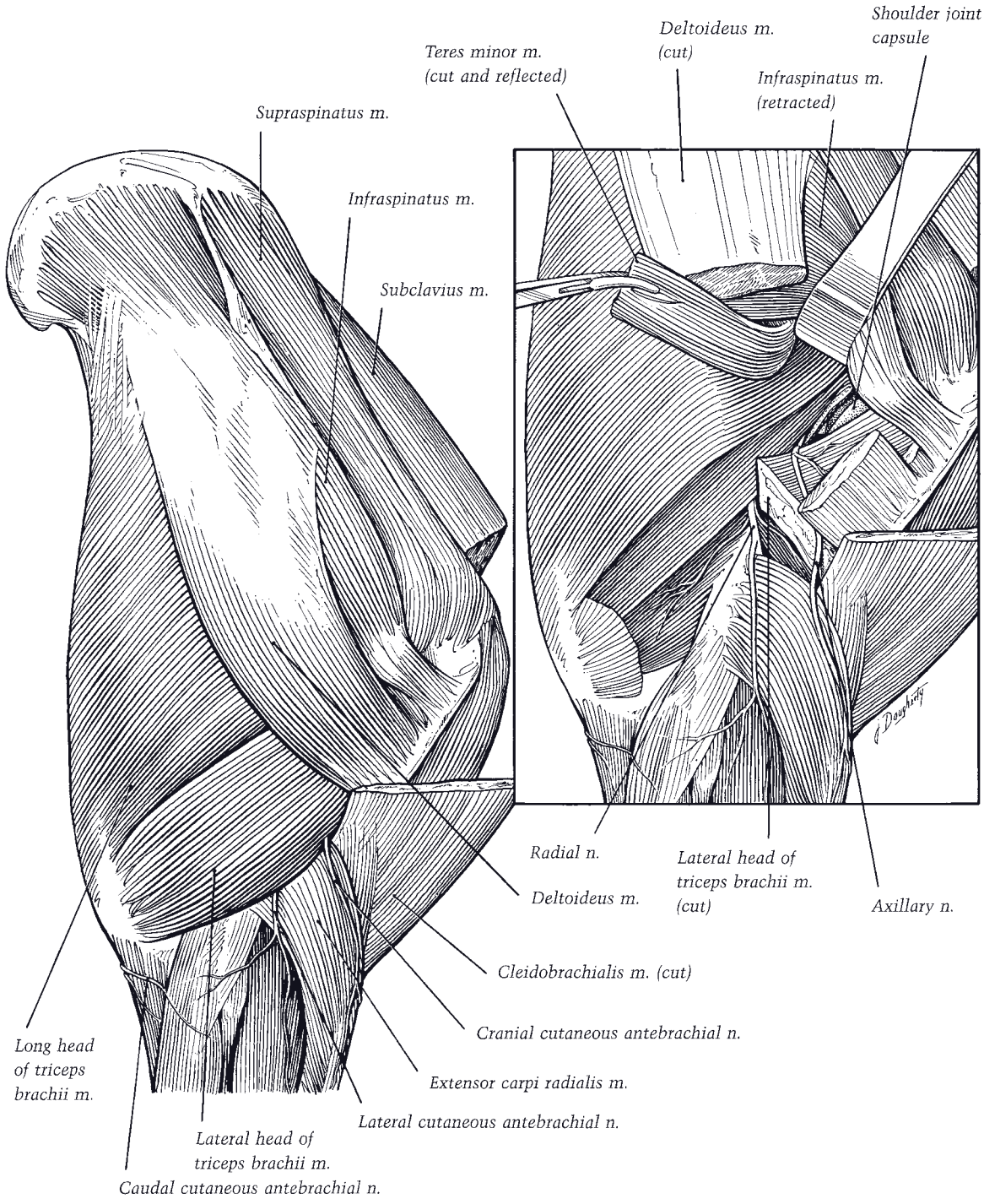


**Figure 1.27.** Lateral aspect of the right shoulder. Inset: deeper dissection exposing the shoulder joint.

distal part of the tubercle distal to the insertion of the supraspinatus (Figure 1.27). The partly cartilaginous tendon is protected from the underlying caudal eminence by adipose tissue and a constant synovial bursa that may communicate with the shoulder joint cavity. The tendon is the main lateral support of the shoulder joint assisted by the teres minor. The lateral insertion of the supraspinatus muscle also lends some lateral support. The supraspinatus muscle extends the shoulder joint; the teres minor muscle flexes the joint and, together with the infraspinatus, abducts the arm. The infraspinatus also rotates the arm laterad. The supraspinatus and infraspinatus muscles are supplied by the suprascapular nerve that reaches the supraspinous fossa by passing out between the subscapularis and supraspinatus muscles and then going around the distal fourth of the cranial border of the scapula.

The subscapularis muscle supports the shoulder joint medially. This adductor of the arm originates in the subscapular fossa of the scapula and inserts on the caudal eminence of the lesser tubercle of the humerus. Caudal support to the joint is rendered by the long head of the triceps brachii, the only head of this muscle originating from the scapula.

### Flexor Muscles of the Shoulder Joint

In addition to the long head of the triceps brachii muscle, four muscles flex the shoulder joint: laterally, the deltoideus and teres minor (which also abduct the arm), medially, the teres major and coracobrachialis (which also adduct the arm), and the latissimus dorsi. The first three muscles are innervated by branches from the axillary nerve; the coracobrachialis, by the musculocutaneous nerve; and the latissimus dorsi, by the thoracodorsal nerve.

The deltoideus muscle originates from the proximal part of the caudal border of the scapula and the scapular spine via the aponeurosis investing the infraspinatus (Figure 1.27). The muscle lies in a groove on the lateral surface of the triceps brachii and partly on the infraspinatus and teres minor muscles as it extends distad to attach to the deltoid tuberosity of the humerus and the brachial fascia. The teres major muscle extends from the caudal angle and border of the scapula across the medial surface of the triceps brachii to the teres major tuberosity of the humerus, where it inserts with the latissimus dorsi muscle (Figure 1.24). The coracoid process of the scapula gives origin to the coracobrachialis muscle that crosses the medial aspect of the shoulder joint and proximal arm to attach to the humerus just proximal to the teres major tuberosity and on the middle of the cranial surface of the bone.

### Shoulder Joint

The fibrous part of the joint capsule of the shoulder attaches up to 2 cm from the margins of the articular surfaces. Two elastic glenohumeral ligaments reinforce the joint capsule as they diverge from the supraglenoid tubercle to the humeral tuberosities. Within the shoulder joint the articular surface of the humeral head has approximately twice the area of the glenoid cavity of the scapula, even with the small extension afforded by the glenoid lip around the rim. The articular configuration of this ball-and-socket joint and the support of the surrounding muscles give great stability to the joint. Major movements are flexion and extension. While standing, the caudal angle of the shoulder joint is 120° to 130°. The angle increases to approximately 145° in extension and decreases to 80° in flexion. Muscles around the joint restrict abduction and adduction. Rotation is very limited.

### Muscles Overlying the Scapula

Beneath the skin over the scapular region, the broad, triangular flat trapezius muscle covers parts of eight underlying muscles. The cervical part of the trapezius arises by a thin aponeurosis from most of the funicular part of the ligamentum nuchae and inserts on the scapular spine and fascia of the shoulder and arm. The aponeurosis of the thoracic part of the trapezius takes origin from the supraspinous ligament from the third to the 10th thoracic vertebrae, and the muscle inserts on the tuber of the spine of the scapula. An aponeurosis joins the two parts of the trapezius. Innervated by the accessory nerve and dorsal branches of adjacent thoracic nerves, the trapezius muscle elevates the shoulder and draws it either craniad or caudad, depending on the activity of the cervical or thoracic parts, respectively.

Deep to the trapezius, the rhomboideus cervicis originates from the funicular part of the ligamentum nuchae, and the rhomboideus thoracis originates from the superficial surface of the dorsal part of the dorsoscapular ligament. Both parts of the rhomboideus muscle insert on the medial side of the scapular cartilage (Figure 1.24). This muscle is innervated by the sixth and

seventh cervical nerves and dorsal branches of nerves adjacent to the rhomboideus thoracis. The rhomboideus draws the scapula dorsocraniad and, when the limb is stationary, the cervical part helps to raise the neck.

The widest muscle of the shoulder girdle, the latissimus dorsi, has roughly the shape of a right triangle with the origin arising through a broad aponeurosis from the thoracolumbar fascia. Thin at first, the muscle becomes thicker as it passes medial to the long head of the triceps brachii to converge on a flat, common tendon of insertion with the teres major muscle (Figure 1.24).

From deep to superficial, the muscles contributing most substantially to the attachment of the thoracic limb to the trunk and neck are the serratus ventralis, the pectoral muscles, subclavius, brachiocephalicus, and omotransversarius. The serratus ventralis cervicis extends from the transverse processes of the last four cervical vertebrae to the serrated face of the medial surface of the scapula and adjacent scapular cartilage; the serratus ventralis thoracis converges dorsad from the lateral surfaces of the first eight or nine ribs to the serrated face of the scapula and adjacent scapular cartilage. Elastic lamellae from the ventral part of the dorsoscapular ligament are interspersed through the attachments of the serratus ventralis on the scapula. The two parts of the muscle and the contralateral serratus ventralis form a support suspending the thorax between the thoracic limbs. When both muscles contract, they elevate the thorax; acting independently, each serratus ventralis shifts the trunk's weight to the ipsilateral limb. During locomotion the cervical part of the muscle draws the dorsal border of the scapula craniad; the thoracic part draws the scapula caudad. When the limb is fixed, the serratus cervicis extends the neck or pulls it laterad.

Pectoral muscles attach to the sternum. There are two superficial pectoral muscles: (1) the pectoralis descendens muscle descending from the cartilage of the manubrium sterni to the deltoid tuberosity and the crest of the humerus and the brachial fascia, and (2) the pectoralis transversus muscle extending from the ventral part of the sternum between the first to the sixth sternebrae to the superficial fascia of the medial aspect of the antebrachium and to the humeral crest. The superficial pectoral muscles adduct the thoracic limb and tense the antebrachial fascia. The largest pectoral muscle, the deep pectoral (pectoralis ascendens) muscle (Figure 1.26), ascends from its attachments on the xyphoid cartilage, the ventral part of the sternum, the fourth to ninth costal cartilages, and the abdominal tunic to the cranial parts of the lesser and greater humeral tubercles and the tendon of origin of the coracobrachialis muscle. The subclavius has been traditionally grouped with the pectorales. It arises from the first four costal cartilages and the cranial half of the sternum and ends in an aponeurosis over the dorsal part of the supraspinatus muscle and the scapular fascia (Figures 1.26 and 1.27).

As has been noted, the cleidobrachialis part of the brachiocephalicus muscle extends from the indistinct clavicular intersection to the arm. The mastoid part of the muscle (cleidomastoideus) lies between the intersection and its attachments to the mastoid process and nuchal crest, partly overlapping the omotransversius muscle dorsally. The omotransversarius originates from the wing of the atlas and the transverse processes of the second, third, and fourth cervical vertebrae, and inserts on the humeral crest and fascia of the shoulder and arm. The dorsal branch of the accessory nerve passes through the cranial part of the omotransversius and then between that muscle and the trapezius.

### Dorsoscapular Ligament

Further attachment of the shoulder to the trunk is afforded by a thickened, superficial lamina of the thoracolumbar fascia, the dorsoscapular ligament. It consists of two parts: a collagenous portion attaches to the third, fourth, and fifth thoracic spines under the flattened part of the nuchal ligament subjacent to the supraspinous bursa (Figure 1.26). This part of the dorsoscapular ligament passes ventrad, ultimately attaching to the medial surface of the rhomboideus thoracis muscle. As it curves under the muscle, the collagenous part changes to an elastic part. A horizontal lamina of the elastic part forms the ventral sheath of the rhomboideus thoracis muscle. Several vertical laminae project from the ventral aspect of the horizontal lamina, surrounding bundles of the serratus ventralis muscle that insert on the scapula.

The neurovascular supply to the arm and shoulder is illustrated in Figures 1.24 and 1.27.

## *Stay Apparatus of the Thoracic Limb*

In the standing position, interacting muscles, tendons, and ligaments constituting the stay apparatus of the thoracic limb fix the alignment of the bones of the manus, suspend the fetlock,

lock the carpus, and stabilize the elbow and shoulder joints. This complex of structures functions almost entirely as a passive, force-resisting system. It permits the horse to stand (and sleep) with a minimum of muscular activity (Figure 1.28).

The suspensory apparatus of the fetlock is a ligamentous continuum extending from the proximal end of the third metacarpal bone to the proximal and middle phalanges. It consists of the suspensory ligament, metacarpointersesamoidean ligament with its embedded proximal sesamoid bones, and distal sesamoidean ligaments. The SDFT and the DDFT and their accessory (check) ligaments assist the suspensory apparatus of the fetlock in suspending the fetlock and preventing excessive overextension of the joint and collapse of the fetlock during weight bearing, especially on striking the ground. Disruption of the suspensory ligament alters its support of the fetlock, resulting in "sinking" or hyperextension of the fetlock.

A certain amount of muscle tone prevails in all "resting" muscles of the limb, even during most stages of sleep. Tension exerted by the long head of the triceps brachii muscle is essential to prevent flexion of the elbow joint and collapse of the forelimb. The elbow's eccentrically placed collateral ligaments allow it to exhibit considerable stability in the extended position, a stability enhanced by the triceps' tone. Flexion of the joint is further limited by the muscle belly and fibrous components of the superficial digital flexor muscle descending from its attachment of the medial epicondyle of the humerus.

A tendinous continuum extending from the supraglenoid tubercle to the metacarpal tuberosity is formed by the main tendon of the biceps brachii muscle, its fibrous "internal tendon," and its superficial tendon (lacertus fibrosus) that blends into the fascia of the extensor carpi radialis muscle and via it to the tendon of insertion of this muscle. This complex prevents flexion of the shoulder joint caused by the weight of the trunk via the scapular attachments of the serratus ventralis muscle and the dorsoscapular ligament. Additionally, the tendon of the extensor carpi radialis opposes flexion of the carpus.

## HINDLIMB

### *Digit and Fetlock*

The hind foot is somewhat smaller and more elongate than the fore foot. It has been commonly reported that, compared to the fore hoof, the angle of the toe of the hind hoof is slightly greater. Within the hind pastern the middle phalanx is narrower and longer and the proximal phalanx somewhat shorter than their counterparts in the thoracic limb (Figure 1.29).

The long digital extensor muscle's tendon attaches to the dorsal surfaces of the proximal and middle phalanges and the extensor process of the distal phalanx, but the tendon of the lateral digital extensor usually does not attach to the proximal phalanx as it does in the thoracic limb. Digital flexor tendons, tendon sheaths, and bursae of the hind digit are not remarkably different. The suspensory apparatus of the fetlock and the configuration of the fetlock (metatarsophalangeal) joint are much the same as in the thoracic limb except that the dorsal articular angle of the fetlock is approximately 5° greater (i.e., is slightly more "upright").

The neurovascular supply to the hind digit and fetlock is illustrated in Figures 1.30 and 1.31

### *Metatarsus*

The equine metatarsus is about 16% longer than the corresponding metacarpus, and the third metatarsal bone is more rounded than the third metacarpal bone. The fourth metatarsal bone, particularly its proximal extremity, is larger than the second metatarsal bone.

Dorsally, the tendon of the long digital extensor muscle extends the length of the metatarsus beneath the skin and fascia. At the proximal third of the metatarsus, the long digital extensor tendon is joined by the tendon of the lateral digital extensor muscle. The angle formed by the conjoined long and lateral digital extensor tendons is occupied by the thin, triangular short digital extensor muscle. The short digital extensor originates on the lateral collateral ligament of the hock, the lateral tendon of the fibularis tertius muscle (most commonly called the peroneus tertius), and the middle extensor retinaculum, and inserts on the two large digital extensor tendons. All digital extensor muscles are bound down by the distal extensor retinaculum in the proximal third of the metatarsus (Figure 1.30).

Plantarly, the SDFT is similar to the corresponding tendon in the metacarpus. The deep digital flexor muscle's principal tendon is intimately related to the dorsomedial aspect of the SDFT. In the proximal third of the metatarsus, the principal tendon is joined by the tendon of the medial digital flexor muscle (the medial head of the deep digital flexor muscle). A weakly developed, slender accessory ligament (tarsal or

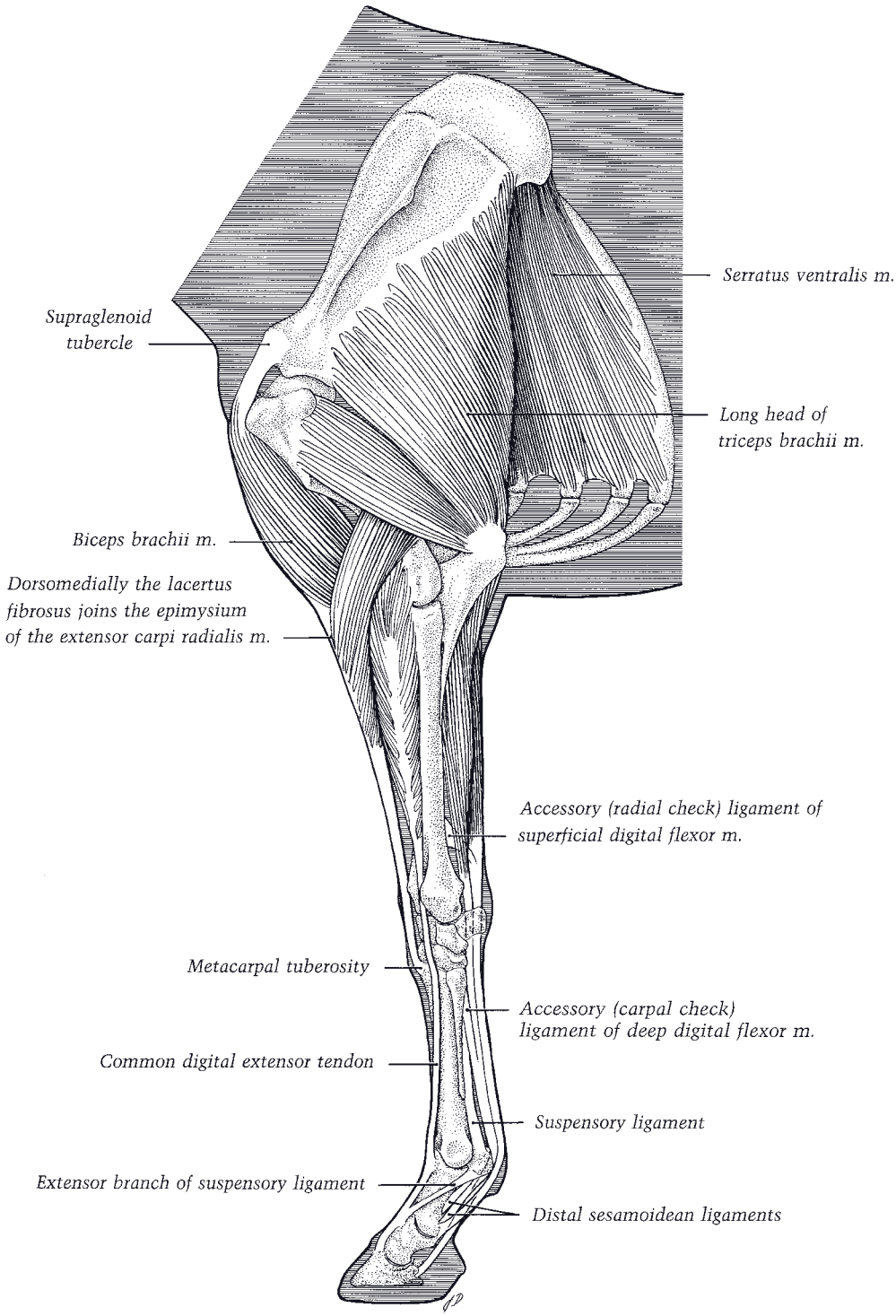


**Figure 1.28.** Stay apparatus of the left thoracic limb.

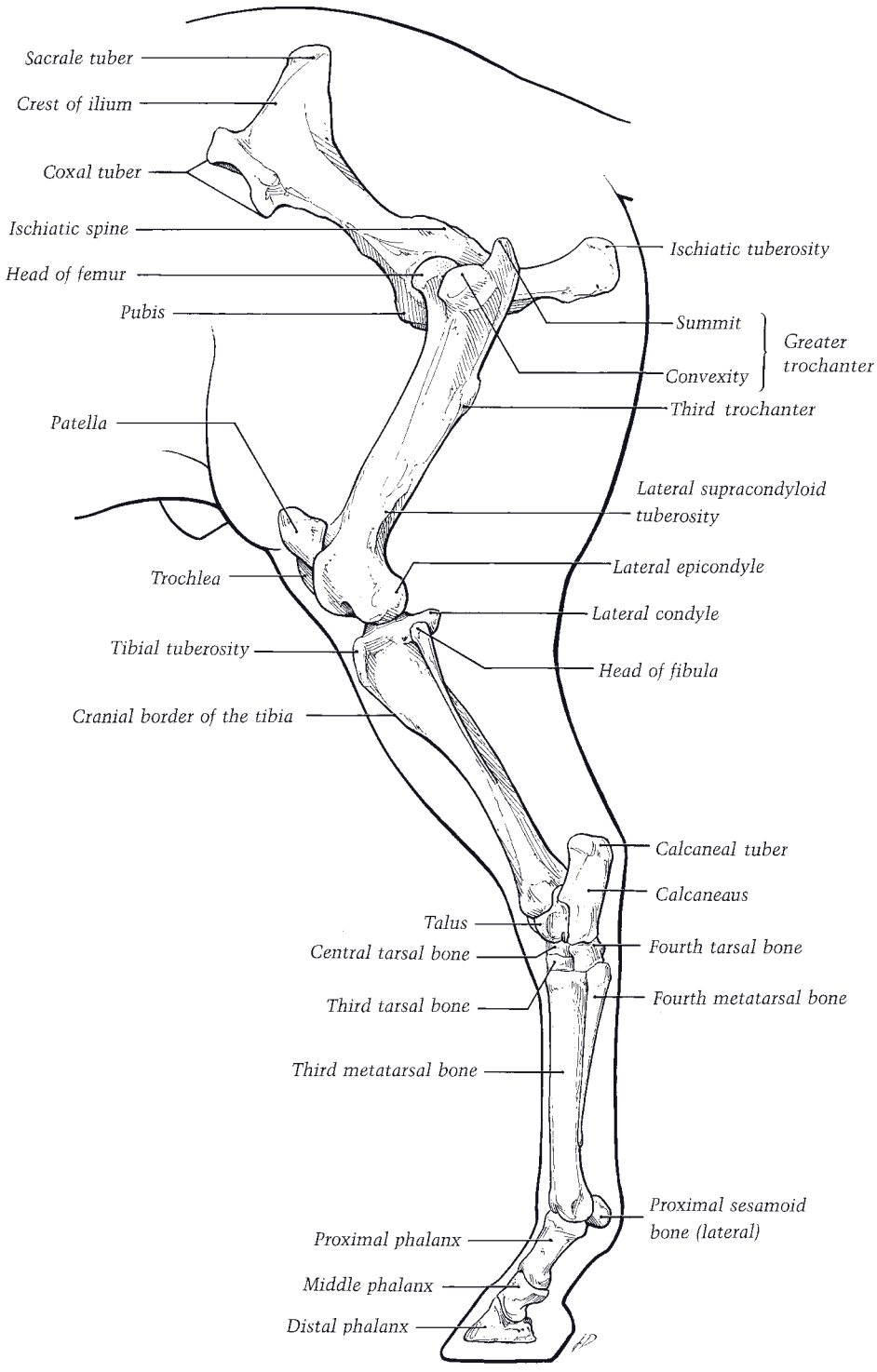


**Figure 1.29.** Bones of the left pelvic limb, lateral view.

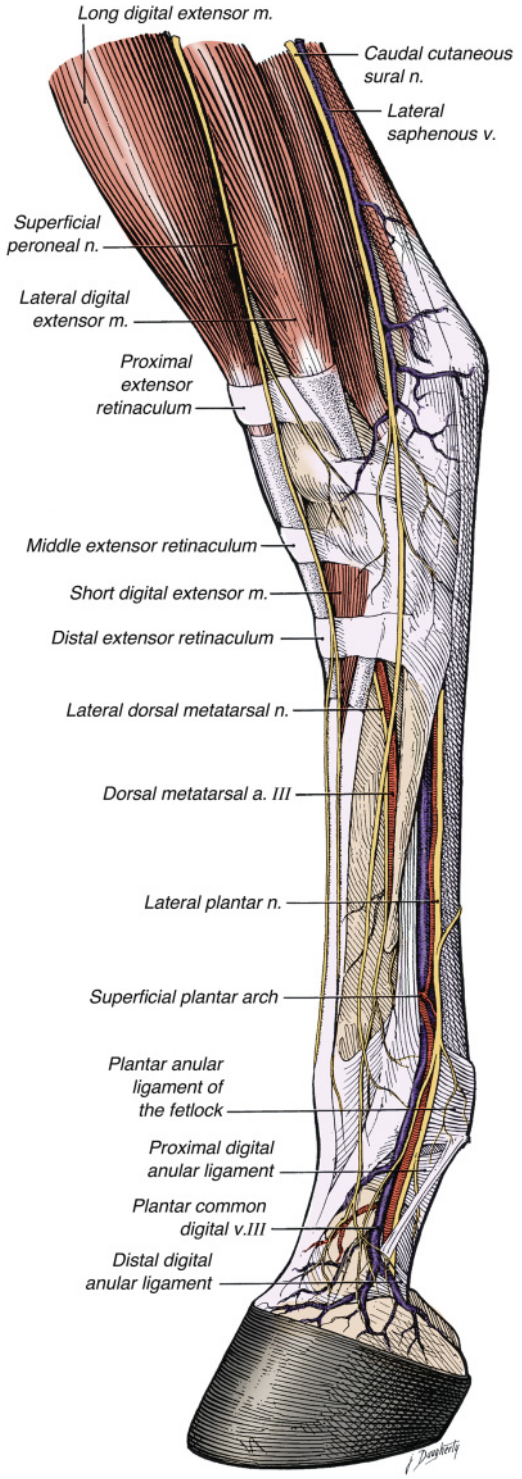


**Figure 1.30.** Lateral view of the left distal crus and pes. Please note that the term "fibularis" is currently preferred over "peroneus" (fibular rather than peroneal), although both are widely used.

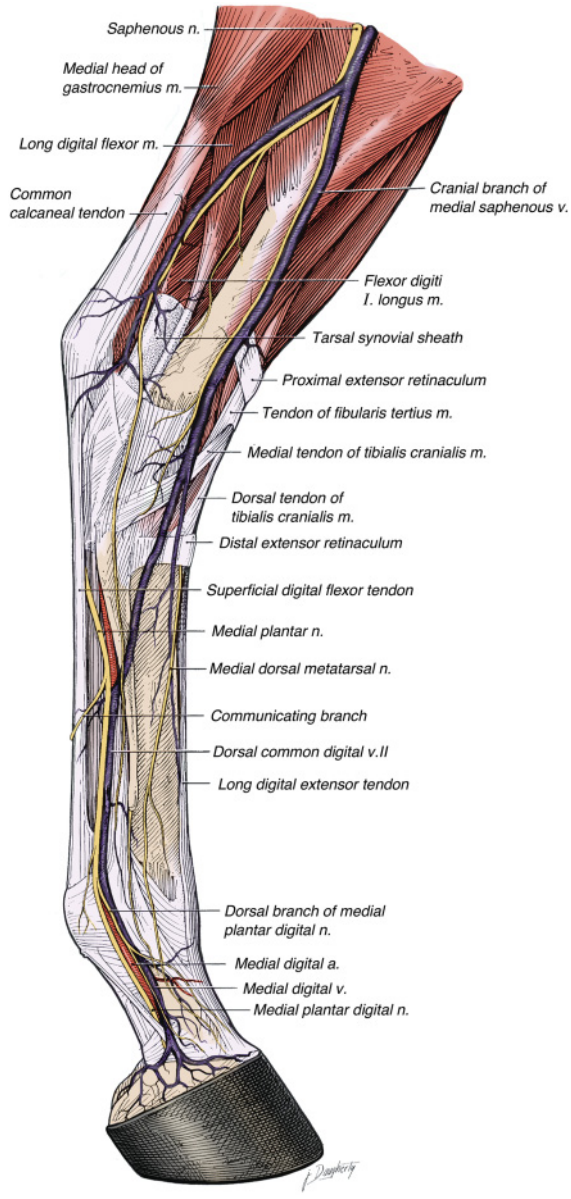


**Figure 1.31.** Medial view of the left distal crus and pes. Please note that the term "fibularis" is currently preferred over "peroneus" (fibular rather than peroneal), although both are widely used.

"inferior" check ligament) arises from the plantar aspect of the fibrous joint capsule of the hock. Longer than its counterpart in the forelimb, it joins the DDFT near the middle of the metatarsus. This slender accessory ligament may be absent in horses, and it is usually absent in mules and ponies.

The suspensory ligament (middle or third interosseous muscle) takes origin from a large area on the proximal aspect of the third metatarsal bone and a smaller attachment on the distal row of tarsal bones. Lying within the metatarsal groove deep to the DDFT, the suspensory ligament of the hindlimb is relatively thinner, more rounded, and longer than the ligament of the forelimb. In some horses, e.g., Standardbreds, the suspensory ligament of the hindlimb contains more muscle than the suspensory ligament of the forelimb. The two extensor branches pursue courses similar to those in the forelimb.

The neurovascular supply to the metatarsus is illustrated in Figures 1.30 and 1.31

### *Tarsus (Hock)*

The bones of the tarsus include the talus; calcaneus; and the central, first and second (fused), and third and fourth tarsal bones (Figure 1.29). Proximally, the trochlea of the talus articulates with the cochlear surface of the tibia in the tarsocrural joint; distally, the distal row of tarsal bones and the three metatarsal bones articulate in the tarsometatarsal joint. Extensive collateral ligaments span the latter two joints and the intertarsal joints. In the horse, nearly all of the movement of the hock arises from the tarsocrural joint.

#### Dorsal Aspect

The long digital extensor tendon's synovial sheath extends from the level of the lateral malleolus distad nearly to the junction of the tendon with the tendon of the lateral digital extensor muscle (Figure 1.30). The long digital extensor tendon is located just lateral to the palpable medial ridge of the trochlea of the talus. The proximal part of the short digital extensor muscle covers the tarsal joint capsule (Figure 1.30). As it crosses the dorsal surface of the tarsocrural joint, the tendon of the fibularis (peroneus) tertius muscle is superficial to the tendon of the tibialis cranialis muscle (Figures 1.31, 1.32). Then the tendon of the fibularis tertius forms a sleeve-like cleft through which the tendon of the tibialis cranialis and its synovial sheath pass. The latter tendon then bifurcates into a dorsal tendon, which inserts on the large metatarsal bone, and a medial ("cunean") tendon, which angles mediodistad under the superficial layer of the long medial collateral ligament, to insert on the first tarsal bone. A bursa is interposed between the cunean tendon and the long medial collateral ligament (Figure 1.33).

After forming the cleft that admits passage of the tibialis cranialis tendon, the fibularis tertius divides into two tendons. The dorsal tendon passes under the cunean tendon and inserts on the third tarsal and metatarsal bones, medial to the dorsal tendon of the cranial tibial muscle (Figure 1.33). The lateral tendon of the fibularis tertius extends distad deep to the long digital extensor tendon and continues laterad distal to the lateral ridge of the trochlea of the talus. The lateral tendon then bifurcates and inserts on the calcaneus and the fourth tarsal bone.

#### Lateral Aspect

The tendon of the lateral digital extensor muscle is bound by a fibrous band in a groove in the lateral malleolus of the tibia and then traverses a passage through the long lateral collateral ligament of the tarsus as the tendon angles dorsodistad. A synovial sheath enfolds the tendon from just proximal to the lateral malleolus to a point just proximal to the tendon's junction with the long digital extensor tendon. Plantar to the lateral extensor tendon, the lateroplantar pouch of the tarsocrural joint capsule protrudes between the lateral malleolus and the calcaneus.

#### Medial Aspect

A palpable feature of the medial aspect of the hock is the medial tendon of the tibialis cranialis muscle (colloquially called the "cunean tendon") as it inserts on the first tarsal bone. The bursa between the cunean tendon and the distal part of the long medial collateral ligament of the tarsus is not normally palpable (Figure 1.33). The tendon of the medial digital flexor (medial head of the deep digital flexor muscle, sometimes called long digital flexor muscle) passes through a fascial tunnel plantar to the medial collateral ligament (Figure 1.34). A compartment of the tarsocrural joint capsule, the medioplantar pouch, is located a short distance plantar to the medial digital flexor tendon and proximal to the sustentaculum tail of the calcaneus at the level of the medial malleolus. The tarsal fascia thickens into a flexor retinaculum, bridging the groove

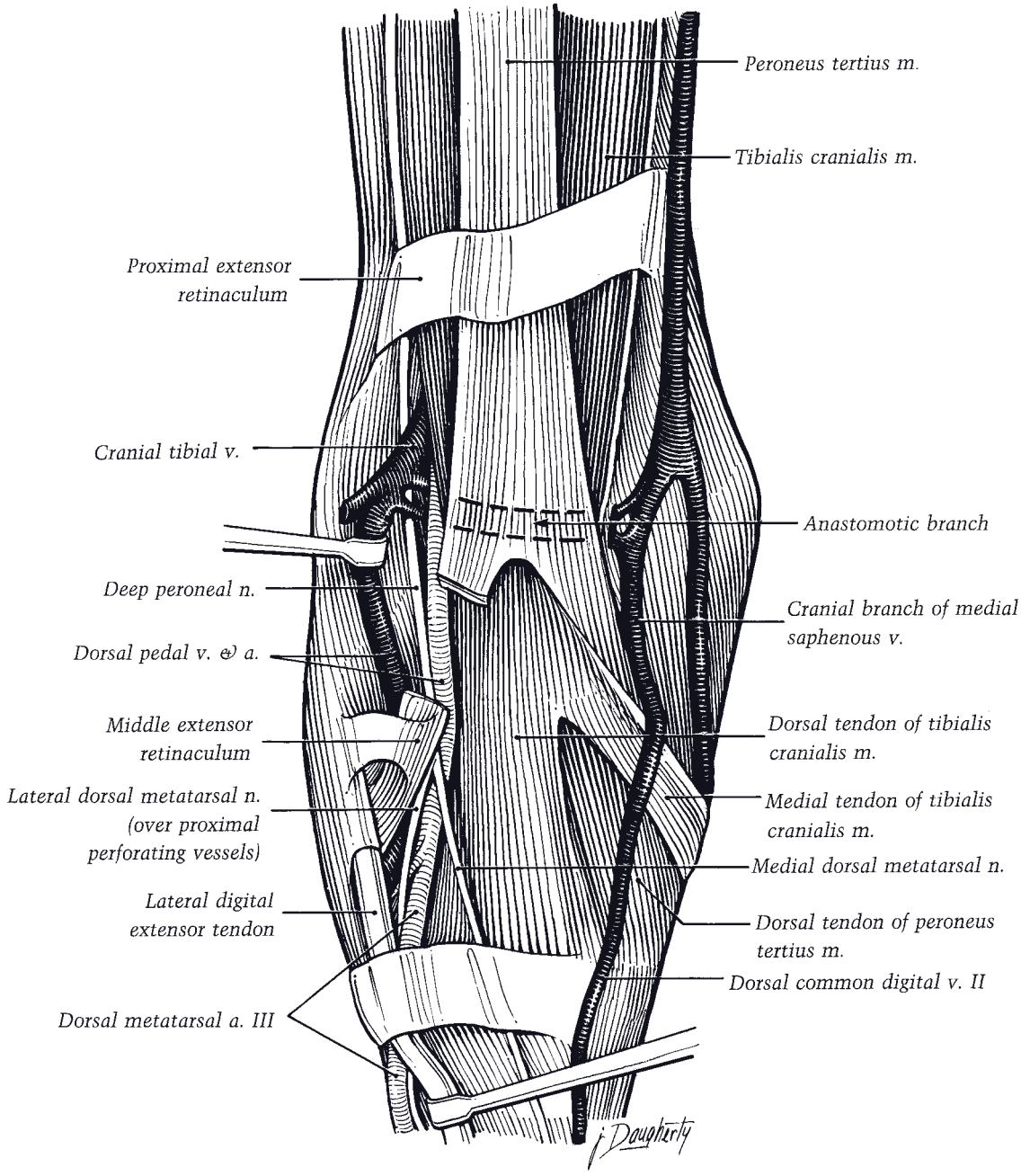


**Figure 1.32.** Dorsal dissection of the right tarsus. The long digital extensor and short digital extensor muscles have been removed. The lateral tendon of the fibularis (peroneus) tertius is sectioned. Please note that the term "fibularis" is currently preferred over "peroneus" (fibular rather than peroneal), although both are widely used.

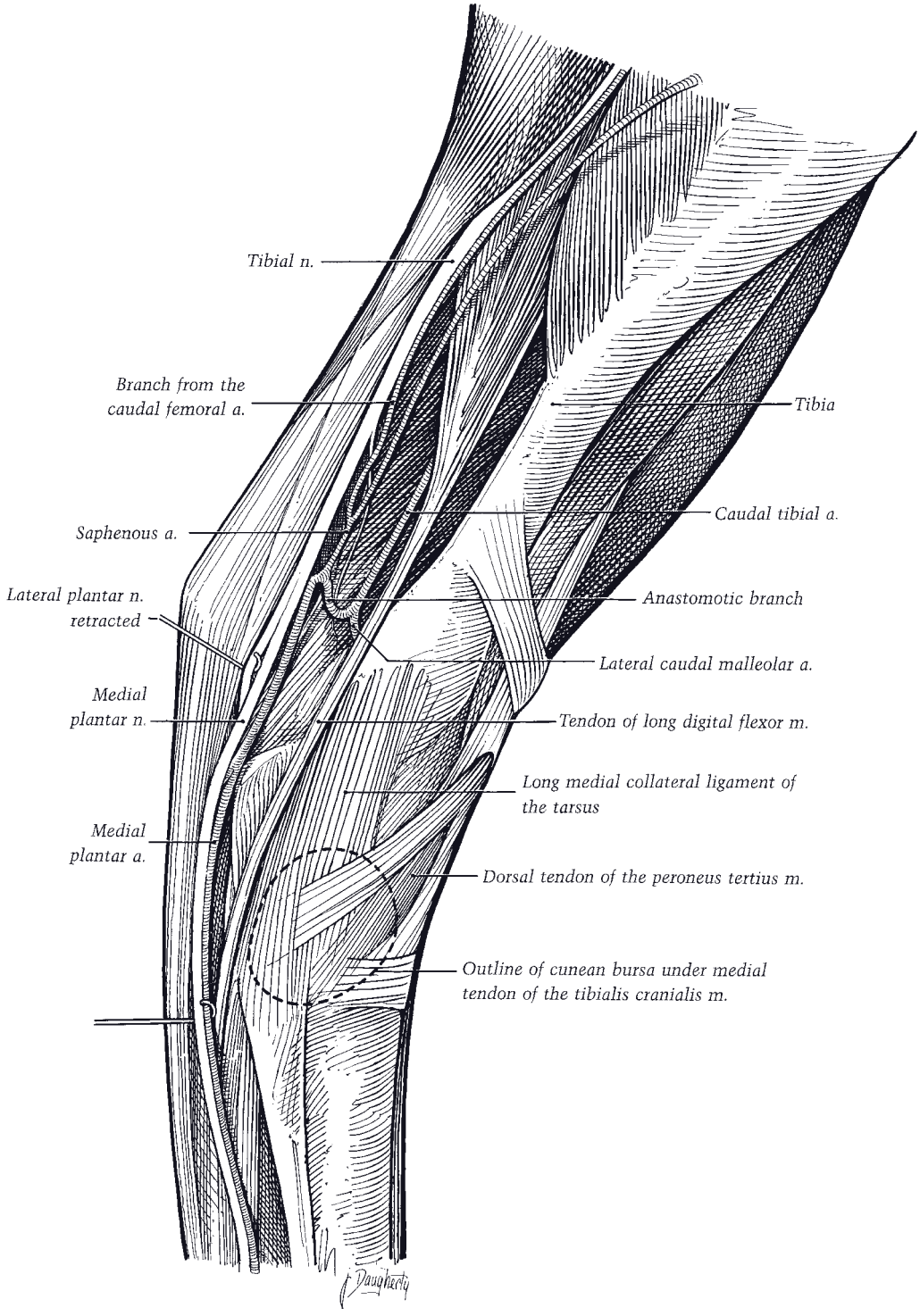


**Figure 1.33.** Medial dissection of the left distal crus, tarsus, and metatarsus, medial view. Please note that the term "fibularis" is currently preferred over "peroneus" (fibular rather than peroneal), although both are widely used.

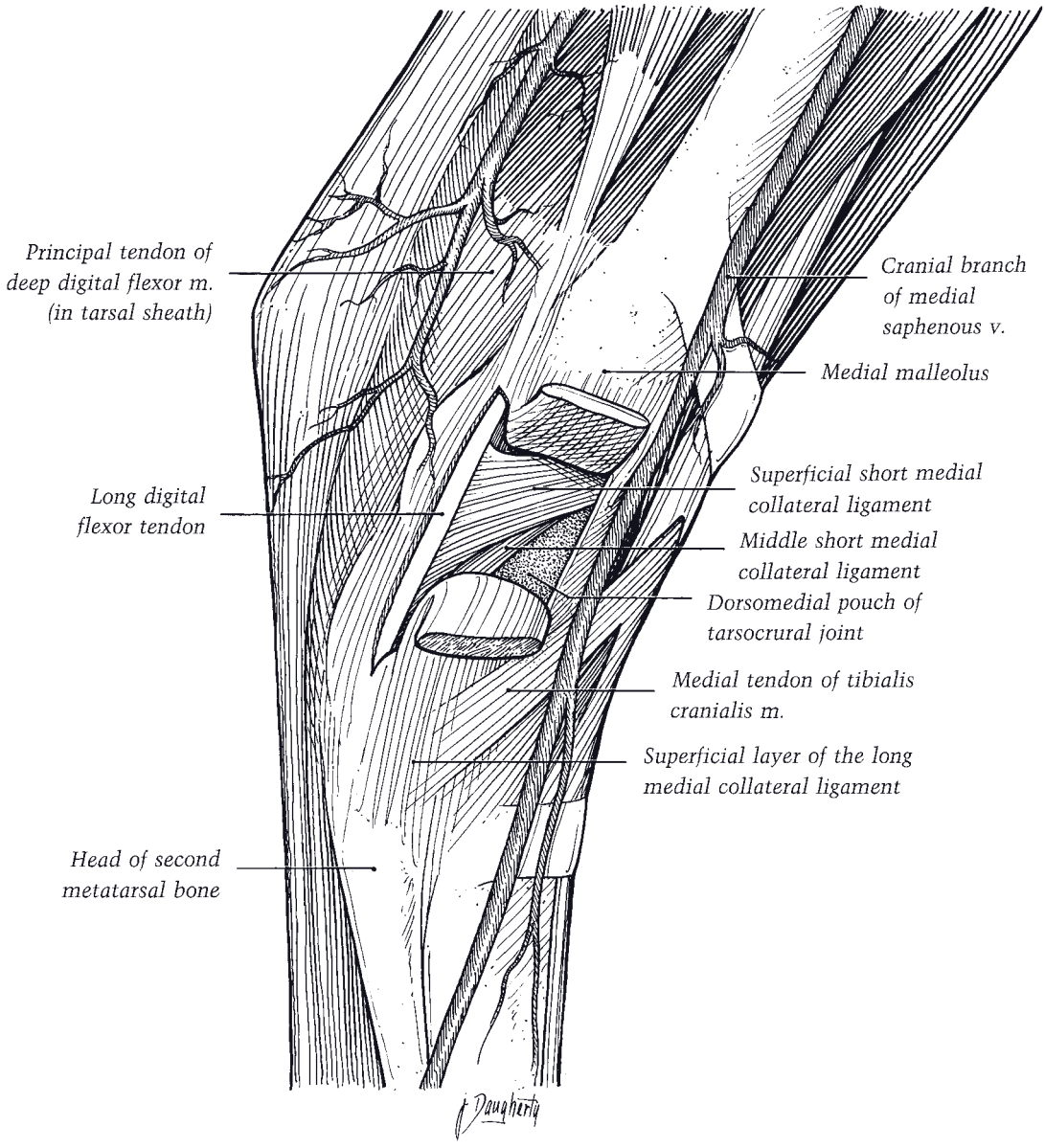


**Figure 1.34.** Medial view of the left tarsus. The long medial collateral ligament has been cut and reflected. The tendon sheath of the long digital flexor tendon has been opened. Please note that "long digital flexor" is an older term for the medial head of the deep digital flexor muscle.

on the sustentaculum tail of the calcaneus to form the tarsal canal containing the principal tendon of the deep digital flexor muscle. The tendon's synovial sheath, the tarsal sheath, extends from a level proximal to the medial malleolus to the proximal fourth of the metatarsus (Figure 1.31).

## Plantar Aspect

In the distal third of the crus, the tendon of the superficial digital flexor muscle curls around the medial side of the tendon of the gastrocnemius muscle to become superficial as the tendons approach the calcaneal tuber. The SDFT flattens

and is joined by aponeurotic connections of the biceps femoris and semitendinosus muscles. This tendinous complex attaches to the point and sides of the calcaneal tuber. The tendon proper of the superficial digital flexor then narrows and continues distad superficial to the long plantar ligament. The calcaneal tendon of the gastrocnemius lies deep to the superficial digital flexor at the hock and inserts on the plantar surface of the calcaneal tuber. An elongated bursa is interposed between the two tendons just above the tarsus. A smaller bursa is present between the SDFT and the calcaneal tuber. These two bursae usually communicate across the lateral surface of the gastrocnemius tendon. An inconstant subcutaneous bursa may develop over the superficial digital flexor at the level of the calcaneal tuber. Dorsolateral to the superficial digital flexor, the long plantar ligament is attached to the plantar surface of the calcaneus, terminating distally on the fourth tarsal and metatarsal bones.

### *Tarsal Joint (Hock Joint)*

The principal component of the composite tarsal joint is the tarsocrural joint. Deep grooves of the cochlear articular surface of the distal end of the tibia articulate with the surface of the trochlea of the talus at an angle of 12° to 15° dorsolateral to the limb's sagittal plane. The interarticular and tarsometatarsal joints are plane joints capable of only a small amount of gliding movement. In the standing position, the dorsal articular angle of the hock is around 150°. During flexion of the tarsocrural joint, the pes is directed slightly laterad due to the configuration of the joint.

A long collateral ligament and three short collateral ligaments bind each side of the equine tarsus (Figures 1.34 and 1.35). The long lateral collateral ligament extends from the lateral malleolus caudal to the groove for the tendon of the lateral digital extensor, attaching distally to the calcaneus, fourth tarsal bone, talus, and the third and fourth metatarsal bones. The three short lateral collateral ligaments are fused proximally where they attach to the lateral malleolus cranial to the groove for the lateral digital extensor tendon. The superficial component, its fibers spiraling 180°, attaches distoplantarly to both the talus and calcaneus, whereas the middle and deep short lateral collateral ligaments attach solely on the lateral surface of the talus.

The long medial collateral ligament of the hock has less well-defined borders than its lateral counterpart. From its proximal attachment on the medial malleolus cranial to the groove for the long digital flexor muscle, the long medial collateral ligament extends distad and divides into two layers along its dorsal border. The superficial layer goes over the cunean tendon of the tibialis cranialis muscle and attaches to the fused first and second tarsal bones and the second and third metatarsal bones just distal to the distal edge of the cunean bursa. The deep layer attaches distally to the distal tuberosity of the talus and the central and third tarsal bones. The plantar edge of the ligament attaches to the deep fascia over the sustentaculum tail and the interosseous ligament between the second and third metatarsal bones.

The flat superficial short medial collateral ligament extends from the medial malleolus to the tuberosities of the talus and the ridge between them (Figure 1.34). The middle short medial collateral ligament extends obliquely from the medial tibial malleolus to the sustentaculum tali and central tarsal bone. It lies on the medial surface of the talus between the two tuberosities, varying in position during movement of the joints. The smallest component, the deep short medial collateral ligament, courses from the distal edge of the medial tibial malleolus obliquely to the ridge between the two tuberosities of the talus.

A dorsal tarsal ligament fans out distad from the distal tuberosity of the talus and attaches to the central and third tarsal bones and the proximal extremities of the second and third metatarsal bones (Figure 1.35). A plantar tarsal ligament attaches to the plantar surface of the calcaneus and fourth tarsal bone and the fourth metatarsal bone. Smaller, less distinct ligaments join contiguous tarsal bones.

The tarsal joint capsule is thinnest dorsally and thickest in its plantar and distal parts. Three pouches can protrude (most notably with joint effusion) from the large tarsocrural synovial sac where it is not bound down by ligaments: the dorsomedial (largest), medioplantar, and lateroplantar pouches. This large synovial space consistently communicates with the synovial sac associated with the proximal intertarsal joint formed by the talus and calcaneus proximally and the central and fourth tarsal bones distally. The distal intertarsal sac, between the central tarsal and contiguous bones and the distal tarsal row, typically does not communicate with the proximal intertarsal sac, but may communicate with the synovial sac of the tarsometatarsal joint.

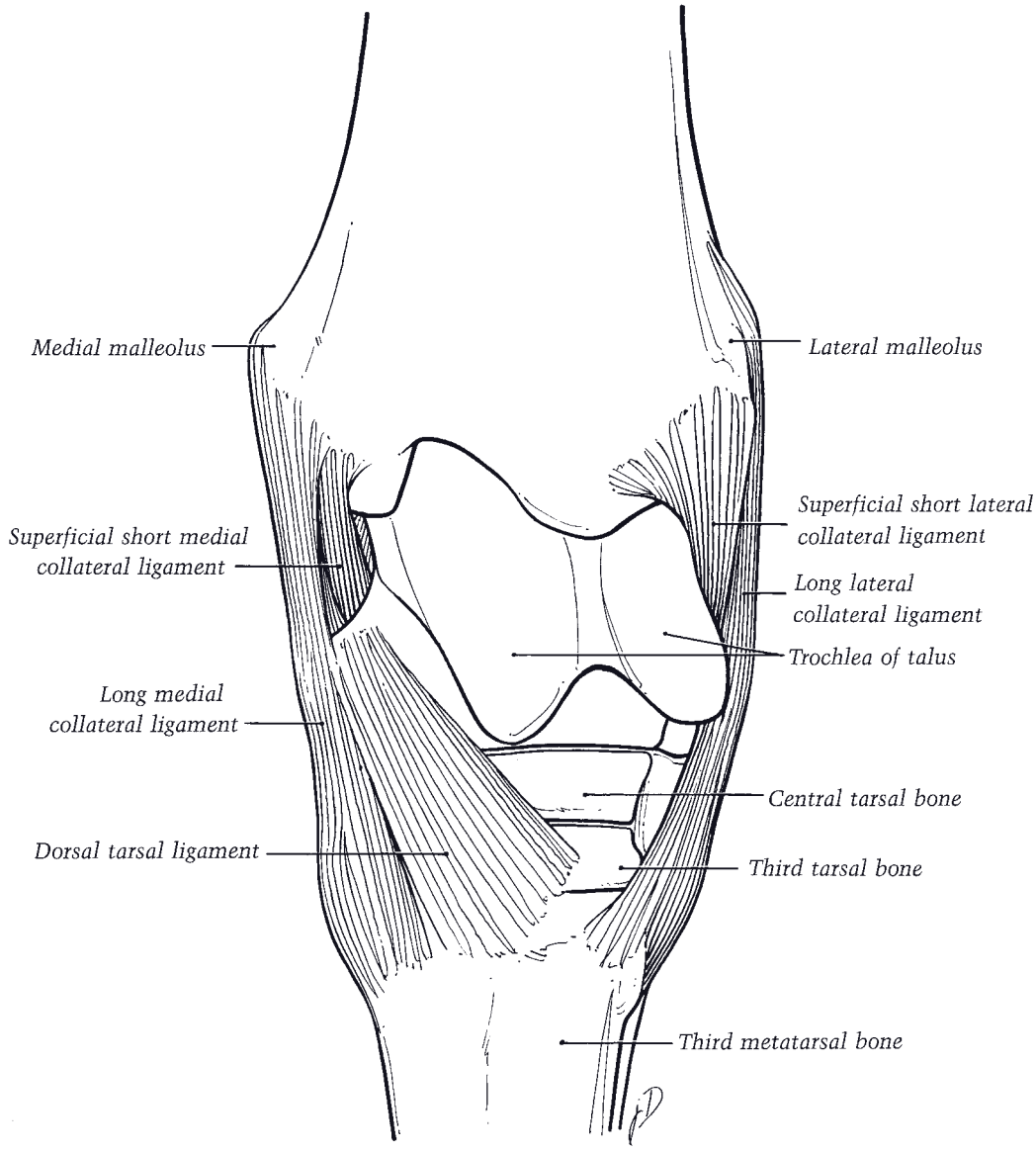


**Figure 1.35.** Dorsal view of the right tarsus.

## Movements of the Tarsocrural Joint

The tarsocrural joint is flexed by contraction of the tibialis cranialis muscle and the passive pull of the tendinous fibularis (peroneus) tertius muscle. Contraction of the gastrocnemius, biceps femoris, and semitendinosus muscles and the passive pull of the tendinous superficial digital flexor muscle extends the joint. By virtue of its attachments in the extensor fossa of the femur proximally, and on the lateral aspect of the tarsus and dorsal surface of the third metatarsal bone distally, the fibularis tertius passively flexes the tarsocrural joint when the stifle joint is flexed. The superficial digital flexor muscle originates in the supracondyloid fossa of the femur and attaches to the calcaneal tuber. This part of the superficial digital flexor serves to passively extend the tarsocrural joint when the femorotibial joint is extended. The two tendinous, passively functioning muscles constitute the reciprocal apparatus (Figure 1.36).

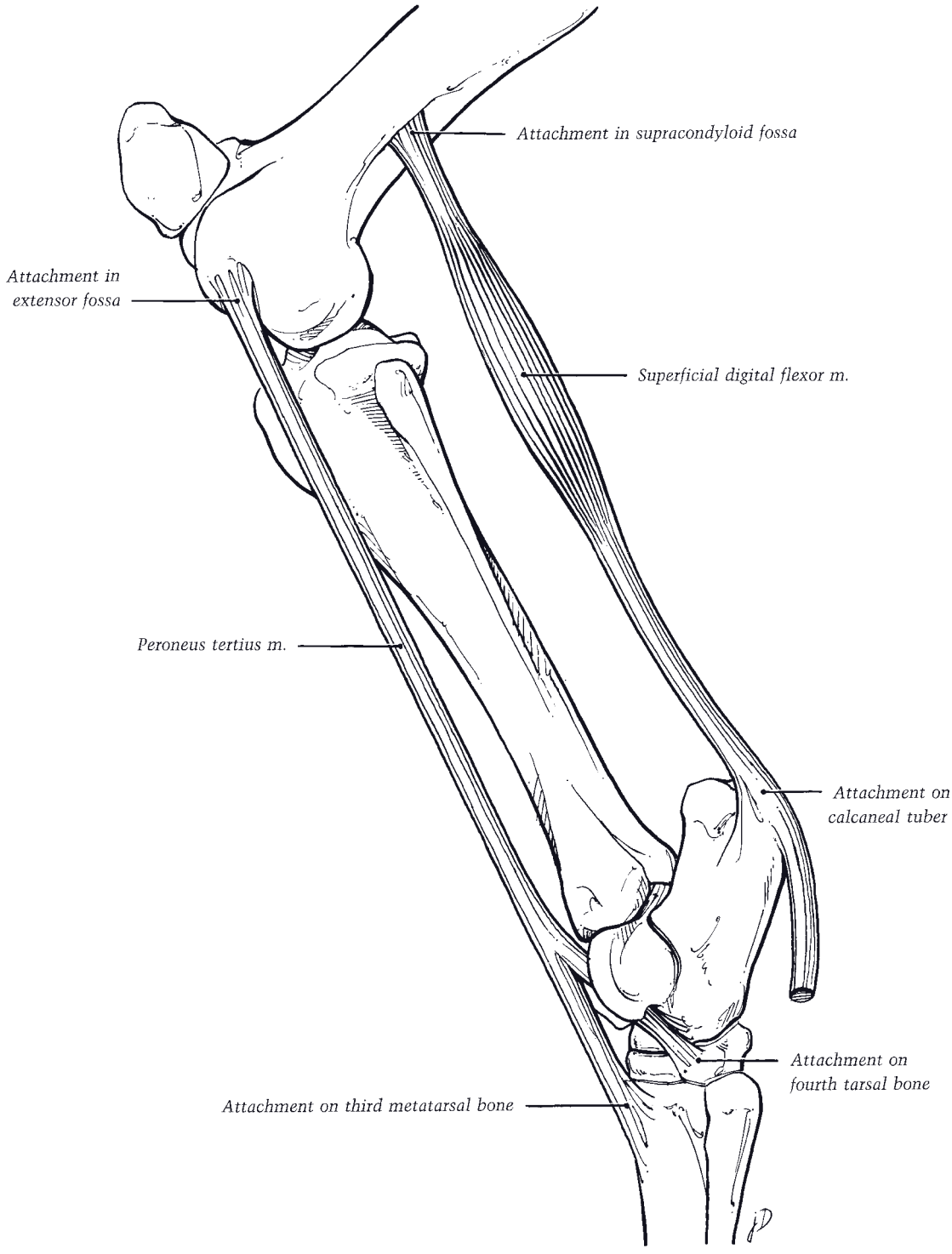


**Figure 1.36.** Reciprocal apparatus, lateral view of left hindlimb. Please note that the term "fibularis" is currently preferred over "peroneus" (fibular rather than peroneal), although both are widely used.

The neurovascular supply to the tarsus is illustrated in Figures 1.30, 1.31 and 1.32.

## *Crus (Leg or Gaskin)*

The crus or true leg is the region of the hindlimb containing the tibia and fibula. Thus, it extends from the tarsocrural joint to the femorotibial joints. The transversely flattened proximal end of the fibula articulates with the lateral condyle of the tibia. Distally, the fibula narrows to a free end, terminating in the distal one-half to two-thirds of the crus as a thin ligament. An interosseous ligament occupies the space between the two bones. The cranial tibial vessels pass through the proximal part of the ligament. It should be noted that the current preference among anatomists is to replace the Greek word "peroneus" with its Latin equivalent "fibularis" in the naming of crural structures.

Beneath the skin and superficial fascia a heavy crural fascia invests the entire crural region. The superficial layer of the deep crural fascia is continuous with the femoral fascia; the middle layer is continuous with tendons descending from the thigh. The two layers are inseparable in several places. The crural fascia blends with the medial and lateral patellar ligaments and attaches to the medial tibia in the middle of the leg. Caudally, the crural fascia forms the combined aponeuroses of the biceps femoris and semitendinosus muscles that attach with the SDFT to the calcaneal tuber. Under the two common fasciae a deeper layer covers the muscles of the leg.

### Cranial Aspect

The belly of the long digital extensor muscle is prominent beneath the skin on the craniolateral aspect of the crus. It originates in common with the fibularis tertius from the extensor fossa of the femur, the common tendon descending through the extensor sulcus of the tibia (Figure 1.37). The long digital extensor muscle is related deeply to the tendinous fibularis tertius and the fleshy cranial tibial muscles, and caudally to the lateral digital extensor muscle from which it is separated by a distinct intermuscular septum. Deep to and intimately associated with the fibularis tertius, the cranial tibial muscle covers the craniolateral surface of the tibia, originating from the tibial tuberosity, lateral condyle, and lateral border, and from the crural fascia (Figure 1.37). The superficial fibular nerve courses distad in the groove between the digital extensor muscles and angles craniad toward the hock.

The deep fibular nerve courses distad between the two muscles on the cranial surface of the intermuscular septum. At its origin, this nerve sends branches to the digital extensor muscles and the fibularis tertius and tibialis cranialis muscles.

### Lateral Aspect

The tibial attachment of the biceps femoris muscle, a broad aponeurosis, sweeps across the proximal third of the lateral aspect of the crus to attach to the cranial border of the tibia. Deep to the belly of the biceps femoris, the common fibular nerve crosses the lateral surface of the lateral head of the gastrocnemius muscle and divides into superficial and deep fibular nerves (Figure 1.38). Caudal to these, the lateral digital extensor muscle extends distad from its origins on the fibula, interosseus ligament, lateral surface of the tibia, and lateral collateral ligament of the femorotibial joint. The lateral head of the deep digital flexor muscle lies caudal to the belly of the lateral digital extensor.

The lateral head of the gastrocnemius originates on the lateral supracondyloid tuberosity of the femur. Under the crural fascia in the proximal half of the crus, the small soleus muscle extends from its origin on the fibula along the lateral aspect of the gastrocnemius muscle to join the gastrocnemius tendon (Figure 1.38).

### Caudal Aspect

Descending from their origins on the supracondyloid tuberosities of the femur, the two heads of the gastrocnemius enclose the round, mostly tendinous superficial digital flexor. The tendon of the latter wraps medially from deep to superficial around the gastrocnemius tendon in the distal third of the crus. The deep digital flexor muscle possesses three heads with a variety of names that appear in anatomical texts (Figure 1.38). In the horse, the tendons of all three heads unite with the main deep digital flexor tendon. In the distal third of the crus, the flat tendon of the caudal tibial (the superficial head) joins the larger tendon of the lateral head, whereas the tendon of the medial head (medial digital flexor muscle) pursues its course over the medial aspect of the hock to join the principal tendon in the metatarsus (Figure 1.34).

## *Stifle (Genu)*

The stifle is the region including the stifle joint (femorotibial joints plus the femoropatellar joint) and surrounding structures.

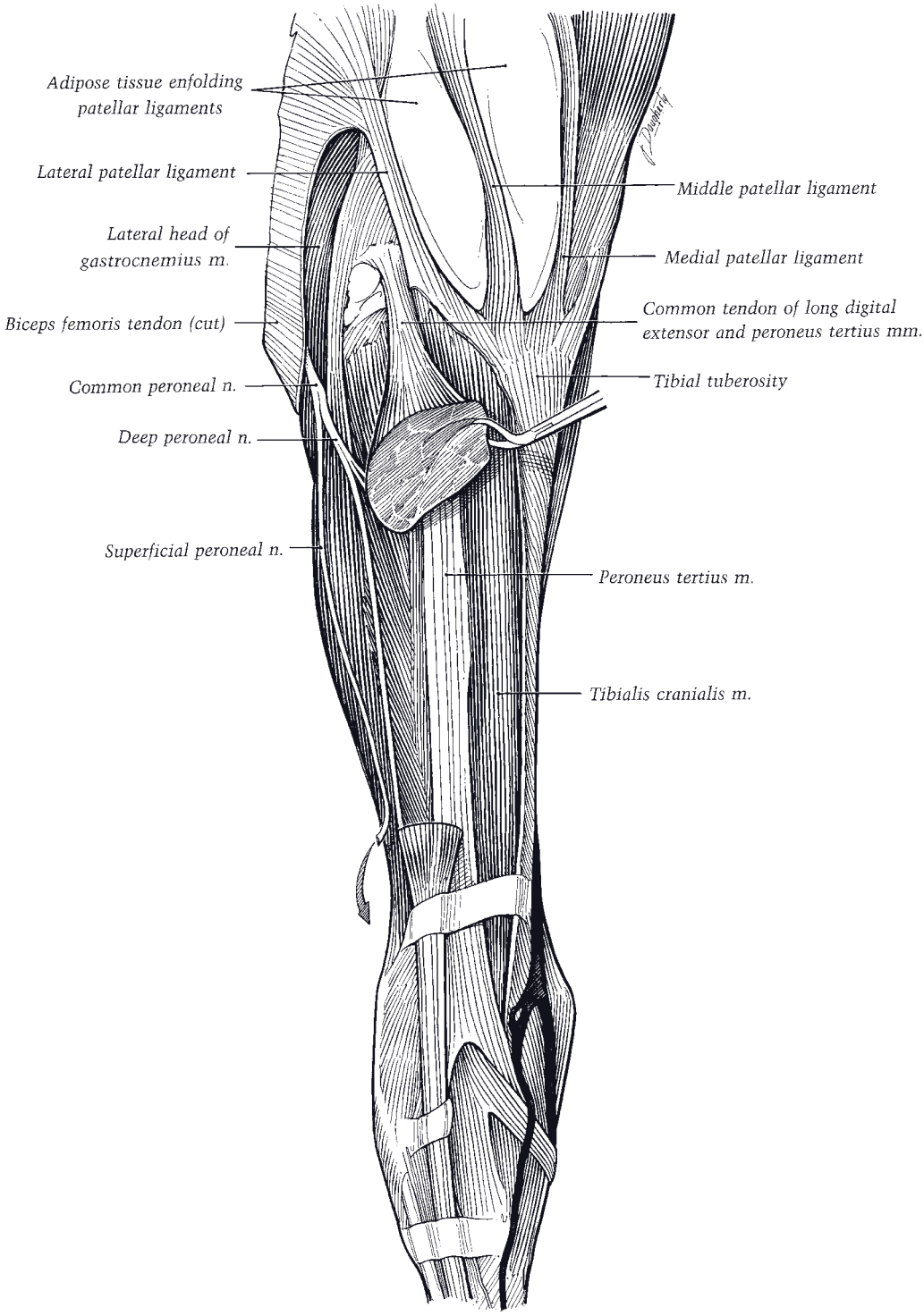


**Figure 1.37.** Dorsal view of the right stifle, crus, and tarsus. The long digital extensor muscle belly has been removed, along with the terminal parts of the superficial fibular (peroneal) nerve (arrow). Please note that the term "fibularis" is currently preferred over "peroneus" (fibular rather than peroneal), although both are widely used.

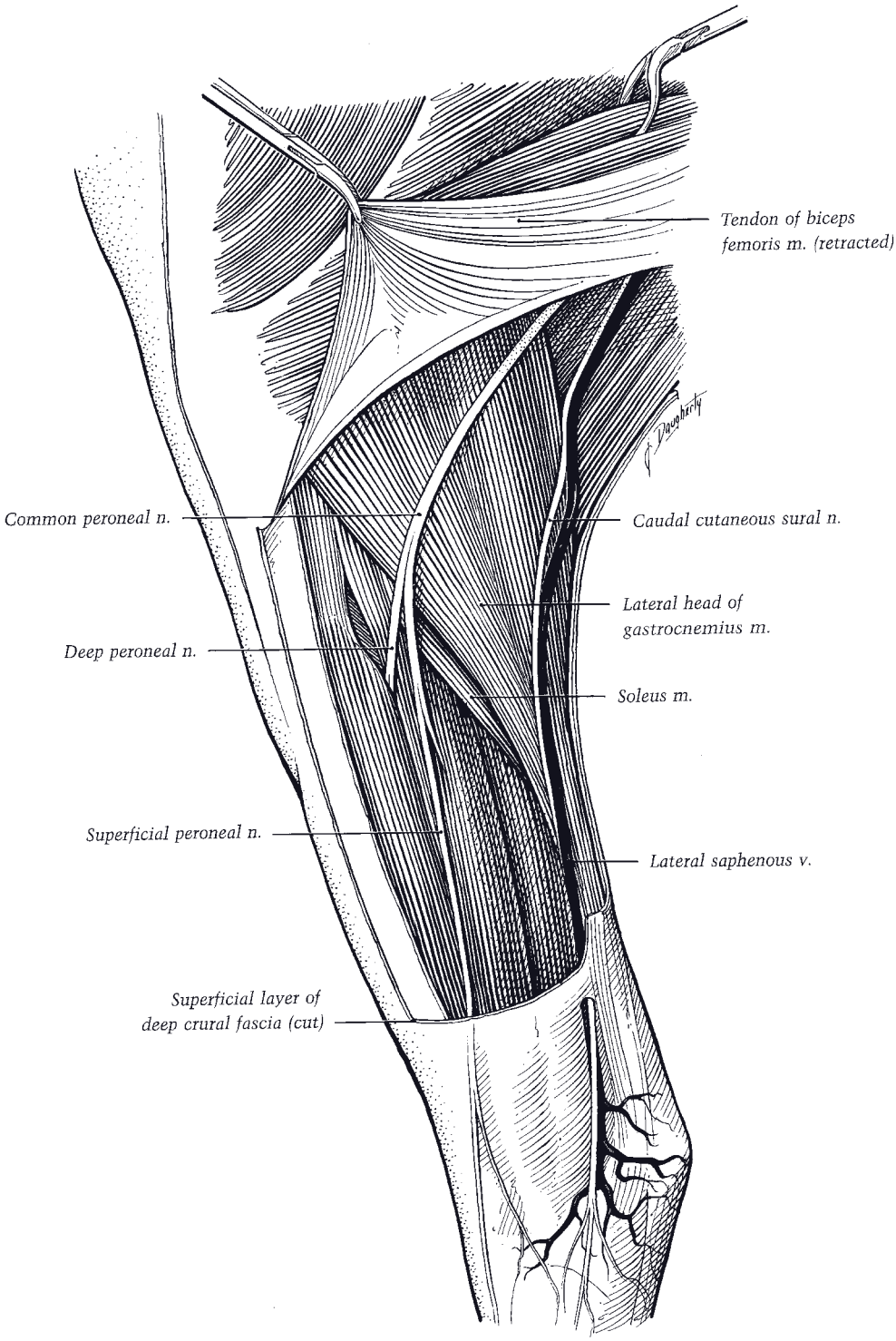


**Figure 1.38.** Superficial dissection of lateral aspect of left stifle, crus, and tarsus. Please note that the term "fibularis" is currently preferred over "peroneus" (fibular rather than peroneal), although both are widely used.

### Cranial Aspect

Deep to the skin, three patellar ligaments descend from the patella, converging to their attachments on the tibial tuberosity. An extensive pad of adipose tissue is interposed between the ligaments and the joint capsule of the femoropatellar joint (Figure 1.37). The adipose tissue enfolds the ligaments, wrapping around their sides. The space between the medial and middle patellar ligaments is greater than the space between the middle and lateral ligaments. This difference reflects the origin of the medial patellar ligament from the parapatellar fibrocartilage. This is a large mass extending mediad from the patella in such a manner that its continuation, the medial patellar ligament, courses proximal and then medial to the medial ridge of the trochlea on the femur. The medial patellar ligament attaches to the medial side of the tibial tuberosity. As it descends from the patella to its insertion, two bursae lie under the middle patellar ligament, one between the proximal part of the ligament and the apex of the patella, and the other between the ligament and the proximal part of the groove. Inclining mediad from the lateral aspect of the cranial surface of the patella, the lateral patellar ligament serves as an attachment for a tendon from the biceps femoris muscle and then for the fascia lata just before the ligament attaches to the lateral aspect of the tibial tuberosity. The tendon from the biceps femoris continues on to the cranial surface of the patella.

The base, cranial surface, and medial border of the patella, and the parapatellar fibrocartilage and femoropatellar joint capsule, serve as attachments for the insertions of the quadriceps femoris muscle.

### Lateral Aspect

The insertional parts of the biceps femoris muscle and, caudally, the semitendinosus muscle, dominate the lateral aspect of the stifle region. The tendon from the cranial division of the biceps femoris inserts on the lateral patellar ligament and the patella, and the tendon from the middle division of the muscle sweeps craniodistad to the cranial border of the tibia.

Reflection of the distal part of the biceps femoris muscle reveals the following (Figure 1.39): The lateral femoropatellar ligament extends obliquely from the lateral epicondyle of the femur to the lateral border of the patella. The lateral surface of the lateral head of the gastrocnemius muscle is crossed by the common fibular nerve and, further caudad, by the caudal cutaneous sural nerve and the lateral saphenous vein carrying blood to the caudal femoral vein. As it extends from the lateral epicondyle of the femur to the head of the fibula, the thick lateral collateral ligament of the femorotibial joint covers the tendon of origin of the popliteus muscle that also originates from the lateral epicondyle. A pouch from the lateral femorotibial joint capsule lies beneath the tendon. A common tendon of the long digital extensor and fibularis (peroneus) tertius takes origin from the extensor fossa in the distal surface of the lateral epicondyle of the femur. The tendon is cushioned as it extends distad by an elongated pouch from the lateral femorotibial joint capsule.

### Caudal Aspect

Under the skin and fascia on the caudal aspect of the stifle (supplied by branches of the caudal femoral nerve) the caudal part of the biceps femoris muscle covers the lateral head of the gastrocnemius, the tibial tendon of the biceps femoris going to the cranial border of the tibia, its tarsal tendon continuing distad. The semitendinosus muscle sweeps to its insertion on the cranial border of the tibia and distad toward its tarsal insertion, covering the medial head of the gastrocnemius. The tendons of the smaller medial head and larger lateral head of the gastrocnemius combine and, at first, the tendon lies superficial to the tendon of the superficial digital flexor muscle. Separation of the two heads of the gastrocnemius muscle reveals the tendinous superficial digital flexor muscle that arises in the supracondyloid fossa of the femur between the two heads, its initial part embedded in the lateral head (Figure 1.40).

The triangular popliteus muscle extends mediodistad from its origin on the lateral epicondyle of the femur (Figure 1.39). The tendon of origin passes deep to the lateral collateral ligament of the stifle joint, cushioned by an extension of the lateral pouch of the femorotibial joint capsule. The popliteus spreads out and inserts on the medial part of the caudal surface of the tibia proximal to the popliteal line, contacting the medial head of the deep digital flexor (Figure 1.40, 1.41).

### Medial Aspect

Cranially, the vastus medialis of the quadriceps femoris muscle attaches to the parapatellar

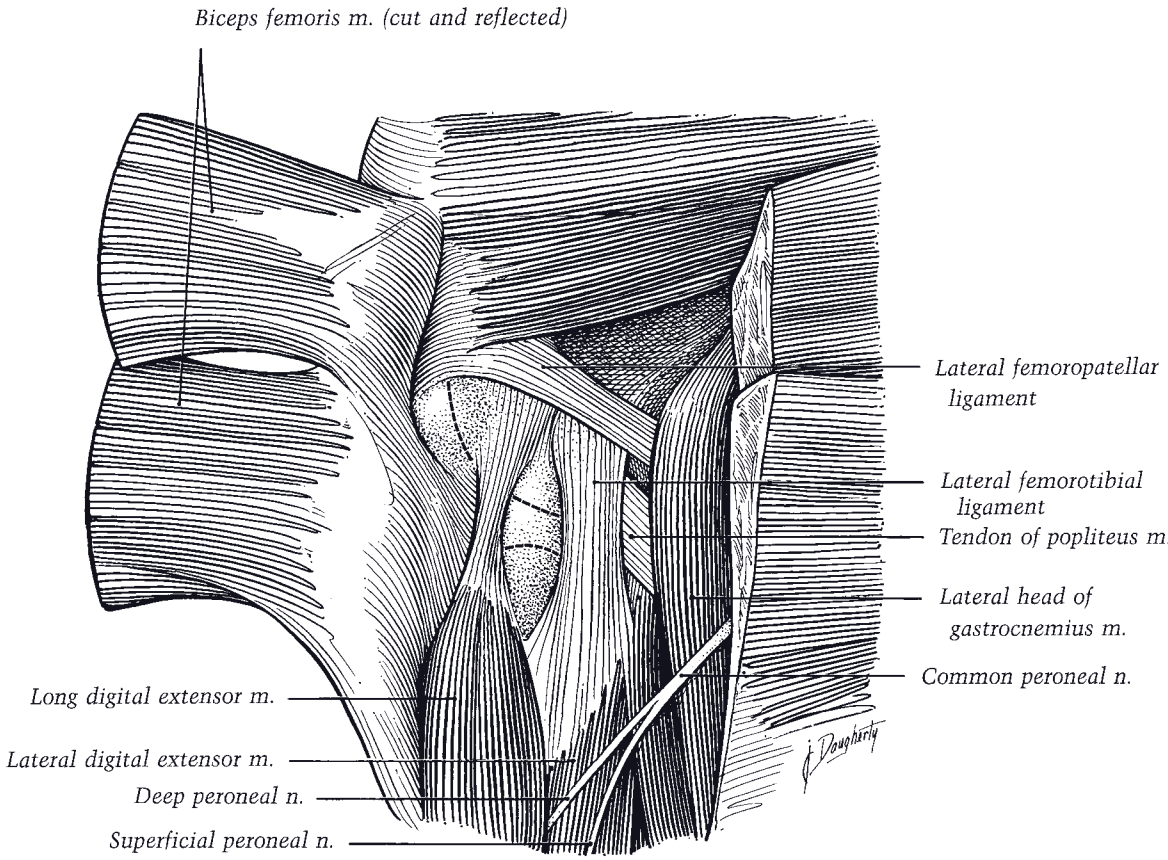


**Figure 1.39.** Deep dissection of the lateral aspect of the left stifle with femoral and tibial condylar surfaces indicated by dashed lines. Please note that the term "fibularis" is currently preferred over "peroneus" (fibular rather than peroneal), although both are widely used.

fibrocartilage, medial border of the patella, and medial patellar ligament. The straplike sartorius muscle attaches to the medial patellar ligament and the tibial tuberosity. Caudal to the sartorius, the gracilis muscle also attaches to the medial patellar ligament and to the medial collateral ligament of the femorotibial joint and the crural fascia (Figure 1.42).

A thinner medial collateral ligament of the femorotibial joint reaches from the medial epicondyle of the femur to just distal to the margin of the medial tibial condyle, detaching fibers to the medial meniscus (Figure 1.42). The adductor muscle inserts on the ligament and the medial epicondyle. The medial femoropatellar ligament is also thinner than its lateral counterpart, blending with the femoropatellar joint capsule as the ligament extends from the femur proximal to the medial epicondyle to the parapatellar fibrocartilage.

## *Stifle Joint*

The stifle is the "true knee." It comprises two joints, the femoropatellar and femorotibial joints, which together form a hinge joint. The synovial space of the stifle is significantly partitioned into three distinct sacs: the femoropatellar sac and the right and left femorotibial joint sacs.

### Femoropatellar Joint

The patella is a sesamoid bone intercalated in the termination of the quadriceps femoris muscle with the three patellar ligaments, constituting the tendon of insertion. A thin, voluminous joint capsule attaches peripheral to the edge of the femoral trochlea, with its patellar attachment close to the edge of the patellar articular surface. A large pouch from the joint capsule protrudes proximad under a mass of adipose tissue and the

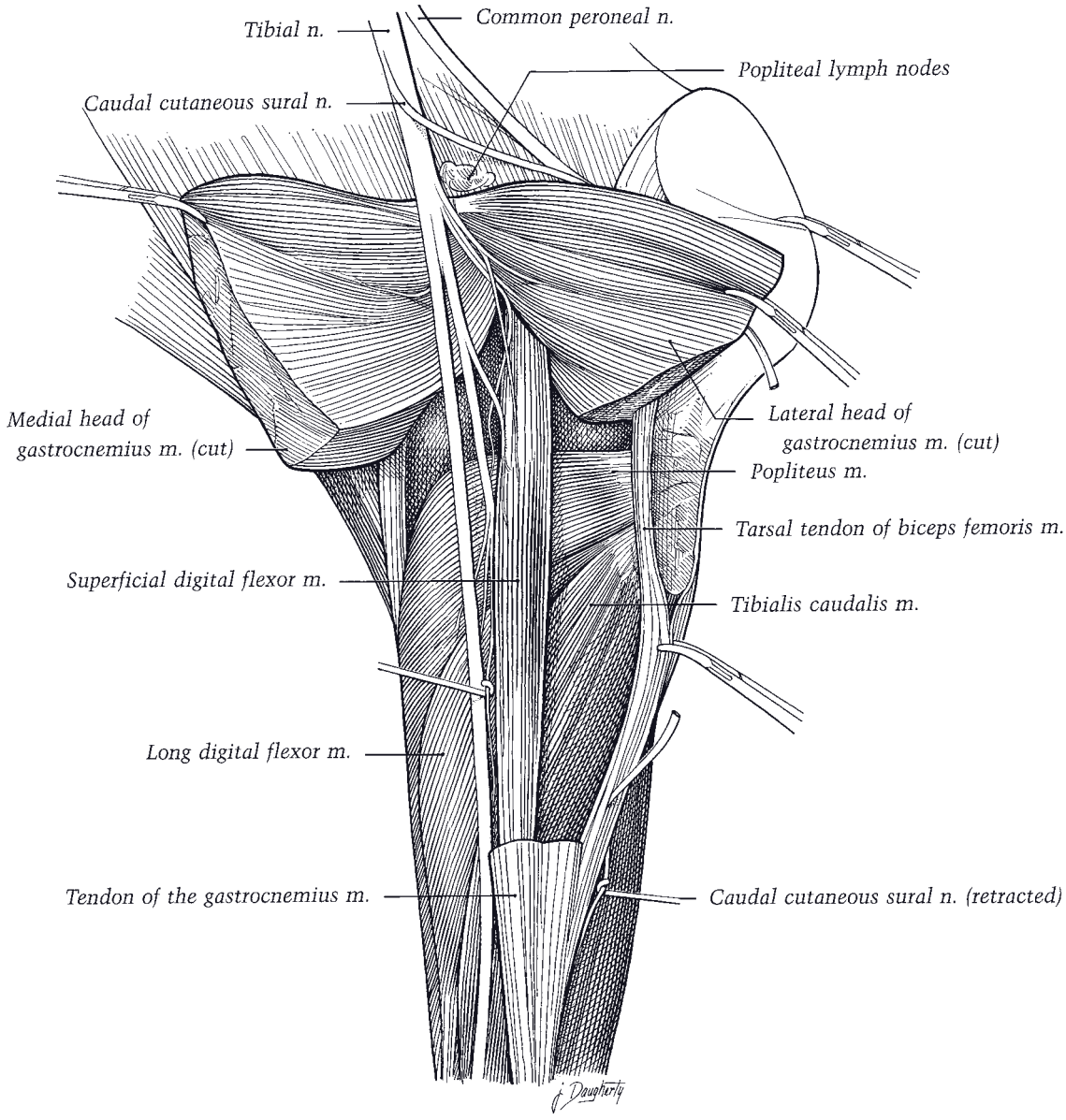


**Figure 1.40.** Dissection of the caudal aspect of the right stifle and crus. Please note that the term "fibularis" is currently preferred over "peroneus" (fibular rather than peroneal), although both are widely used.

distal part of the quadriceps femoris muscle. The distal extremity of the femoropatellar joint capsule contacts the femorotibial joint capsule. The vastus intermedius of the quadriceps femoris attaches, in part, to the femoropatellar joint capsule, acting to tense the capsule during extension of the femoropatellar joint.

The articular surface of the patella is much smaller than the trochlear surface of the femur; the larger gliding surface of the trochlea accommodates the proximal-distal movements of the patella. A wide groove separates the substantially larger medial ridge of the trochlea from the smaller, slightly more distal lateral ridge. Articular cartilage covers the entire large, rounded medial ridge; the cartilage covering the more regularly rounded lateral ridge extends only part way over the lateral surface. Contact between the patella

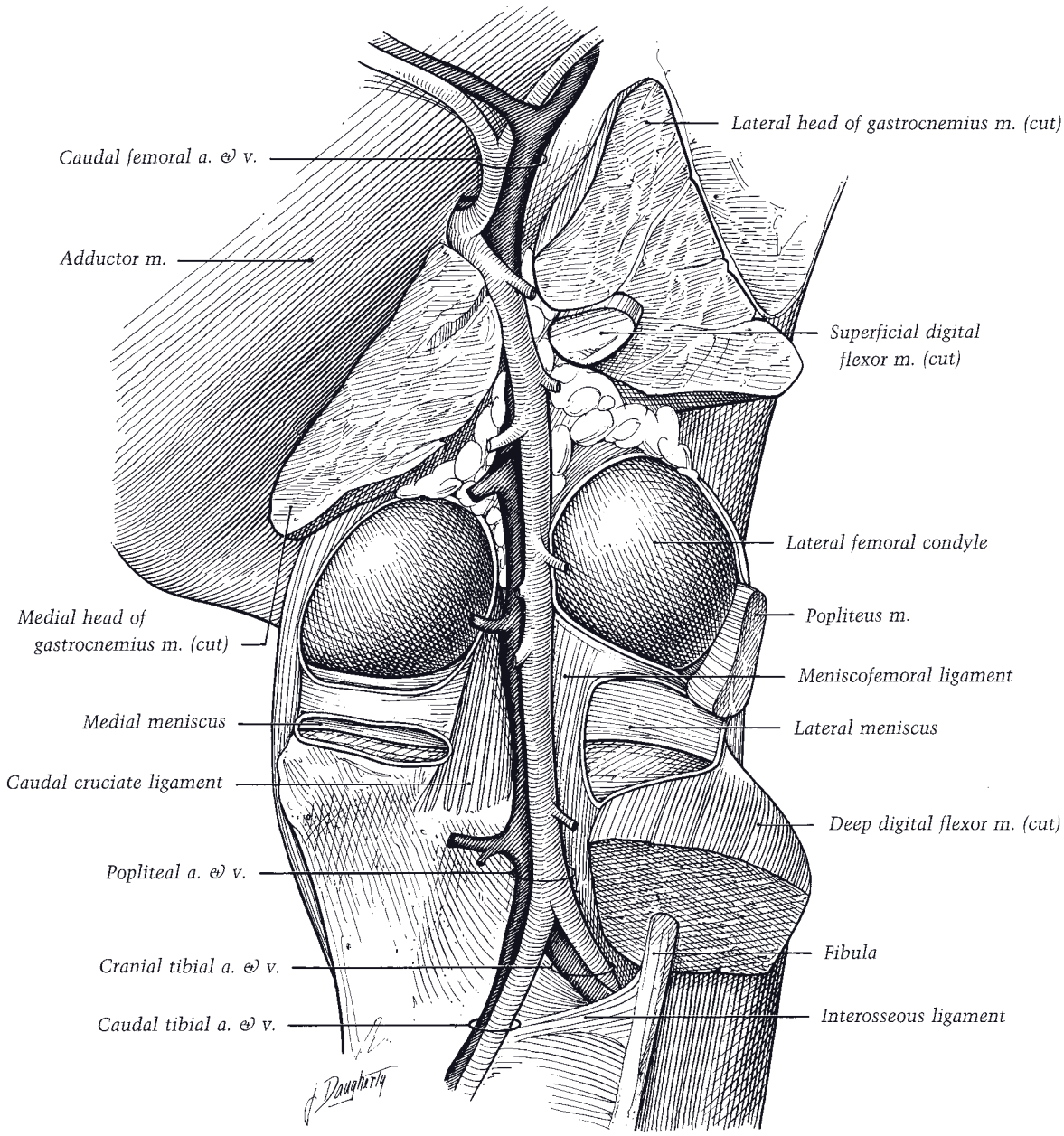


**Figure 1.41.** Deep dissection of the caudal aspect of the right stifle. The joint capsule of the femorotibial joint has been opened.

and trochlea changes as the patella moves on its larger gliding surface during flexion of the stifle joint. The patella rolls on to its narrow distal articular surface (resting surface) as the parapatellar fibrocartilage maintains its tight relationship over the trochlea's medial ridge due to tension exerted by the medial patellar ligament. The narrow craniodorsal surface of the proximal part of the trochlea may be termed its resting surface.

### Femorotibial Joint

The fibrous part of the joint capsule is thick caudally, thin cranially. The cranial and caudal

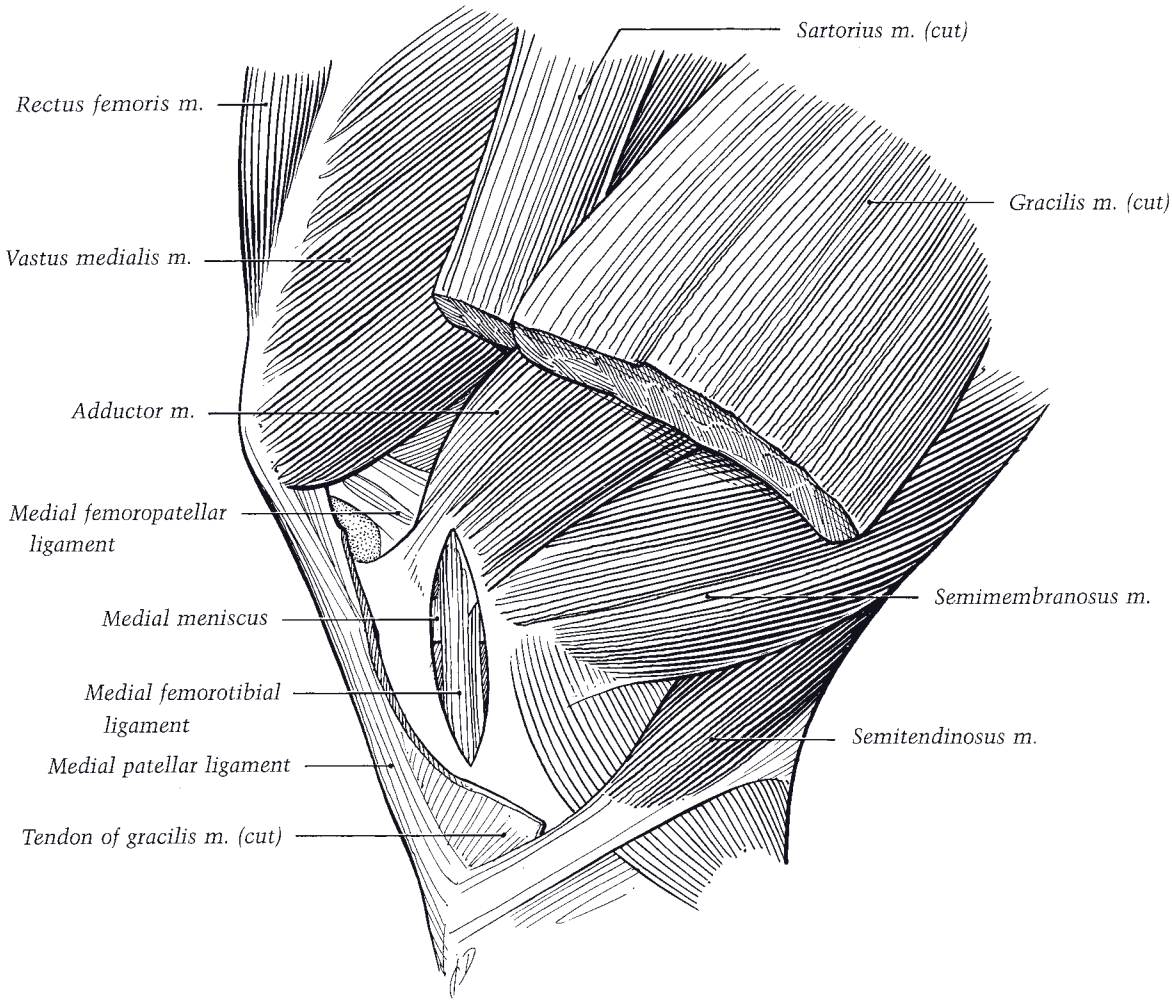


**Figure 1.42.** Deep dissection of the medial aspect of the left stifle. The tendon of the adductor muscle is incised to reveal the medial collateral ligament of the stifle.

cruciate ligaments of the femorotibial joint lie between the joint capsule's medial and lateral synovial sacs. Two fibrocartilaginous menisci intervene between the femoral and tibial articular surfaces, thus partially subdividing each sac. An extension of the lateral synovial sac encloses the tendon of origin of the popliteus muscle and another protrudes distad under the common tendon of origin of the long digital extensor and fibularis tertius muscles.

The two fibrocartilaginous menisci are crescent-shaped, being thicker peripherally and thinner along the concave edge. Their proximal surfaces are concave to accommodate the convexity of the femoral condyles. Distally they conform to the peripheral parts of the articular surfaces of the tibial condyles. Cranial and caudal ligaments anchor each meniscus to the tibia, and a meniscofemoral ligament attaches the caudal aspect of the lateral meniscus to the caudal surface of the intercondyloid fossa of the femur.

In addition to the support rendered by medial and lateral collateral ligaments, the femur and tibia are joined by the two cruciate ligaments that cross one another in the intercondyloid space between the two synovial sacs of the femorotibial joint. The caudal cruciate ligament, the more substantial of the two, extends from the cranial surface of the intercondyloid fossa of the femur to the popliteal notch of the tibia, crossing the medial aspect of the cranial (or

lateral) cruciate ligament. From its attachment on the lateral wall of the intercondyloid fossa, the cranial cruciate ligament attaches to a central fossa between the articular surfaces of the condyles.

### Movements of the Stifle Joint

In the standing position, the caudal angle of the stifle joint is around 150°. The quadriceps femoris muscle is relatively relaxed in this position. Extension of the stifle joint through action of the quadriceps femoris, tensor fasciae lata, and cranial division of the biceps femoris muscles plus passive traction exerted by the fibularis tertius is limited by tension from the collateral and cruciate ligaments. Flexion of the joint by the semitendinosus, middle division of the biceps femoris, popliteus, and gastrocnemius muscles, plus passive traction exerted by the superficial digital flexor, is limited only by the caudal muscle masses. During flexion the crus is rotated slightly mediad, and the femoral condyles and menisci move slightly caudad on the tibial condyles with somewhat more movement on the lateral surfaces.

When a horse shifts its weight to rest on one hindlimb, the supportive limb flexes slightly as the contralateral relaxed limb is brought to rest on the toe. The pelvis is tilted so that the hip of the supporting limb is higher. The stifle on the supporting limb is locked in position due to a slight medial rotation of the patella as the medial patellar ligament and parapatellar cartilage slip farther caudad on the proximal part of the medial trochlear ridge. The loop created by the parapatellar cartilage and medial patellar ligament is pulled proximad and mediad to engage the medial ridge of the femoral trochlea. The locked position achieved by this configuration together with the support rendered by the other components of the stay apparatus minimizes muscular activity in the supporting limb while the relaxed contralateral hindlimb is resting. A very small amount of muscle tone, confined to the vastus medialis, is necessary to stabilize the stifle in the locked position.

The neurovascular supply to the stifle region is illustrated in Figures 1.38, 1.39, 1.40, 1.41.

## *Thigh and Hip*

### Lateral Aspect

From caudal to cranial, the superficial muscles of the lateral thigh and hip are the semitendinosus, biceps femoris, gluteus superficialis, gluteus medius, and tensor fasciae lata. Both the semitendinosus and biceps femoris have ischiatic and vertebral origins. The semitendinosus attaches to the first and second caudal vertebrae and fascia of the tail and the biceps femoris attaches to the dorsal sacroiliac ligament and the gluteal and tail fasciae. A prominent longitudinal groove marks the site of the intermuscular septum between the semitendinosus and the biceps femoris muscles.

The strong gluteal fascia gives origin to and unites the long caudal head and the cranial head of the gluteus superficialis (superficial gluteal) muscle. The two heads of the superficial gluteal muscle unite in a flat tendon that attaches to the trochanter tertius of the femur. Extending caudad from the aponeurosis of the longissimus lumborum muscle, the large gluteus medius (middle gluteal) muscle forms most of the mass of the rump. The middle gluteal muscle also takes origin from the gluteal surface of the ilium, the coxal tuber and sacral tuber, the sacrotuberal and dorsal sacroiliac ligaments, and the gluteal fascia. Distally the muscle attaches to the greater trochanter, a crest distal to the greater trochanter and the lateral surface of the intertrochanteric crest.

The tensor fasciae latae muscle arises from the coxal tuber and fans out distally to insert into the fascia lata. An intermuscular septum attaches the caudal part of the muscle to the cranial head of the superficial gluteal. The fascia lata attaches to the patella and the lateral and middle patellar ligaments (Figure 1.43). The intermuscular septum between the biceps femoris and semitendinosus, the septa between the three divisions of the biceps femoris, and a septum between the biceps femoris and vastus lateralis all arise from the fascia lata (Figures 1.43).

Deeply on the lateral aspect of the hip, the smaller deep part of the gluteus medius, the gluteus accessorius, has a distinct flat tendon that plays over the convexity of the greater trochanter on its way to attach on the crest distal to the trochanter. The large trochanteric bursa lies between the tendon and the cartilage covering the convexity (Figure 1.43). The small gluteus profundus muscle is deep to the caudal part of the gluteus medius, arising from the ischiatic spine and body of the ilium and attaching on the medial edge of the convexity of the greater trochanter (Figure 1.44). This muscle covers the hip joint and parts of the articularis coxae and rectus femoris muscles. A bursa is commonly present under the tendon of insertion of the gluteus profundus.

On the caudal side of the proximal part of the femur, the gemelli, external obturator, and

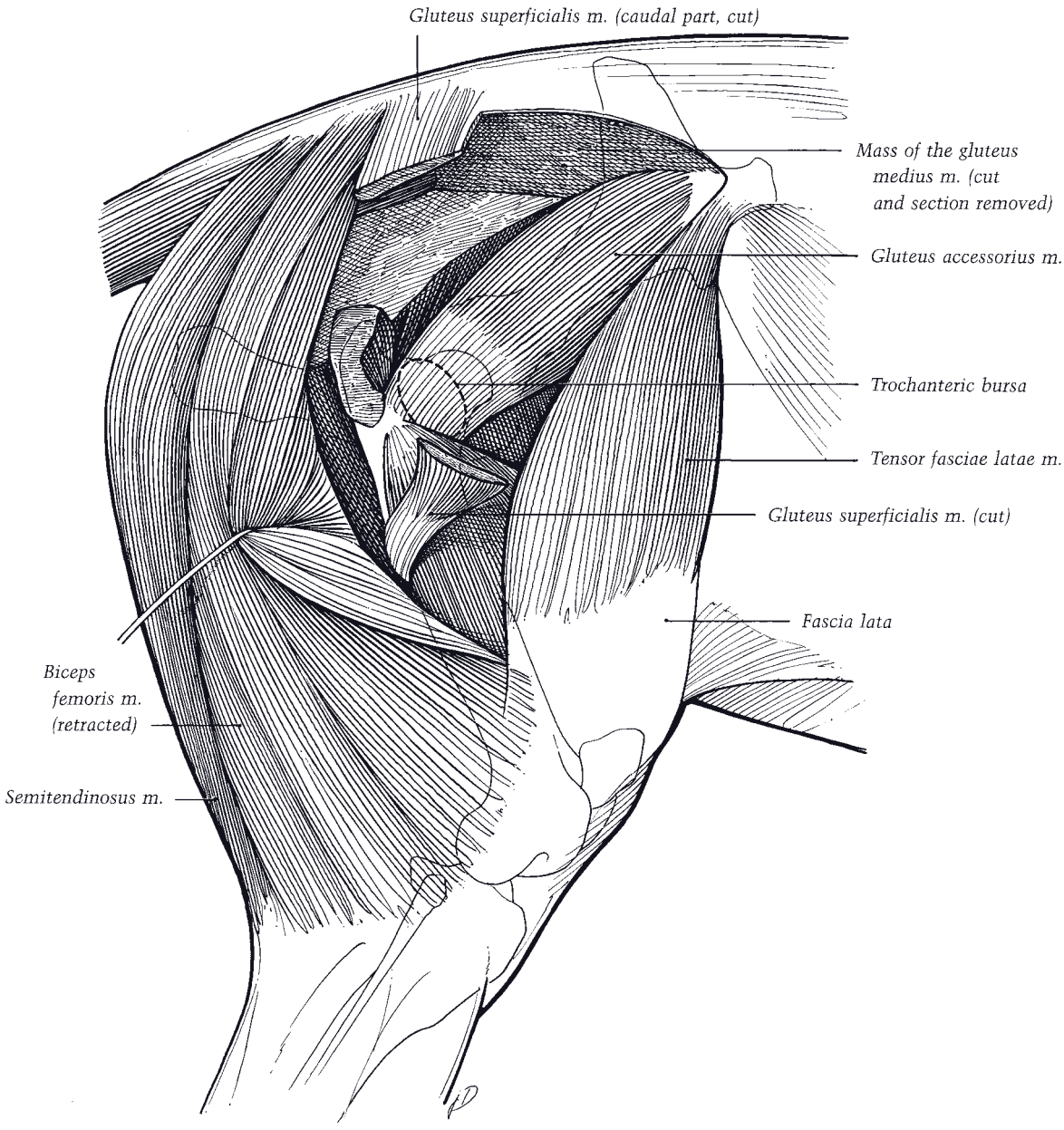


**Figure 1.43.** Lateral dissection of the right thigh and hip. Most of the superficial and middle gluteal muscles have been removed.

internal obturator muscles come from their respective origins on the ischium, pubis, ilium, and wing of the sacrum to insert in the trochanteric fossa. The quadratus femoris muscle extends from the ventral aspect of the ischium to a line on the femur near the distal part of the lesser trochanter.

A broad sheet of dense white fibrous connective tissue, the sacrotuberous (sacrotuberal) ligament, forms most of the lateral wall of the pelvic cavity, attaching dorsally to the sacrum and first two caudal vertebrae and ventrally to the ischiatic spine and ischiatic tuber (Figure 1.44). The ventral edge of the sacrotuberal ligament completes two openings along the dorsal edge of the ischium: the lesser and greater ischiadic foramina, which allow passage of neurovascular bundles from the lumbosacral region to the muscle of the rump.

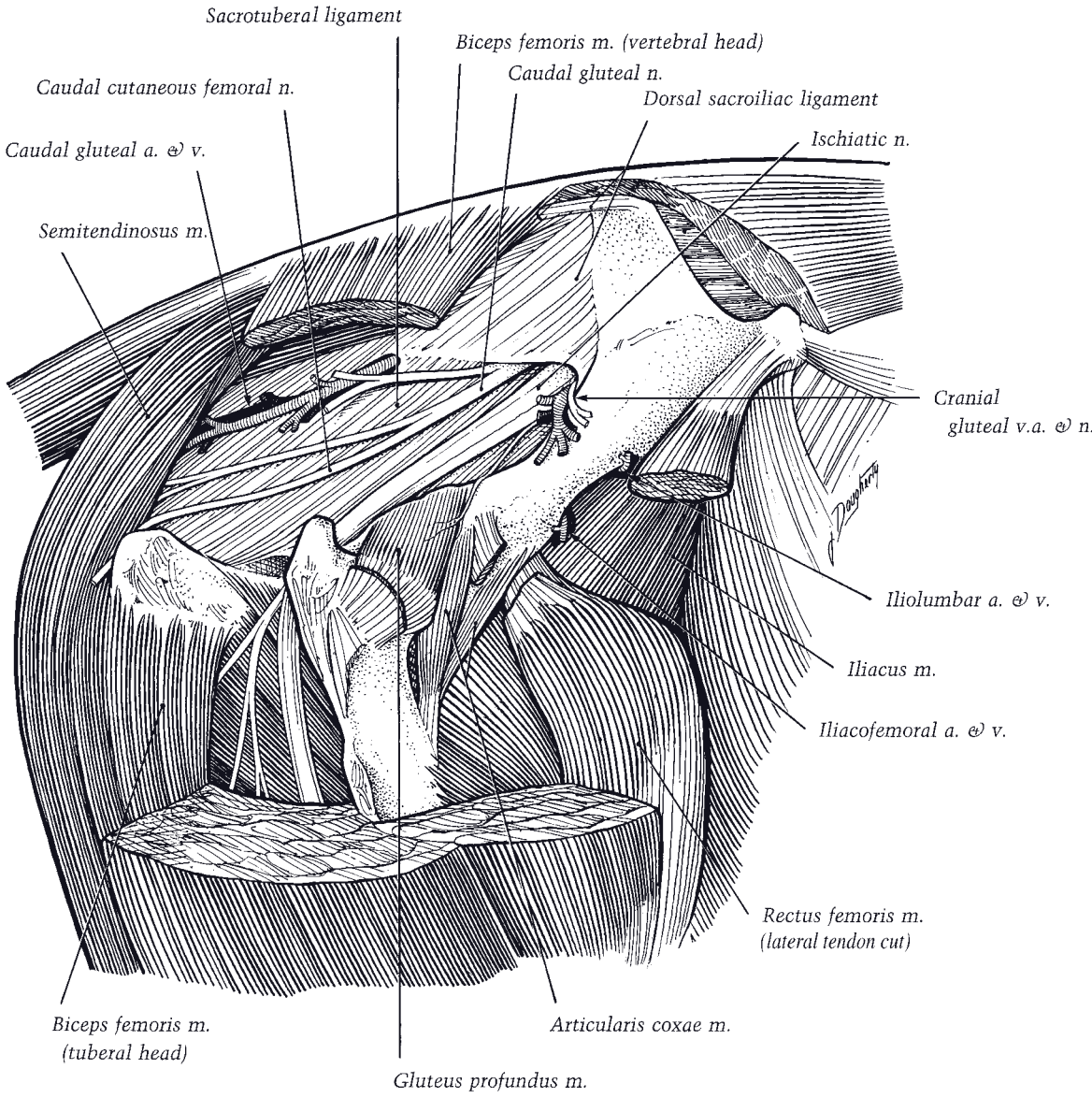


**Figure 1.44.** Deep dissection of the right hip, lateral view.

The large, flat sciatic nerve passes through the greater ischiatic foremen and courses ventrocaudad on the sacrotuberale ligament and then on to the origin of the gluteus profundus (deep gluteal). Turning distad, the ischiadic nerve passes over the gemelli, the tendon of the internal obturator, and the quadratus femoris, supplying branches to these muscles. A large branch is detached from the deep side of the nerve. This branch supplies branches to the semimembranosus, the biceps femoris and semitendinosus, and adductor medially and the biceps femoris laterally. The sciatic nerve terminates by dividing into common fibular and tibial nerves.

## Medial Aspect

The broad gracilis muscle covers most of the medial aspect of the thigh, attaching proximally to the prepubic tendon, adjacent surface of the pubis, accessory femoral ligament, and middle of the pelvic symphysis. The muscle belly ends

distally by joining a wide thin aponeurosis of insertion. The narrow sartorius muscle takes origin from the tendon of the psoas minor and ilial fascia and descends toward its insertion in the stifle, which blends with the tendon of the gracilis. Deep to the gracilis lies the pectineus muscle. It attaches proximally to the cranial border of the pubis, the prepubic tendon, and accessory femoral ligament. Distally, the pectineus attaches to the medial border of the femur. The femoral canal, containing the neurovascular bundle supplying the pelvic limb, is delimited caudally by the pectineus, cranially by the sartorius, laterally by the vastus medialis and iliopsoas, and medially by the femoral fascia and cranial edge of the gracilis. The canal contains the femoral artery and vein, the saphenous nerve, and an elongated group of several lymph nodes of the deep inguinal lymphocenter embedded in adipose tissue (Figure 1.45).

Caudal to the pectineus and vastus medialis, the thick adductor muscle extends from the ventral surface of the ischium and pubis and the origin of the gracilis muscle to the caudal surface of the femur, the medial femoral epicondyle, and the medial collateral ligament of the femorotibial joint. The obturator nerve passes through the cranial part of the obturator foramen and external obturator muscle and branches to supply the external obturator, adductor, pectineus, and gracilis muscles (Figure 1.45).

### Cranial Aspect

The quadriceps femoris, articularis coxae, and sartorius muscles lie in the cranial part of the thigh and hip. In addition, the iliacus muscle crosses the cranial aspect of the hip where the muscle encloses the psoas major, creating the conjoined iliopsoas muscle. Their common tendon inserts on the lesser trochanter. The psoas major arises from the last two ribs and the lumbar transverse processes; the iliacus comes from the wing of the sacrum, ventral sacroiliac ligaments, sacropelvic surface of the ilium, and tendon of the psoas minor muscle.

Three heads of the quadriceps femoris muscles (the vastus lateralis, vastus intermedius, and vastus medialis) take origin from the shaft of the femur. The fourth head, the rectus femoris, originates from two tendons, one arising from a medial depression on the ilium craniodorsal to the acetabulum, and one from a lateral depression (Figure 1.44). All four heads of the quadriceps femoris attach to the patella.

### Caudal Aspect

The main muscle mass is that of the semimembranosus with the semitendinosus, with the caudal division of the biceps femoris related to it laterally and the gracilis medially. The long head of the semimembranosus attaches to the caudal border of the sacrotuberous ligament. The thicker short head attaches to the ventral part of the ischiatic tuber. The thick, roughly three-sided belly of the semimembranosus ends on a flat tendon that attaches to the medial femoral epicondyle.

The neurovascular supply to the thigh region is illustrated in Figures 1.40, 1.41, 1.44.

### Hip (Coxofemoral) Joint

The acetabulum of the os coxae is formed where the ilium, ischium, and pubis meet. The lunate surface of the acetabulum, a cup-shaped cavity arcing around a deep nonarticular fossa, articulates with the head of the femur. A fibrocartilaginous rim, the acetabular labrum, increases the articular surface of the acetabulum. The transverse acetabular ligament bridges the labrum across the medially located acetabular notch, binding two ligaments as they emerge from the fovea capitis of the femoral head (Figure 1.45). The shorter ligament of the head of the femur comes from the narrow apex of the fovea and attaches in the pubic groove. The thick accessory femoral ligament arises from the wider, peripheral part of the fovea and passes out through the acetabular notch to lie in the pubic groove. After giving partial origin to the gracilis and pectineus muscles, the accessory femoral ligament blends into the prepubic tendon.

The capacious joint capsule of the hip attaches to the acetabular labrum and on the neck of the femur a few millimeters from the margin of the femoral head (Figure 1.45). Within the joint the synovial membrane wraps around the ligaments. An outpocketing of the synovial membrane passes out through the acetabular notch to lie between the accessory femoral ligament and the pubic groove. A small pouch also lies under the ligament of the head of the femur. The articularis coxae muscle is related to the lateral aspect of the hip joint, detaching some fibers to the joint capsule. During flexion of the hip joint, the articularis coxae can serve to tense the joint capsule.

While the hip joint is a ball-and-socket joint, capable only of very limited rotation, its principal movements are flexion and extension. Abduction of the thigh is restricted by the

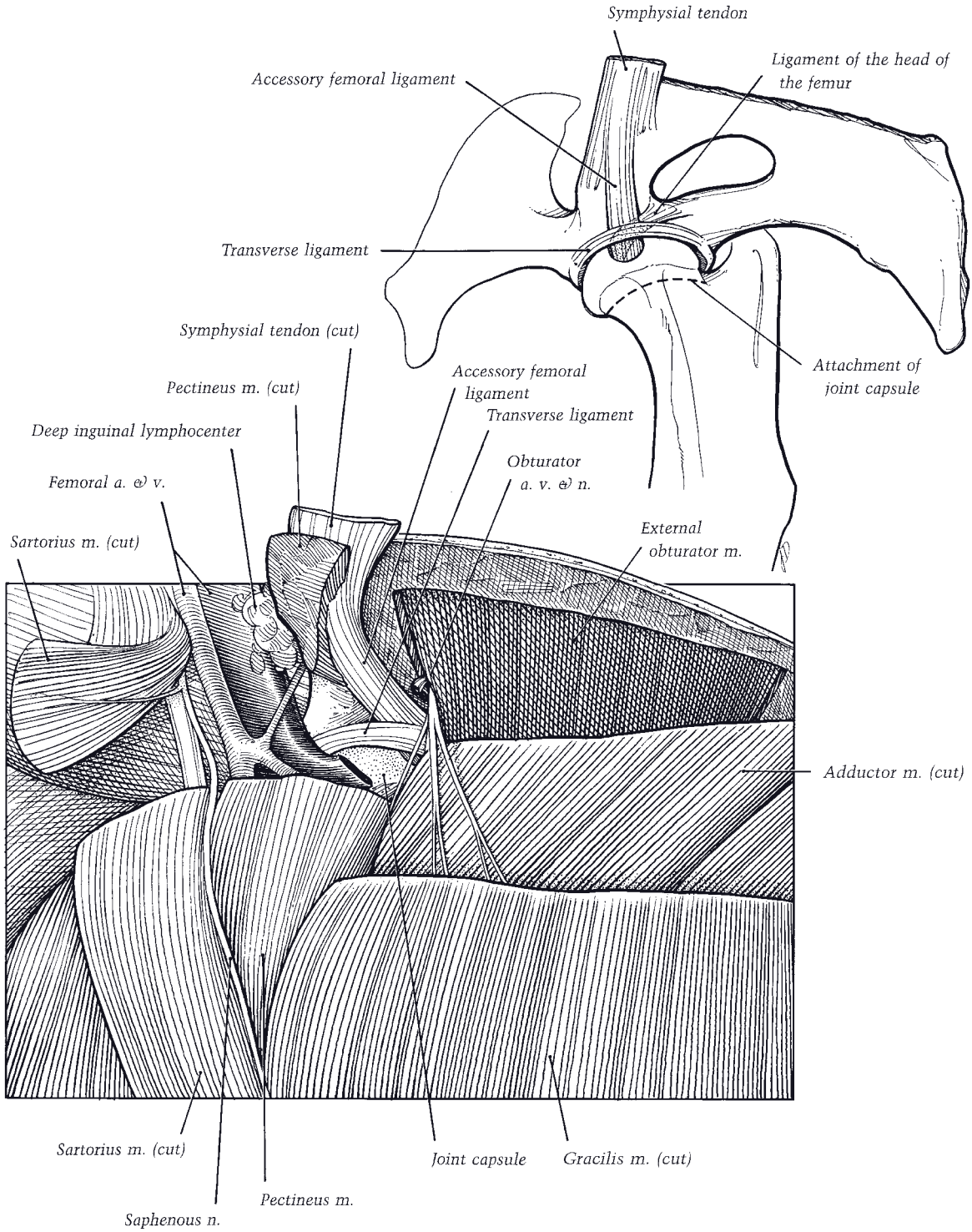


**Figure 1.45.** Deep dissection of the right hip, ventromedial view.

ligament of the head of the femur and the accessory femoral ligament. Adduction is checked by the attachments of the gluteal muscles on the femur. In the normal standing position, the caudolateral part of the head of the femur lies outside the acetabulum. The hip joint is slightly flexed in this position, the cranial angle being around 115°.

### Pelvis

The equine pelvis, like that of other animals, comprises the ilium, ischium, and pubis; these bones are individually identifiable in the young but have fused by 10 to 12 months of age.

The wing-shaped ilium presents two prominences, visible landmarks on the horse. The dorsally directed tuber sacrale inclines mediad toward its fellow, so that the two sacral tubers come within 2 to 3 cm over the first sacral spinous process. The ilial wing projects ventrolaterad in a bulky tuber coxae, creating the point of the hip.

Caudally, the ischial tuberosity presents as a laterally directed ridge to which muscles of the thigh attach. The acetabulum is formed through contributions from all three bones of the pelvis.

The pubis and ischium from each side meet ventrally at the symphysis pelvis. In the young animal fibrocartilage joins the bones. Later in life, a synostosis is formed as the cartilage ossifies in a cranial to caudal sequence.

### *Stay Apparatus of the Pelvic Limb*

The quadriceps femoris muscle and the tensor fasciae latae act to pull the patella, parapatellar cartilage, and medial patellar ligament proximad to the locked position over the medial trochlear ridge of the femur when the limb is positioned to bear weight at rest (Figure 1.46). Through the components of the reciprocal apparatus (cranially, the fibularis tertius from the femur to the lateral tarsus and proximal metatarsus and, caudally, the superficial digital flexor from the femur to the calcaneal tuber) the tarsus is correspondingly locked in extension. A small amount muscular activity in the quadriceps muscle assures continuation of this locked configuration, preventing flexion of the stifle and tarsocrural joints. Distal to the hock the digital flexor tendons support the plantar pes, the SDFT extending distad from its connection to the calcaneal tuber and the DDFT usually receiving the accessory (tarsal check) ligament from the thick plantar part of the tarsal fibrous joint capsule. Prevention of overextension of the fetlock joint during the fixed, resting position is accomplished through the support rendered by the digital flexor tendons and the suspensory apparatus (suspensory ligament, proximal sesamoid bones, and their ligaments).

## AXIAL COMPONENTS

### *Vertebral Column*

The vertebral formula of the horse is 7 cervical, 18 thoracic, 6 lumbar, 5 sacral, and an inconsistent number of caudal vertebrae (ranging from 15 to 21). There is some individual variation in numbers of other vertebrae, most commonly in the number of lumbar vertebrae, where 5 or 7 are sometimes seen (there is an increased incidence of 5 lumbar vertebrae in Arabian horses). The typical vertebra possesses a ventrally placed, roughly cylindrical body whose cranial and caudal ends articulate with adjacent vertebrae at the intervertebral disc (Figure 1.47). A bony vertebral arch attaches to the body and surrounds the spinal cord. The aperture created within a given vertebrae by the dorsal aspect of the body and the medial and ventral parts of the arch is the vertebral foramen; where vertebral foramina of adjacent vertebrae are aligned to admit the spinal cord, the resulting passageway is called the vertebral canal. The vertebral canal is widest in the caudal cervical-cranial thoracic region, where it accommodates the cervical enlargement of the spinal cord; a second dilation of the canal occurs in the lumbar region where the lumbosacral enlargement of the cord resides.

The vertebral arch comprises the pedicles and laminae, which together create the "roof" over the spinal cord. The pedicles are the vertical bony attachments to the vertebral body. The dorsal part of the arch is created by the right and left laminae. The pedicles are characterized by vertebral notches, indentations on the cranial and caudal aspects of the pedicle. When individual vertebrae are articulated, the cranial vertebral notch of one vertebra abuts the caudal vertebral notch of another, creating an intervertebral foramen through which the spinal nerve emerges from the vertebral canal.

The vertebral arch features other bony processes that bear synovial joints between adjacent vertebrae and which serve as sites of attachment for epaxial muscles. Each vertebra has a single dorsal midline spinous process and two transverse processes that arise near the point at which the pedicle attaches to the body. The dorsal

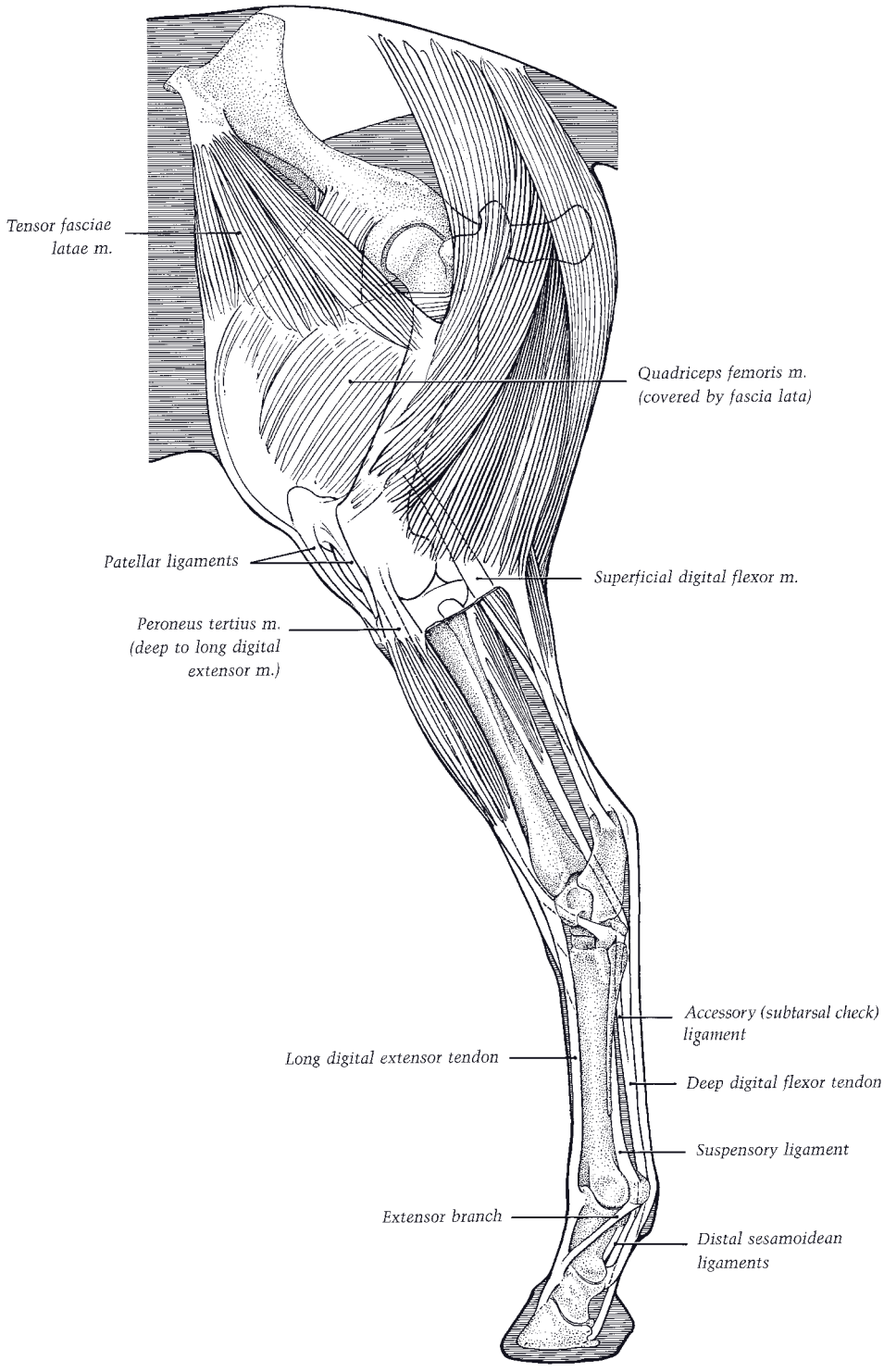


**Figure 1.46.** Stay apparatus of the pelvic limb.

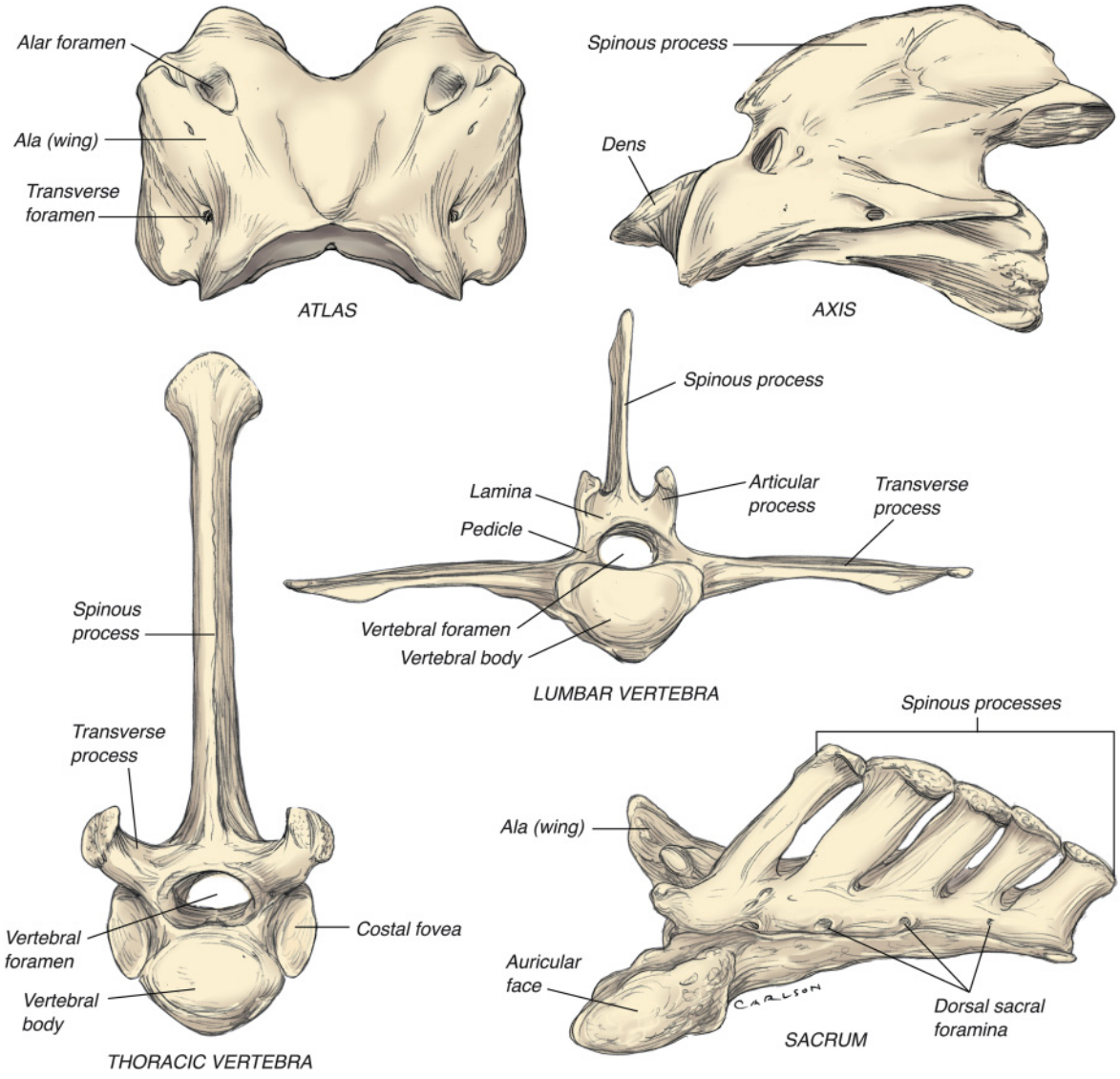


**Figure 1.47.** Vertebrae.

contour of the equine thorax and loin is largely determined by the relative size and prominence of the spinous processes of thoracic and lumbar vertebrae. Arising adjacent to the spinous process are a pair of cranial articular processes and a pair of caudal articular processes.

## Cervical Vertebrae

The first two cervical vertebrae are highly modified to meet their specialized function in permitting movement of the head. The first vertebra is the atlas. It lacks the cylindrical body characteristic of other vertebrae, instead taking the form of a bony ring comprising dorsal and ventral arches. The spinous process is likewise absent. The transverse processes are modified into the wings of the atlas. These are robust, bent in a ventrolateral direction, and strongly concave ventrally. Their craniolateral edges form a prominent palpable ridge caudal to the ramus of the mandible. The dorsal aspect of the wing of the atlas bears three foramina: the transverse foramen, the alar foramen, and the lateral verte-

bral foramen. The cranial aspect of the atlas possesses two deeply concave cranial articular foveae which form a synovial joint (the atlanto-occipital joint) with the occipital condyles. The caudal articular foveae are also concave and participate in the synovial atlantoaxial joint.

The second cervical vertebra is the axis. The body of the axis is long; its cranial extremity is modified into a scoop-like projection called the dens, which features a rounded ventral articular surface that articulates with the floor of the atlas. The caudal extremity of the axis' body is deeply concave where it articulates with the body of the third cervical vertebra. The spinous process of the axis is tall and long, modified into a thick midline sail. The transverse processes are small and caudally directed.

The third through seventh vertebrae are similar to one another and follow the basic pattern of most vertebrae. They are progressively shorter from cranial to caudal. Cervical vertebrae three, four, and five bear a distinct ventral crest on their bodies. This crest is diminished in size on the sixth and absent from the seventh cervical vertebra. Articular processes on these cervical vertebrae are large, with prominent oval fovea for articulation between vertebral arches. Transverse processes are broad, each with two thick tubercles for muscular attachment. The spinous process of the seventh cervical vertebra is tall compared to other cervical vertebrae.

### Thoracic Vertebrae

There are usually 18 thoracic vertebrae in the horse, although there may on occasion be one more or one less than typical. The bodies of the thoracic vertebrae tend to be short with a small vertebral arch dorsally. The spinous processes are relatively tall, with the first four or five increasing in height and more caudal spinous processes gradually decreasing in height until at the level of the twelfth thoracic vertebra, after which they are the same height as those of the lumbar vertebrae.

The tall spinous processes of those first twelve vertebrae constitute the withers. The dorsal apex of the spines is somewhat expanded and in young horses surmounted by cartilage. The cartilage is replaced by bone as the horse ages, with the cartilages associated with the prominence of the withers persisting the longest at ten years or more. The anticlinal vertebra is defined as the one whose spinous process is perpendicular to the long axis of the vertebral column; the spinous processes of more cranial vertebrae incline caudad, while those of more caudal vertebrae incline craniad. In the horse, the anticlinal vertebra is usually the 16th, and occasionally the 14th.

The vertebral bodies possess cranial and caudal costal foveae for articulation with the heads of ribs, except for the last thoracic vertebra which features only cranial costal foveae. Transverse processes are irregular, largest in the cranial thoracic vertebrae, and gradually decreasing in size toward the lumbar region.

### Lumbar Vertebrae

There are usually six lumbar vertebrae, although five and seven also have been reported. The cylindrical bodies of the lumbar vertebrae are somewhat flattened dorsoventrally, especially the last three; except for the seventh and sometimes the sixth lumbar vertebrae, a ventral crest is prominent. The spinous processes project slightly craniad. The vertebral arches tend to overlap dorsally, except at the L5-L6 and L6-S1 interspaces, where the larger interarcuate spaces are much larger and clinically accessible. The cranial and caudal articular processes articulate in an approximation of the sagittal plane, an orientation which allows for a very slight degree of flexion and extension of the vertebral column but prevents lateral flexion. The transverse processes of the lumbar vertebrae are large and blade-like. They project laterad. The caudal aspect of the fifth transverse process articulates with the cranial aspect of the sixth. The caudal aspect of the sixth transverse process features a large concave facet through which it articulates with the sacrum.

### Sacrum

The equine sacrum is a single bone formed through fusion of embryologically distinct sacral vertebrae, generally five of these, with four, six, and seven sacral vertebrae also being reported. Fusion is usually complete by five years of age. The sacrum is triangular and gently curving so as to present a slightly concave ventral aspect. Intervertebral foramina are transformed by the fusion of adjacent vertebrae into a row of four dorsal sacral foramina and four ventral sacral foramina, through which pass dorsal and ventral branches, respectively, of the sacral spinal nerves. The spinous processes remain individually distinct and incline slightly caudad, and the second through fifth end in slight enlargements that are not uncommonly bifid.

The first sacral vertebra gives rise to the wings of the sacrum. Their articular surfaces face dorsolateral to articulate with the auricular surface of the ilium. The ventral aspect of the first sacral vertebra is slightly rounded, forming the promontory of the sacrum, the point from which the conjugate diameter of the pelvis is measured.

## Caudal Vertebrae

Although there is considerable individual variation, the average horse has 18 caudal vertebrae. Only the first three or so have vertebral arches, the remaining being represented by cylindrical bodies only. The first caudal vertebra is not uncommonly fused with the sacrum, especially in old horses.

## Vertebral Articulations

Excluding the atlantoaxial joint (a pivot joint), the joints of the vertebral column all permit flexion, extension, lateral flexion, and limited rotation. These movements are fairly limited through the thoracic and lumbar regions, but the cervical vertebral column is capable of extensive movement. Intervertebral discs of fibrocartilage are interposed between adjacent vertebral bodies. Further stabilization is provided to the vertebral column by (1) the continuous dorsal and ventral longitudinal ligaments on their respective surfaces of the vertebral bodies; (2) a supraspinous ligament that passes along the dorsal aspect of the spinous processes of the thoracic, lumbar, and sacral vertebrae; and (3) interspinal ligaments that pass between adjacent spinous processes. In the thoracic region, intercapital ligaments pass transversely between the heads of contralateral ribs over the dorsal aspects of the intervertebral disks. Articulations between articular processes on vertebral arches are true synovial joints. In the cervical region, these constitute broad plates, oriented in a nearly horizontal plane so as to permit significant lateral bending. Articular facets on the cranial articular processes face dorsomediad, whereas the complementary facets on the caudal articular processes face ventrolaterad. True joints also exist between the transverse processes of the fifth and sixth lumbar vertebrae and between the transverse processes of the sixth lumbar vertebra and the wings of the sacrum.

## Sacroiliac Region

The axial skeleton and appendicular skeleton of the hindlimb are united at the sacroiliac joint

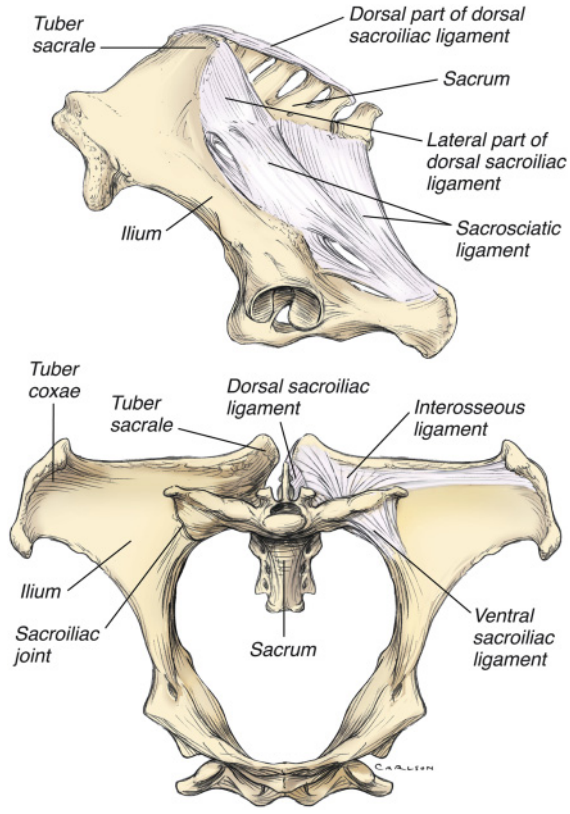


**Figure 1.48.** Sacroiliac joint, lateral (top) and cranial (bottom) views.

(Figure 1.48). This planar joint is created by the auricular face of the wings of the sacrum, which face dorsolaterad, and the auricular face of the wings of the ilia, which face ventromediad. This joint is histologically synovial, but is capable of only extremely limited gliding movement; its principle purpose is most likely absorption of concussive forces transmitted through the appendicular skeleton to the vertebral column. The joint capsule is close-fitting and is substantially reinforced by a series of sacroiliac ligaments that contribute markedly to the overall stability of the joint and probably act to transfer most of the weight of the trunk to the pelvic limbs. These ligaments can be summarized as comprising the ventral sacroiliac ligament, the dorsal sacroiliac ligament, and the interosseous ligament.

The ventral sacroiliac ligament surrounds the joint and fills the space between the ilium and the wing of the sacrum. The dorsal sacroiliac ligament presents two distinct portions. One arises from the tuber sacrale and inserts on the spinous processes of the sacral vertebrae. The

other, a more laterally placed sheet, arises from the tuber sacrale and the caudal edge of the ilial wing and inserts along the lateral aspect of the sacrum. From here it blends ventrad into the broad sacrosciatic ligament that fills the space between the pelvis and sacrum. The interosseous ligament consists of strong, vertically oriented fibers between the ventral part of the wing of the ilium and the dorsal aspect of the wing of the sacrum.

### Ligamentum Nuchae

The topline of the neck is in part determined by the presence of the ligamentum nuchae (nuchal ligament), which in horses extends from its cranial attachments on the external occipital protuberance to the spinous process of the third or fourth thoracic vertebra. Both parts of the nuchal ligament (funicular and laminar) are paired. The rope-like funicular part is connected to sheets which comprise the laminar portions. These midline elastic sheets arise from the second through seventh cervical vertebrae and insert on the spines of the second and third thoracic vertebrae. Bursae are consistently found between the funicular part of the nuchal ligament and the atlas (bursa subligamentosa nuchalis cranialis) and between the nuchal ligament and the second thoracic spine (bursa subligamentosa supraspinalis). A third bursa (bursa subligamentosa nuchalis caudalis) is inconsistently found between the nuchal ligament and the spine of the axis.

## *Muscles of the Trunk and Neck*

Muscles of the torso (neck, trunk, and tail) are roughly divided into those dorsal to the transverse processes (i.e., epaxial muscles) and those ventral to the transverse processes (i.e., hypaxial muscles). The epaxial muscles are innervated by dorsal branches of the spinal nerves, while hypaxial muscles receive their innervation from ventral branches.

The epaxial muscles are extensors of the vertebral column and are roughly divided into three parallel bundles of fascicles: from lateral to medial these are the iliocostalis system, the longissimus system, and the transversospinalis system (Figure 1.49). The iliocostalis system (named for its attachments to the ilium and ribs) does not extend into the neck; the others continue into the cervical region and are associated with additional distinct muscles. Of these, the splenius muscle is most superficial (Figure 1.50). The splenius possesses capital and cervical parts.

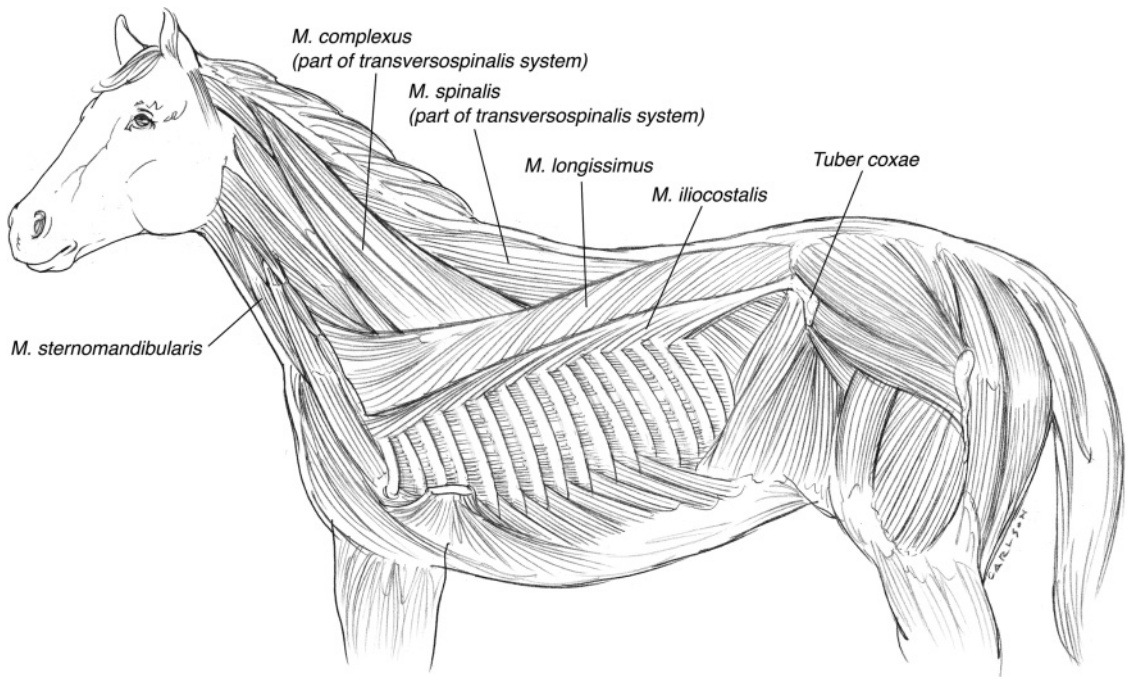


**Figure 1.49.** Deep muscles of the trunk.

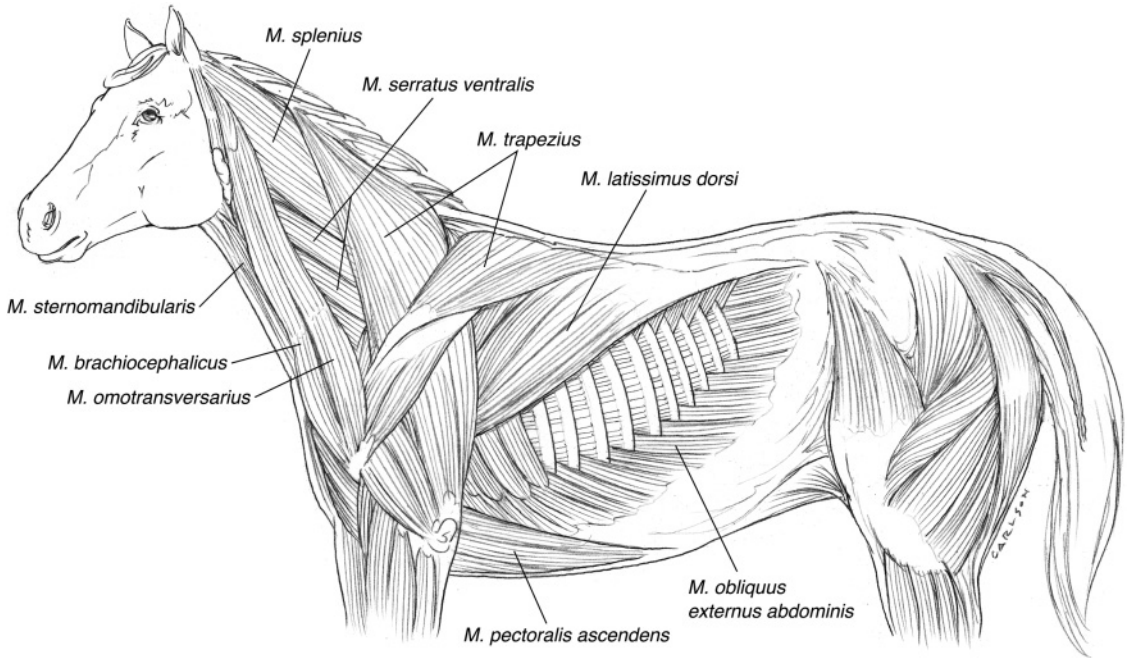


**Figure 1.50.** Superficial muscles of the trunk. The cutaneous muscles have been removed.

Both arise from the third, fourth, and fifth thoracic spines and from the funicular part of the nuchal ligament, and they insert on the nuchal crest of the skull, the wing of the atlas, and the transverse processes of the third, fourth, and fifth cervical vertebrae. The splenius extends the neck and elevates the head, and it is largely the rhythmic contraction of this powerful muscle that creates the thrusting movements of the neck during the gallop.

The longissimus group is described as having lumbar, thoracic, cervical, atlantal, and capital portions. It is most robust in the lumbar region, where it gives a well-conditioned horse's back its typical rounded appearance.

Hypaxial muscles of the trunk (the psoas minor, quadratus lumborum, and the four abdominal muscles on each side) act to flex the vertebral column during the gallop. Epaxial muscles extend the vertebral column. When they contract unilaterally, both hypaxial and epaxial muscles create lateral movement of the trunk and neck.

In the ventral neck, the equine longus colli muscle is particularly well developed relative to other domestic species. The cervical portion of this muscle arises from the transverse processes and bodies of the third through sixth cervical vertebrae in paired bundles that converge to an insertion on the preceding vertebral bodies, sometimes bridging more than one intervertebral space. The most cranial attachment is on the ventral aspect of the atlas. The thoracic portion of the longus colli arises on the lateral vertebral bodies of thoracic vertebrae one through six, passing craniad to insert on the transverse processes of cervical vertebrae six and seven.

The omohyoideus muscle is well-developed in the horse. It arises from an aponeurosis in the fascia near the shoulder joint; its muscle belly is closely attached to the deep side of the brachiocephalicus until the neck's midpoint, where the omohyoideus becomes evident as a distinct muscle as it passes craniad deep to the sternocephalicus muscle. The right and left sternomandibularis muscles of the horse are fused on midline near their origin on the manubrium. Near mid-neck, the two halves separate, moving from their position ventral to the trachea to a more lateral location. As the muscle approaches its insertion on the sternomandibular tuberosity on the ramus of the mandible, it narrows to a distinct tendon that is visible in the cranial neck just caudal to the caudal border of the mandible. The tendon of insertion is classically considered one side of Viborg's triangle.

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**Revised from "Functional Anatomy of the Equine Musculoskeletal System" in *Adams and Stashak's Lameness in Horses, Sixth Edition,* by Anna D. Fails and Robert A. Kainer.**

2

CHAPTER

# Fundamentals of Lameness Diagnosis

## DEFINITION OF LAMENESS

Lameness is an indication of a structural or functional disorder in one or more limbs or the axial skeleton that is evident while the horse is standing or at movement. Usually lameness is most evident visually at a trot or jog. Significant lameness often is seen at a walk, whereas more subtle lameness may not become apparent until the horse is worked.

Lameness can be caused by trauma (single event or repetitive work), congenital or acquired anomalies, developmental defects, infection, metabolic disturbances, circulatory and nervous disorders, or any combination of these. It is important to differentiate between lameness resulting from pain and nonpainful alterations in gait, often referred to as "mechanical lameness," and lameness resulting from neurologic (nervous system) dysfunction. Lameness due to pain originating from the musculoskeletal system is most common in the horse. A complete lameness examination is used to help differentiate among the many types of lameness problems that can occur in horses. The objectives of a lameness examination are to determine:

1. If the horse is lame
2. Which limb or limbs are involved
3. The site or sites of the problem
4. The specific cause of the problem
5. The appropriate treatment
6. The prognosis for recovery

The steps to perform a routine or traditional lameness examination include:

1. Complete history, including signalment and use
2. Visual exam of the horse at rest
3. Palpation of the musculoskeletal system, including hoof tester examination of the feet
4. Observation of the horse in motion (usually at a straight walk and trot/lope followed by circling)
5. Manipulative tests such as flexion tests
6. Diagnostic anesthesia if necessary
7. Diagnostic imaging

Palpation of the limbs and axial skeleton, and hoof tester examination of the feet, are usually performed prior to exercising the horse. However, some clinicians prefer to observe the horse at exercise prior to palpation of the musculoskeletal system. Diagnostic anesthesia and imaging often follow to document the location of the pain, the specific cause of the problem, the extent of injury, and the prognosis for recovery.

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

## CLASSIFICATION OF LAMENESS

There are a variety of ways to classify lameness in the horse. In most cases, a primary or baseline lameness contributes to the most obvious gait abnormalities. Compensatory, secondary, or complementary lameness results from overloading of the other limbs as a result of the primary lameness. Lameness also may be classified according to when it occurs (or is best observed) within the stride. The different classifications of lameness are defined below.

**Supporting limb lameness** is apparent when the foot first contacts the ground or when the limb is supporting weight (stance phase). Injury to bones, joints, soft tissue support structures (e.g., ligaments and flexor tendons), and the foot are considered causes of this type of lameness. This is by far the most common type of lameness identified in the horse.

**Swinging limb lameness** is evident when the limb is in motion. A variety of pathologic changes may be the cause, and the majority of these problems are thought to involve the upper limbs or axial skeleton.

**Mixed lameness** is evident both when the limb is moving (swing phase) and when it is supporting weight (stance phase). Mixed lameness can involve any combination of structures affected in swinging or supporting limb lameness.

**Primary or baseline lameness** is the most obvious lameness or gait abnormality that is observed before flexion or manipulative tests. This can be complicated by lameness in multiple limbs, but in most cases the lameness that is the worst is considered the primary lameness. Evaluation of the primary lameness should be performed initially before scrutinizing complementary lameness problems.

**Compensatory or complimentary lameness** is pain and therefore lameness in a previously sound limb that is caused by uneven distribution of weight on another limb or limbs. It is common to have complementary lameness in a forelimb as a result of lameness in the opposite forelimb. Also, lameness in a hindlimb can result in lameness in the opposite forelimb (e.g., left hind and right fore) or can mimic a forelimb lameness on the ipsilateral side (right hind and right fore).

Quantitative lameness evaluations suggest that an apparent lameness in the forelimb and hindlimb on the same side often indicate a primary hindlimb lameness and a false compensatory forelimb lameness. Additionally, lameness in one hindlimb may contribute to lameness in the opposite hindlimb. Even minor changes in weight bearing can produce complementary lameness at high speeds, especially over long distances.

By observing the gait from a distance, one can usually determine whether the lameness is supporting limb, swinging limb, or mixed. Some conditions that cause supporting limb lameness may cause the horse to alter the movement of the limb to protect the foot when it lands. This can be mistaken for swinging limb lameness. Because of this and other adaptive strategies that occur in lame horses, some clinicians feel that mixed lameness occurs most commonly in horses.

## WHAT TO LOOK FOR

In general, unilateral forelimb lameness is the easiest for most people to observe. This is followed in increasing order of difficulty by bilateral forelimb lameness, unilateral hindlimb lameness, unilateral forelimb and hindlimb lameness, and multiple limb lameness. The more limbs or sites involved, the more difficult it is to accurately "see" the adaptive changes that are occurring in the horse.

With most lameness conditions, the horse will attempt to "unload" the lame limb during weight-bearing on the stance phase of the stride. In a kinetic study of forelimb lameness, the peak vertical force had the highest sensitivity and specificity for lameness classification. Horses unload the lame limb by abnormal movement of a body part (head nod or pelvic hike), weight shifting (to the contralateral or diagonal limb or torso), change in joint angles (lack of fetlock extension), and alterations in foot flight. For example, hyperextension of the fetlock has been shown to be an indirect measure for the vertical ground reaction force and is reduced in the lame limb at the stance phase proportionally to the degree of lameness. In addition, in horses with chronic lameness, the limb with the flatter hoof exhibits higher vertical loads because it is the non-lame limb (Figure 2.1).

Detecting these compensatory movements is an integral part of diagnosing lameness in the horse. The most consistent compensatory movements that are observed are the vertical displacement and acceleration of the head in forelimb lameness and of the sacrum and tuber coxae in hindlimb lameness. Although overlap of these movements can occur (head movement with hindlimb lameness and pelvic movement with forelimb lameness), they tend to occur primarily in moderate to severe lameness. In addition, more subtle lameness will cause fewer compensa-

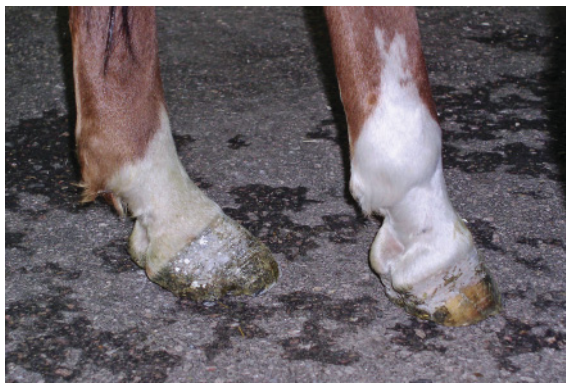

**Figure 2.1.** Chronic hindlimb lameness that has resulted in a wide flat foot on the sound limb (LH) and a narrow, upright hoof on the lame limb (RH).

tory changes, making lameness diagnosis more difficult, and problems within the axial skeleton (back) may also alter the movement of the limbs.

### *Gaits*

Most lameness is detected when the horse is in movement at several different gaits. Knowing the characteristics of the different gaits can be helpful when evaluating the adaptive strategies that horses use to "unweight" the lame limb(s). The walk is a four-beat gait that should have a very even rhythm as the feet land and take off in the following sequence: left hind, left fore, right hind, right fore (Figure 2.2). The pace (Figure 2.3) is a two-beat lateral gait in which the two right limbs rise and land alternately with the two

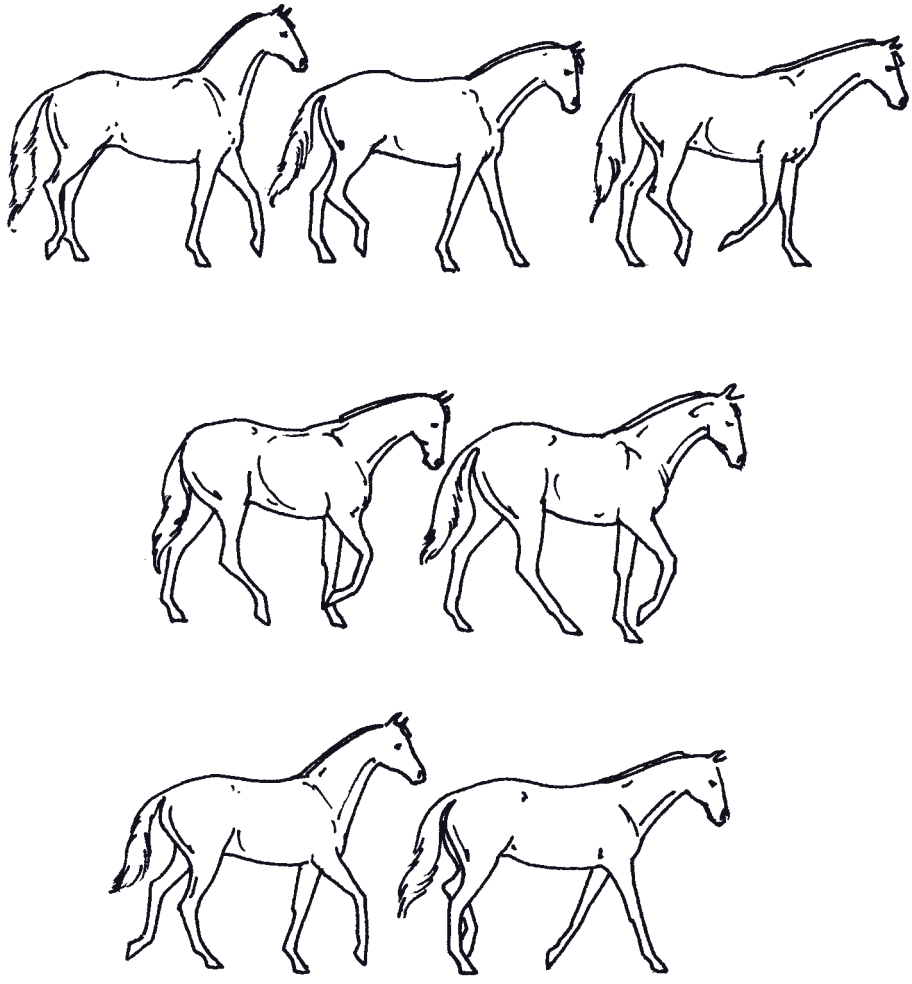

**Figure 2.2.** The walk.

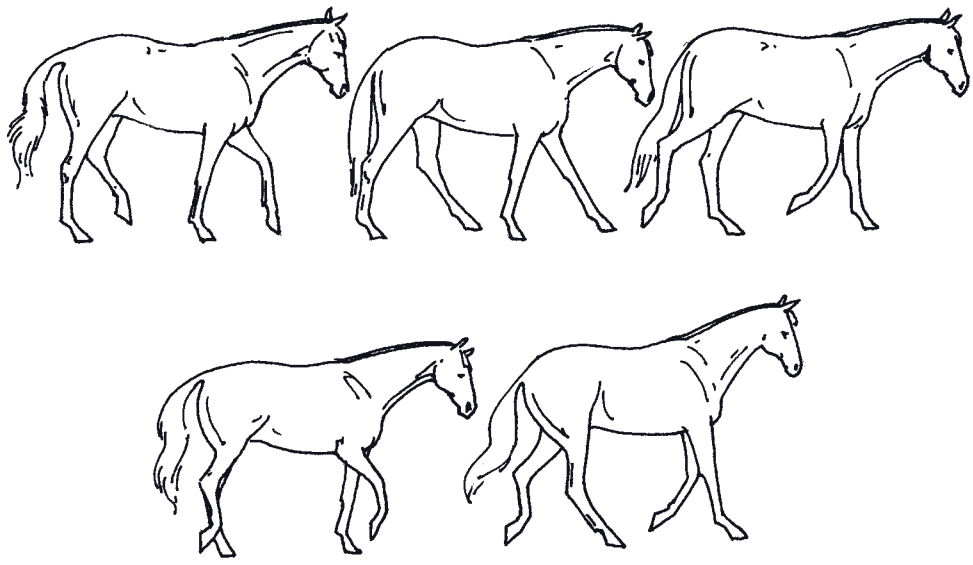

**Figure 2.3.** The pace.

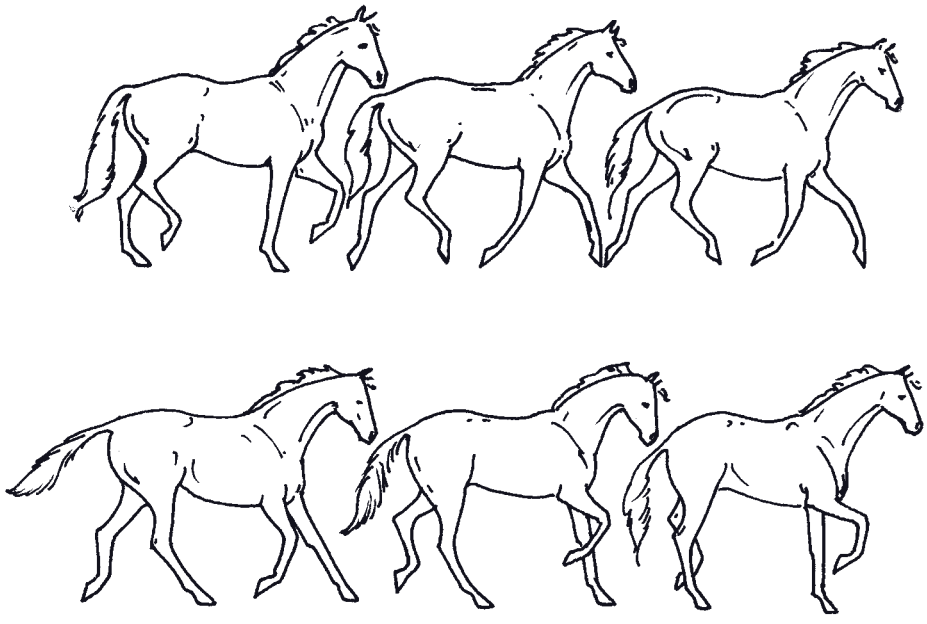

**Figure 2.4.** The trot.

left limbs. The trot or jog is a two-beat diagonal gait in which the right fore and left hind rise and fall together alternately with the diagonal pair left fore and right hind (Figure 2.4). Often, the trot is a horse's steadiest and most rhythmic gait. The canter or lope (Figure 2.5) is a three-beat gait with the following sequence: one hindlimb, then the other hindlimb simultaneously with its diagonal forelimb, and finally the remaining forelimb. If a horse is on the right lead, the initiating hind will be the left hind, the diagonal pair will be the right hind (sometimes referred to as the supporting hind) and the left fore, and the final beat will occur when the leading forelimb (the right fore) lands. Then there is a moment of suspension as the horse gathers its limbs under-

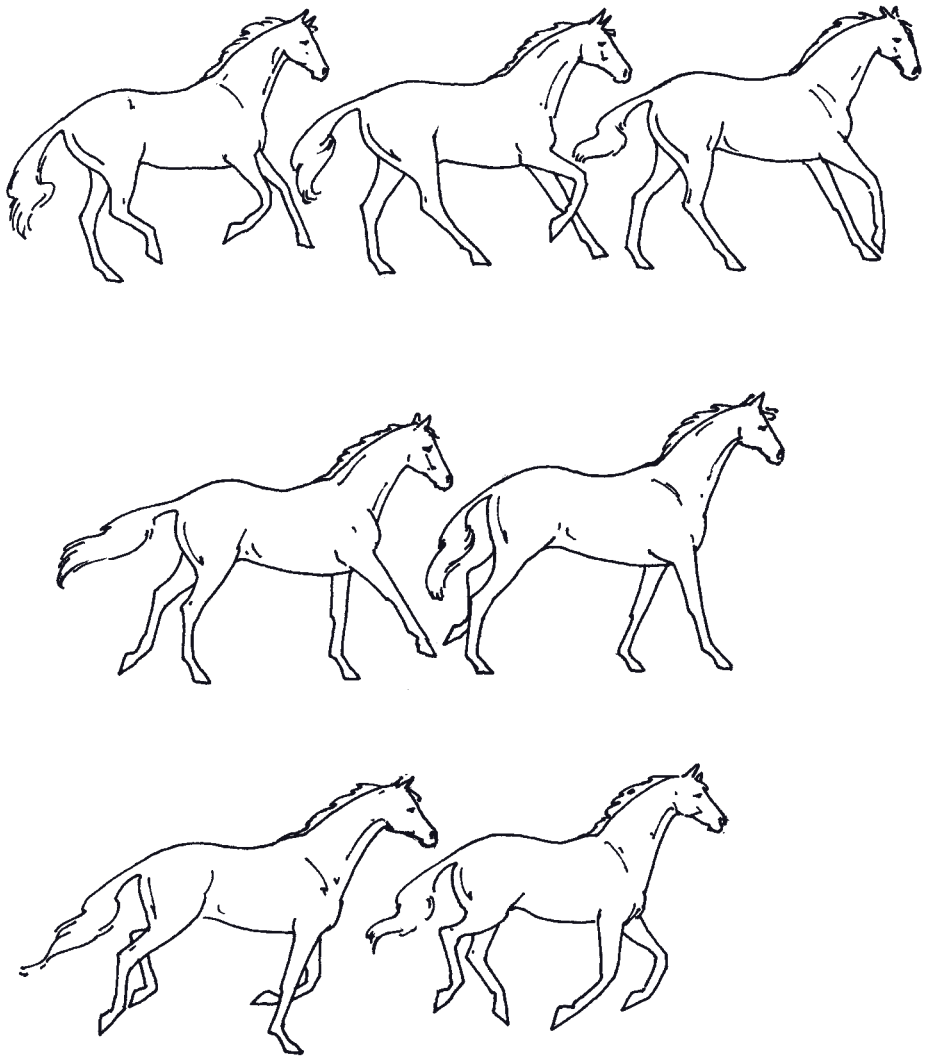

**Figure 2.5.** The canter, right lead.

neath itself to get organized for the next cycle. When observing a horse on the right lead from the side, it is evident that the right limbs will reach farther forward than the left limbs. A change of lead can occur during the moment of suspension so that the horse can change both front and hind simultaneously. The gallop or run is a four-beat variation of the canter (Figure 2.6). With increased impulsion and length of stride, the diagonal pair breaks, resulting in four beats. The footfall sequence of a right lead gallop is left hind, right hind, left fore, and right fore. As in the canter, the right limbs will reach farther forward than the left limbs when the horse is in the right lead. There is more suspension at the gallop than at the canter.

### *The Phases of a Stride*

The character of the stride of a limb is important to the diagnosis of lameness. The five phases of a horse's stride are landing, loading, stance, break-over, and swing (Figures 2.7 to 2.9).

#### Landing

During the landing (Figure 2.7) the hoof touches the ground, and the limb begins to receive the impact of the body's weight.

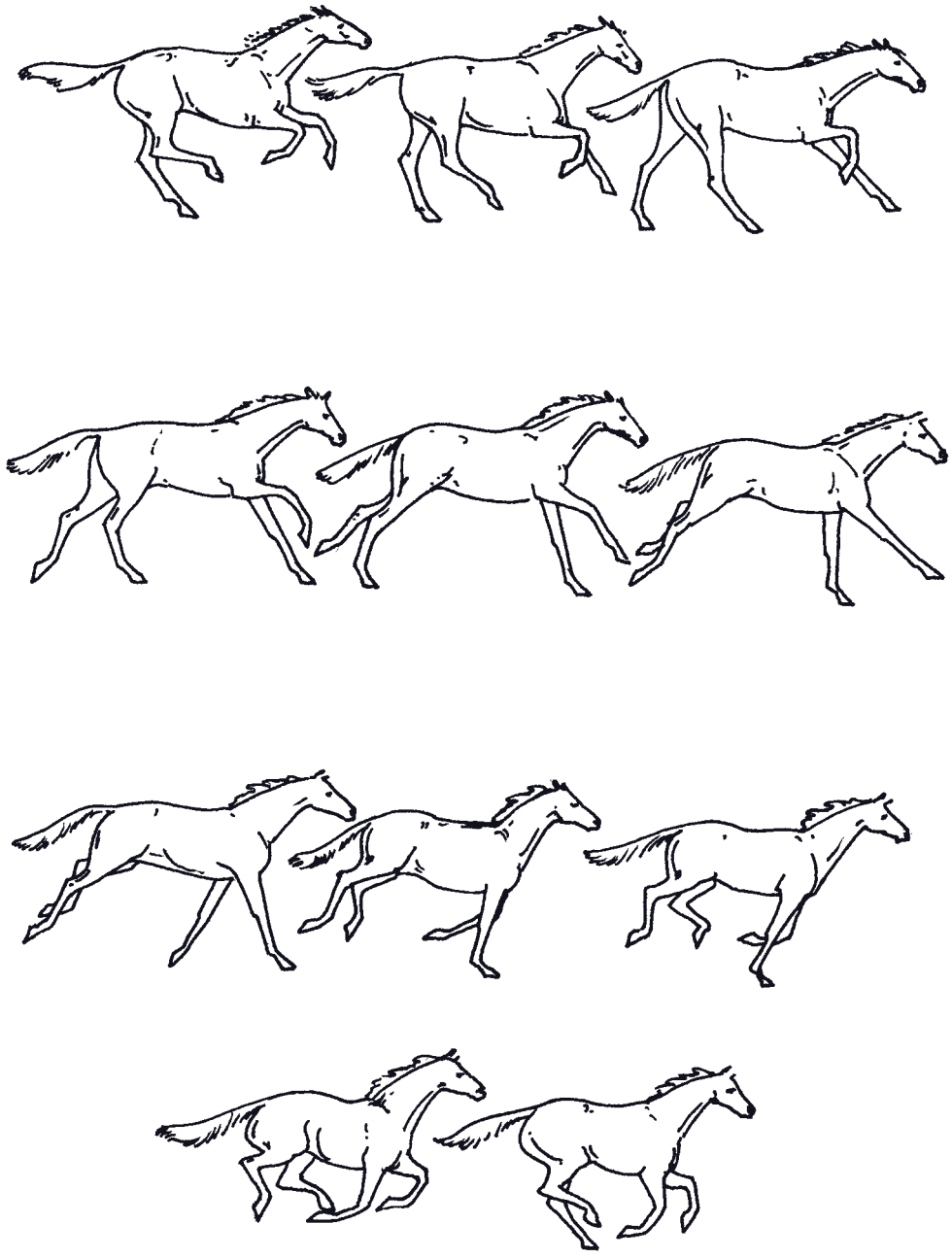

**Figure 2.6.** The gallop, right lead.

### Loading

The body moves forward during loading (Figure 2.7), and the horse's center of gravity passes over the hoof. Usually, this is when the fetlock descends (extends) to its lowest point, sometimes resulting in an almost horizontal pastern. The geometry of the foot also changes when loaded; the heels expand and sink caudally, and the toe retracts.

### Stance

During the stance (Figure 2.8) the fetlock rises to a configuration comparable to the horse's stance at rest. The transition between the loading

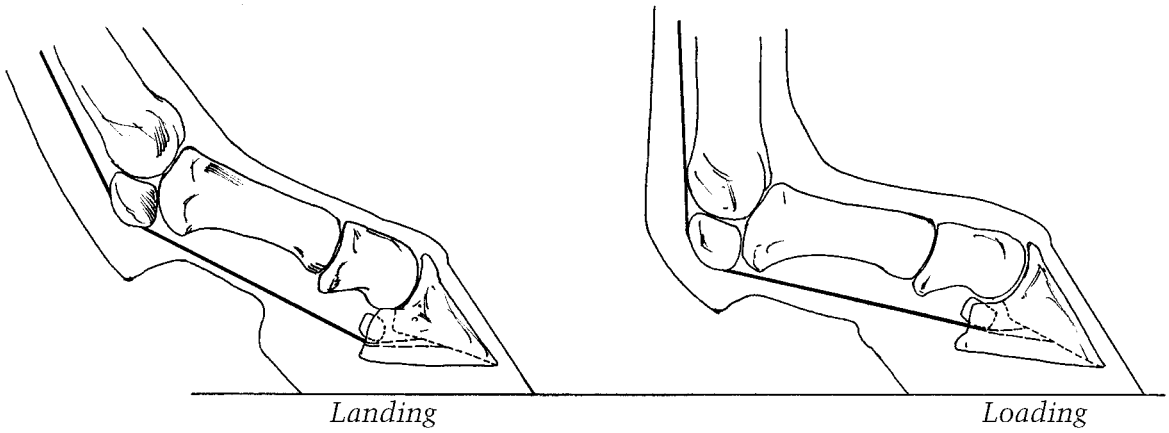


**Figure 2.7.** Phases of the stride: landing and loading. Reprinted with permission from Hill C, Klimesh R: 2009. *Horse Hoof Care*. North Adams, MA, Storey Publishing.

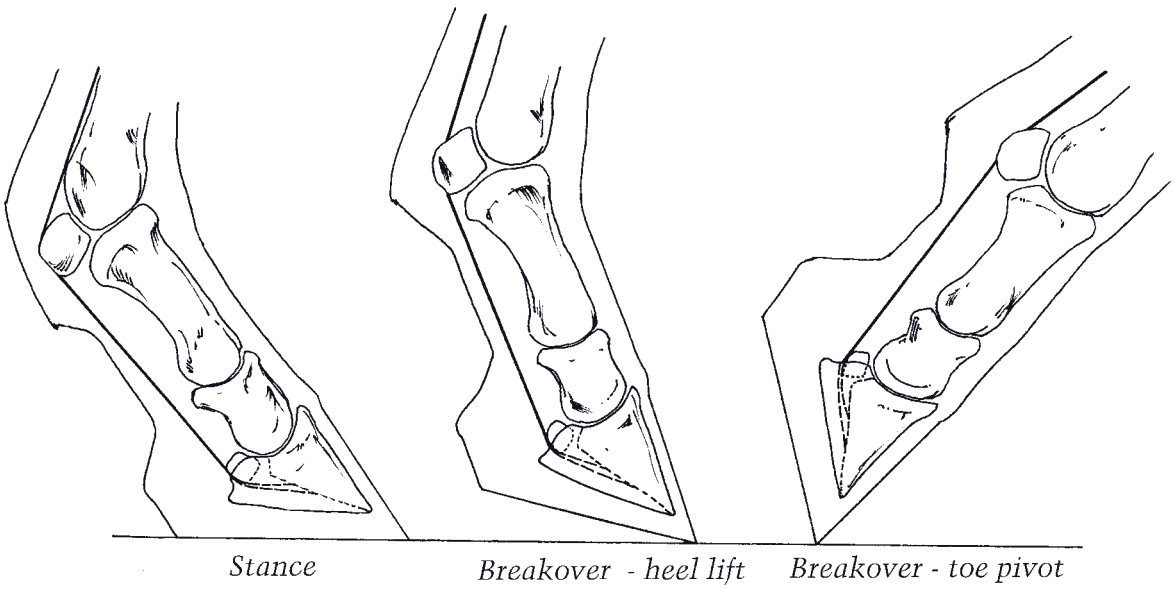


**Figure 2.8.** Phases of the stride: stance, breakover-heel lift, breakover-toe pivot. Reprinted with permission from Hill C, Klimesh R: 2009. *Horse Hoof Care*. North Adams, MA, Storey Publishing.

phase and the stance phase can be stressful to the internal structures of the hoof and lower limb. The horse's center of gravity moves ahead of the hoof. The flexor apparatus lifts the weight of the horse and rider, and the fetlock begins to move upward. The pastern straightens and the limb begins to push off the ground.

### Breakover

Breakover (Figure 2.8) is the phase when the hoof leaves the ground. It starts when the heels lift and the hoof begins to pivot at the toe. The carpus (or hock) relaxes and begins to flex. Breakover is measured from the time the heels leave the ground to the time the toe leaves the ground. The onset and duration of breakover are sensitive to changes in hoof balance, especially hoof angle and toe length. The palmar/plantar soft tissue structures are stretched just prior to the beginning of Breakover to counteract the downward pressure of the weight of the horse's body.

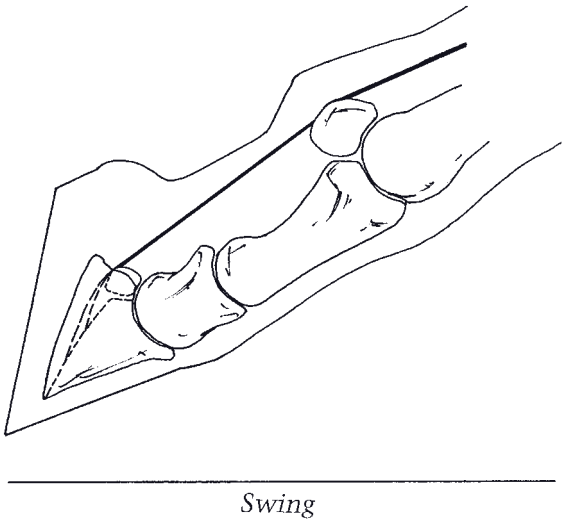


**Figure 2.9.** Phases of the stride: swing. Reprinted with permission from Hill C, Klimesh R: 2009. *Horse Hoof Care*. North Adams, MA, Storey Publishing.

### Swing

During the swing phase (Figure 2.9), the limb moves through the air and straightens out in preparation for landing.

### Stride Length

The stride consists of a cranial phase and a caudal phase. The cranial phase (or length) of the stride is in front of the footprint of the opposite limb, and the caudal phase is behind it (Figure 2.10). With lameness, the cranial or caudal phases may be shortened. If the cranial phase is shortened, there may be a compensatory lengthening of the caudal phase, and vice versa. Alterations in the length of the stride are best viewed from the side of the horse. Potential causes for a shortened cranial phase of the stride include bilateral lameness such as navicular syndrome, upper limb lameness such as in the shoulder or hip, and bone spavin. In general, changes in the cranial phase of the stride are more easily observed than changes to the caudal phase.

Leaving the toe of a hoof long has been thought to increase a horse's stride length, thereby contributing to a smooth and efficient stride and fewer strides over a given distance. However, it has been shown that horses with long toes and an acute hoof angle do not take longer strides. Long toes move the pivot point of the hoof farther forward than normal. The long toe acts as a lever arm during breakover, making

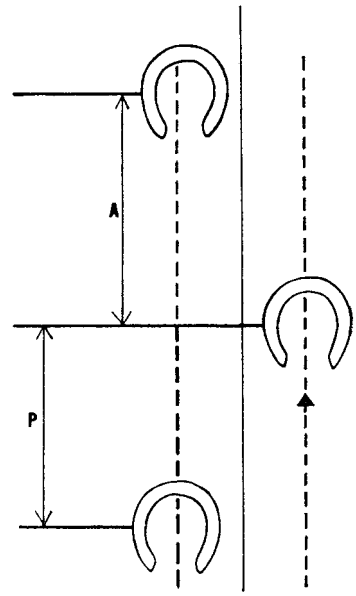


**Figure 2.10.** Phases of the stride. The cranial phase of the stride (A) is the half of the stride in front of the print of the opposite foot. The caudal phase of the stride (P) is the half of the stride in back of the print of the opposite foot.

it more difficult for the heels to rotate around the toe; consequently, tension in the deep digital flexor tendon (DDFT) and navicular bone ligaments may be prolonged and/or exaggerated. The delayed breakover permits the mass of the horse's body to move farther forward over the horse's limbs before the limbs leave the ground. To support this further, rolled toe shoes have been shown to improve the ease of movement and to lower peak loading of the distal limb during breakover.

## *The Forelimbs*

Detecting lameness in the forelimb usually is more straight forward than in the hindlimb. Visual observations that may be used to detect forelimb lameness are listed in Box 2.1. Most forelimb lameness is best viewed from the side, paying close attention to head and neck movement, shoulder musculature, and length of the stride. With an obvious lameness in a forelimb, the head will drop when the sound foot lands and rise when weight is placed on the unsound foot or limb. However, the severity of head and neck movement may be greatly attenuated in horses with bilateral forelimb lameness (such as those with navicular disease). If trotted on a hard surface, the sound foot will produce a louder

**Box 2.1. Visual observations that may be used to detect lameness in the forelimb.**

1. Head and neck movement or "head nod": head up on the lame limb or head down on the sound limb
2. Tensing of the shoulder musculature: shoulder of the lame limb fixes or "props" just before it hits the ground
3. Alterations in the height of the foot flight arc
4. Alterations in foot flight (paddling, winging-in, etc.)
5. Length of stride (cranial or caudal phase)
6. Joint flexion angles
7. Degree of fetlock extension or "drop" with weight-bearing
8. Front-to-back rocking of torso
9. Gait asymmetry: asymmetrical side-to-side motion
10. Audible differences between lame and sound limbs

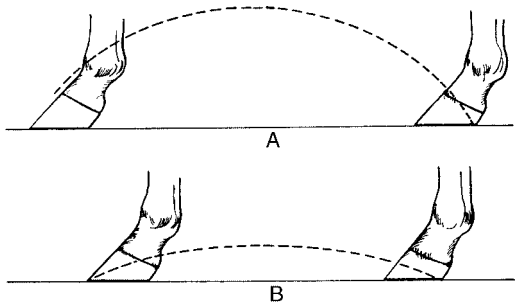


**Figure 2.11.** (A) Normal arc of the foot flight. (B) Low arc of the foot flight caused by lack of flexion in either the forelimbs of hindlimbs.

sound when it contacts the ground. Additionally, splinting of the caudal neck and shoulder muscles in anticipation of weight-bearing on that limb may be seen, and a reduced "fetlock drop" may occur due to less weight placed on the lame limb. The cranial phase of the stride is often reduced with bilateral forelimb lameness and with problems in the upper limb (shoulder).

The arc that the foot makes in flight may suggest the site of pain in the limb (Figure 2.11). If the foot flight is too low in the forelimb, there may be interference with flexion of the shoulder, carpus, or fetlock due to pain or mechanical injury. Fixation of these joints will reduce the arc of the foot flight, limit the cranial phase of the stride, and lengthen the caudal phase. With

**Box 2.2. Observations that may be used to detect lameness in the hindlimb.**

1. "Pelvic hike or rise": upward movement of pelvis or hemi-pelvis and croup on lame limb; unweighting of lame limb and shifting of weight to sound limb
2. Excess vertical displacement of tuber coxae or croup (more movement on one hip than the other)
3. Alterations in height of arc of foot flight
4. Head and neck movement; head down on the lame limb, only in substantial hindlimb lameness and often accompanied by a pelvic hike resulting in a rocking horse motion
5. Length of stride (cranial or caudal phase)
6. Gluteal rise and gluteal use
7. Hip hike or hip roll
8. Reduced flexion of lame leg (joint angles)
9. Degree of fetlock extension or "drop" with weight-bearing
10. Drifting away from lame limb

shoulder problems, the scapulohumeral joint usually remains semi-fixed when the limb is advanced (swing phase of stride), and the head may show marked lifting and may be pulled toward the unaffected side. With bilateral forelimb involvement, the gait often appears unanimated (stilted or stiff), giving the false impression of shoulder involvement.

### *The Hindlimb*

Detecting hindlimb lameness usually is more difficult than forelimb lameness and there is more confusion regarding what to observe for in horses with hindlimb lameness. Clinical observations that may be used alone or in combination to detect hindlimb lameness are listed Box 2.2. There is more subjectivity in evaluating hindlimb lameness than forelimb lameness and many clinicians likely observe the same thing but refer to it as something different. For instance, the rapid elevation of the hip and gluteals recognized as a "hip hike" or "upward flick" on the affected side is most likely the same thing as the "pelvic hike" that others refer to. One set of clinicians prefers to focus on what happens on the lame limb (one side of the pelvis), while others prefer to look at the entire pelvis or hemi-pelvis. In

both scenarios, the horse is attempting to get off the lame hindlimb more quickly, shift weight to the opposite hindlimb, and unweight the limb. This will usually cause upward movement of the pelvis and croup, gluteal muscles, and tuber coxae, resulting in a "pelvic hike," "hip hike," "gluteal rise," "upward flick," and more movement of the tuber coxae. All of these terms most likely describe the same observation that is similar to what we do when we are lame. However, it is important to remember that the upward movement of the pelvis, hemi-pelvis, hip, or tuber coxae in hindlimb lameness is the clinical impression of the change in height and not necessarily the absolute or measured height.

In addition, most horses that are trying to unweight a hindlimb will have reduced gluteal muscle contraction, which may lead to a shortened duration of gluteal use and a subsequent "hip roll" or hip "drop off." The stance phase will be shortened, giving the impression that the hip drops more on the lame limb. In a study of hindlimb lameness by May, the lame hip often was not elevated above the hip on the sound side (six cases were elevated, seven cases were not elevated). In fact, the midpoint of the vertical displacement for each hip marker was lower in the lame limb in all 13 cases. However, the croup (gluteal rise) was always higher when the lame limb started to bear weight. This may explain the confusion as to whether the hip hikes up or drops off in the lame hindlimb. Some clinicians prefer to place markers (usually tape) on the tuber coxae or gluteal muscles and observe for asymmetrical movement between the hindlimbs. The limb with more total movement as viewed from behind is usually the lame limb.

Observation of pelvic hike or movement of the hemi-pelvis is often best performed from the side in a similar manner to observing head and neck movement. A line or marker at a constant height in the background can be a useful reference point to view upward and downward movement of the head and pelvis. The severity of pelvic hike is usually proportional to the severity of the lameness, and therefore pelvic hike may be difficult to observe in horses with subtle lameness. The severity of pelvic hike also may be attenuated in horses with bilateral hindlimb lameness. Observation of subtle pelvic hike can sometimes be facilitated by observing the horse from the front using the horse's topline as a frame of reference. However, objective lameness evaluations of lame horses suggest that pelvic hike may occur only in horses with significant hindlimb lameness.

The arc of the foot flight in the hindlimb is best viewed from the side (Figure 2.11). Problems with the hock and stifle generally reduce the arc of the foot flight and thereby shorten the cranial phase of the stride with a compensating lengthening of the caudal phase. Because of the reciprocal apparatus of the hock and stifle, incomplete limb flexion is characteristic of involvement of both joints. The toe also may be worn excessively with involvement of the hock or stifle.

Although head and neck movements can be observed from the rear, they are best viewed from the side at the trot. With mild hindlimb lameness, abnormal movements of the head and neck are usually not evident. In moderate to severe lameness, the head and neck will rise as the unaffected hindlimb contacts the ground and lower when the affected hindlimb contacts the ground. In severe cases, horses will not only lower their head and neck, but will also extend their head. The lowering of the head and neck reduces the weight placed on the affected hindlimb when it contacts the ground. Movement of the head and neck in horses with severe hindlimb lameness is nearly always accompanied by a pelvic rise, producing a "rocking horse" type of motion.

### Forelimb vs. Hindlimb Lameness

When observing head and neck movement at the trot, one must be cautious not to confuse a left hind lameness with a left fore lameness, or a right hind lameness with a right fore lameness. In most cases a hindlimb lameness will mimic a forelimb lameness on the ipsilateral side, not the diagonal forelimb. This could occur when a hindlimb is lame at the trot because the horse will often land more solidly on the sound opposite forelimb. For example, if a left hindlimb is lame at the trot, the horse will lower its head when the left hind and right fore land, taking more weight on the right fore. On a hard surface, this gives the impression that the horse is yielding or has upward head movement on the left fore, suggesting lameness in that limb. Keegan states that an ipsilateral fore- and hindlimb lameness suggests a primary hindlimb lameness, whereas contralateral fore and hind lameness suggests primary forelimb lameness or lameness in both limbs. Careful attention to the pelvis and croup can help differentiate between a primary forelimb and primary hindlimb lameness. There should be minimal to no movement of the pelvis (no pelvic hike, hip hike, or gluteal asymmetry)

in horses with a forelimb lameness at a straight trot or when circled.

## ANATOMIC PROBLEM AREAS

It is generally considered that the majority of lameness problems in horses occur in the forelimb due to the increased weight bearing on the forelimbs (60 to 65%) and the shock of landing that the forelimbs absorb during movement. The hindlimbs propel the horse during movement and carry less of the overall weight. This results in greater concussion to the structures of the forelimbs. However, breed and use can alter this typical relationship. For instance, horses that pull carts or perform events such as dressage, cutting, and reining, which place greater stress on the hindquarters, often have a higher percentage of hindlimb lameness. Horses within various disciplines also may have different limbs that are affected most commonly. For instance, horses used for heading in team roping have more problems in the right forelimb, and horses used for heeling have more bilateral hindlimb lameness problems. Although there is considerable overlap, common lameness conditions that are associated with the specific type of work should be suspected first.

A large percentage of lameness in the forelimb will originate distal to the carpus. Problems within the foot predominate, but this may depend on the age, breed, and occupation of the horse. For instance, Arabians tend have fewer foot problems compared to other breeds, and young horses do not typically develop foot-related lameness. In addition, racehorses tend to have problems with high-motion joints, such as the fetlock and carpal joints, and the digital flexor tendons. Close evaluation of the distal limb should be performed in any horse with forelimb lameness before considering an upper limb problem, unless a more proximal condition is obvious.

Many hindlimb lameness problems involve the hock and/or stifle. This is especially true for Western performance horses, dressage horses, and Standardbred racehorses. In general, problems within the foot and pastern region are not common causes of hindlimb lameness. Exceptions to this include middle and proximal phalangeal fractures that occur in Western performance horses, and problems within the digital flexor tendon sheath, which tend to occur more frequently in the hindlimb than the forelimb. In contrast, tendinitis of the digital flexor tendons in the metatarsal region occurs less commonly in the hindlimb than forelimb, but suspensory problems appear to be equally common in the hindlimb and forelimb.

In addition to different sites of lameness problems in the fore- and hindlimbs, it is important to remember that many joint-related conditions appear to begin on the medial aspect of the limb. This most likely reflects more weight-bearing in the medial bony column of the limb, or that repetitive concussion of the joint contributes to more damage medially than laterally. Examples include osteoarthritis (OA) of the proximal interphalangeal (PIP) joint; proximal phalanx osteochondral fractures in the metacarpophalangeal (MCP) joint; osteochondral fragmentation and OA in the carpal joints; OA in the distal tarsal joints; and subchondral cystic lesions, meniscal damage, and OA in the medial femorotibial (MFT) joint. High ringbone in the pastern commonly begins on the medial aspect of the joint, and joint space narrowing is frequently initiated medially.

Osteochondral fractures occur more frequently on the medial eminence than the lateral eminence of the proximal phalanx in the MCP joint and on the distal radiocarpal bone in the middle carpal joint. Severe OA affecting the carpometacarpal and/or other carpal joints frequently involves the medial side of the joint, contributing to a carpal varus deformity of the leg. In addition, bone spavin often begins on the medial aspect of the tarsometatarsal (TMT) and the distal intertarsal (DIT) joints, as evidenced both clinically and radiographically. The MFT joint of the stifle is by far the most clinically affected area of the stifle, with a multitude of problems occurring in this area. Because of the medial location of many of these problems, it is important to closely evaluate these areas both clinically and with diagnostic imaging to enable a more accurate diagnosis.

## OCCUPATION-RELATED PROBLEM AREAS

Knowing the occupation of the horse is very important in diagnosing the most likely cause of lameness. Specific jobs contribute to specific problems and knowing these possibilities can alter the management of the horse. For instance, diagnostic anesthesia is less commonly employed in racehorses because of the high occurrence of incomplete or "stress" type fractures that may become complete following anesthesia. In contrast, this risk is minimal in most Western performance, dressage, and endurance horses. In addition, fatigue-associated bone disease (stress

fractures) is common in all types of racehorses and therefore nuclear scintigraphy is an ideal diagnostic tool to determine the cause of lameness. In contrast, the diagnosis of more chronic bone disease or soft tissue injury, such as occurs within the axial skeleton in dressage or show horses, is less suitable for nuclear scintigraphy. Likewise, knowing common problem areas will facilitate a more thorough and complete physical examination that may help detect subtle abnormalities. Common lameness conditions associated with several occupations are presented in Table 2.1.

## EVALUATION OF FOALS WITH LAMENESS

Foals, by nature of their physical make-up, behavior, and environment, are particularly vulnerable to events resulting in lameness. There are multiple causes of lameness, including infectious and noninfectious etiologies. Because of the often rapid clinical progression of lameness with potentially life-threatening consequences, timely recognition along with accurate diagnosis and treatment are critical for a favorable outcome.

Signalment of the foal, including age, sex, breed, and the intended use, is necessary information. Husbandry information, such as housing and level of hygiene, turn-out schedules, terrain and environment, behavior of the mare and foal, and concentration of horses is useful. The medical history of the lame foal and others in the herd should be obtained. The general condition of the mare and presence of any medical or reproductive problems during pregnancy or parturition as well as possible insufficient immunoglobulin transfer to the foal is important information. The presence or absence of any febrile episodes and any alterations in the hemogram also are important. Evidence of any infectious disease on the premises and equine traffic on the farm should be noted.

The physical evaluation and locomotor assessment are the primary means of lameness isolation. It is important to evaluate the entire foal for its overall condition and development compared to other foals on the premises. Illness and/or physical conformation abnormalities also are relevant to correct diagnosis and treatment.

To avoid confusion between behavior response and pain response, it is important to begin palpation of the foal on a sound limb. Thorough evaluation of the limb should begin at the foot, proceed proximally to the shoulder or pelvis, and include the vertebral column. Inspection of the foot includes evaluation for changes in shape and integrity, presence of cracks or separation in the wall or sole, or swelling at the coronary band. Assessment should be made of the foot temperature and digital pulse amplitude in comparison to the contralateral foot. Compression of the foot to assess for pain may be made with the hand and fingers on young foals rather than with hoof testers (Figure 2.12). Care should be taken to evaluate the entire coronary band and all solar surfaces by application of pressure. Compression of the walls between the medial and lateral heels also should be routinely performed.

Moving proximally, any areas of swelling in the limb should be closely evaluated for character and consistency. Inspect for possible joint involvement, temperature alteration relative to surrounding areas, and pain on palpation and/or manipulation.

Isolation of the lame limb and the specific origin of pain may be challenging if the pain is

**Table 2.1.** Occupation-related lameness problems.

| Occupation | Lameness conditions |
|---|---|
| TB and QH racehorse<br>(Fatigue-associated repetitive overuse) | Foot bruising, quarter cracks, heel pain<br>Forelimb fetlock synovitis and fractures<br>Carpal synovitis and fractures<br>Bucked shins in young horses<br>Fatigue/stress fractures: MC/MT, P1, humerus, tibia, pelvis<br>SDFT tendinitis<br>Suspensory injuries<br>Coffin joint arthrosis: QH<br>Proximal suspensory desmitis: QH<br>Catastrophic fractures: sesamoids, P1, MC/MT, and humerus<br>Distal tarsitis/OA |

**Table 2.1.** (*Continued*)

| Occupation | Lameness conditions |
|---|---|
| STD racehorse<br>(hindlimb > forelimb) | Front feet bruising and corns<br>Hindlimb fetlock synovitis and fractures<br>Carpal synovitis and fractures ($C_3$)<br>Fatigue/stress fractures: similar to TB and QH but tibia most common<br>SDFT tendonitis<br>Suspensory injuries, especially PSD<br>Hock, stifle, and sacroiliac problems |
| Endurance horse | Muscle disorders including tying-up and muscle spasm, cramps, and strains<br>Forelimb and hindlimb suspensory desmitis<br>Foot bruising, pedal osteitis, and laminitis<br>Fetlock synovitis/OA<br>SDF tendonitis |
| Show/pleasure horse | Navicular syndrome/disease<br>Coffin joint synovitis/OA<br>Forelimb and hindlimb PSD<br>Distal tarsitis or bone spavin<br>Reverse angle of $P_3$ in hind feet<br>Lumbar and sacroiliac problems |
| Western performance horses (cutting, reining, roping, barrel racing, rodeo, gymkhana, and ranch horses) | Navicular syndrome/disease<br>Phalangeal fractures (primarily $P_2$)<br>Pastern ringbone<br>Fetlock and carpal synovitis/OA<br>Distal tarsitis or bone spavin<br>Stifle synovitis/OA<br>Forelimb and hindlimb PSD<br>Thoracolumbar myositis<br>Hindlimb muscle strains |
| Jumping/dressage/eventing horse | Navicular syndrome/disease including DDFT injuries<br>Suspensory branch injuries<br>Forelimb and hindlimb PSD<br>Fetlock synovitis/OA<br>SDFT tendonitis<br>Distal tarsitis or bone spavin<br>Stifle injuries<br>Thoracolumbar myositis: back problems<br>Sacroiliac problems |
| Draft horse | Hoof cracks and laminitis<br>Subsolar abscesses, canker, and thrush<br>Ringbone<br>Sweeny<br>Bone and bog spavin<br>Stifle synovitis/OA<br>Shivers and PSSM |

**TB, Thoroughbred; QH, Quarter horse; STD, Standardbred. $P_1$, first phalanx; $P_2$, second phalanx; $P_3$, third phalanx; MC/MT, metacarpus/metatarsus; $C_3$, third carpal bone; PSD, proximal suspensory desmitis; SDFT, superficial digital flexor tendon; OA, osteoarthritis; PSSM, polysaccharide storage myopathy**

**Figure 2.12.** Manipulation of the foot to locate the focus of pain in a lameness examination in a foal. Courtesy of Robert Hunt.

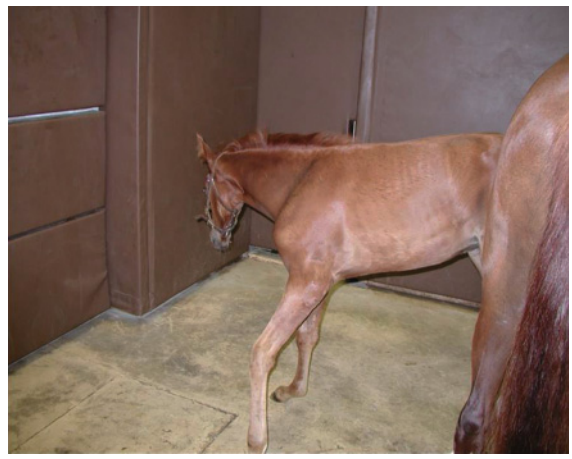

**Figure 2.13.** Typical dropped elbow appearance seen in fractures of the humerus and olecranon. Notice that the limb also is abducted and held in slight flexion. Courtesy of Robert Hunt.

subtle, but in general foals readily display locomotor evidence of the location of pain. Most foals attempt to avoid the painful component of the gait and usually display more exaggerated lameness reactions than adults. Observation of the young foal at rest and unrestrained in the company of the mare may be useful in detecting mild gait alterations or postural changes. Special attention should be given to the stance of the foal, noticing any trends in abnormal posture (Figures 2.13, 2.14). Young, untrained foals may require gait evaluation while unrestrained and following the mare. The gait should be assessed at a slow walk in a straight line and in turns in both directions. Placement of the foot as well as limb and body carriage during ambulation is important in localizing lameness. Although there are some common findings with certain musculoskeletal conditions, there are no absolutes regarding gait characteristics and location of lameness in foals.

Virtually all components of the musculoskeletal system are susceptible to injury and lameness. Intrinsic and external sources of trauma commonly result in structural compromise to tissue of the musculoskeletal system. Other causes of lameness include conditions associated with developmental orthopedic disease (DOD) and disorders of vascular or neurogenic origin. Infectious causes of lameness always should be suspected in foals less than four months of age, and especially in neonates. In general, lameness in foals is much more likely to be due to infection than in weanlings, yearling, or adults.

## PERFORMING A PREPURCHASE EXAMINATION

The purpose of a prepurchase examination is to evaluate the health and serviceability of a horse for a potential buyer. This information is used to establish a prognosis for the buyer relative to the intended use of the horse. The veterinarian must be capable of performing a thorough physical exam including soundness of limb, wind, and sight, and have an understanding of the intended use of the horse.

Prior to the exam, the veterinarian should review the process and goals of the exam with the buyer, providing specific details for the inexperienced buyer. It is desirable to have the buyer and/or buyer's agent and the seller and/or seller's agent all present for the examination. In reality, this may not be possible. If not, extra effort should be made to ensure that all parties receive accurate reporting of the examination information. It is reasonable to expect that the buyer has determined the horse to be suitable for its intended use, and the price for the horse has been established. The findings of the exam could affect the price or value of the horse; however, the examination should not be primary in the negotiation of the price.

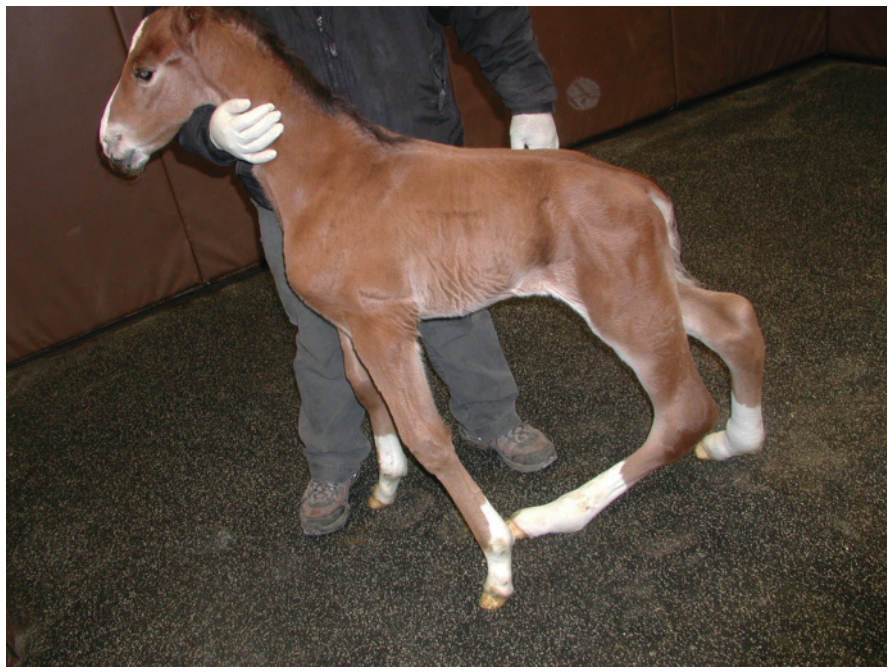

**Figure 2.14.** Excessive flexion of the hock in a foal with rupture of the left gastrocnemius muscle. Courtesy of Robert Hunt.

The choice of veterinarian to conduct the exam should be discussed. To avoid a potential conflict of interest, the veterinarian must be neutral in the transaction. In other words, he should not be the regular veterinarian for the horse or for the seller, and in no position to benefit from the sale of the horse. In many situations the best veterinarian to perform the exam is involved professionally with the horse or the seller because there may not be another qualified veterinarian in the area to do the exam. The veterinarian's involvement with the specific discipline also can make him/her uniquely qualified. In some situations the veterinarian works directly for both the buyer and seller on a regular basis.

The location for the examination requires adequate space to move the horse at the walk, trot, and canter. The surface should be firm and smooth with the option for a second softer arena-like surface. If possible, it is helpful to observe the horse performing under saddle but this is not always an option. Close proximity to a veterinary facility with diagnostic equipment is very helpful. If there is a compromise of the exam due to the facilities, this should be pointed out to the buyer and he/she should be given the option to arrange for a more suitable environment.

## *Musculoskeletal Exam*

In theory, the same examination is performed for all horses without regard for the price or the respective discipline in which the horse will be used. However, each exam carries specific emphasis for consideration. For the young or inexperienced rider, the horse's attitude and respect for the rider becomes especially important. For cutting and reining horses, hindlimb soundness, particularly the stifle, is important. Horses jumping fences must be comfortable in their hocks for pushing off and in their front feet for landing. Horses running at speed and stopping or turning must be especially sound in their front feet, and in horses doing consistent repetitive maneuvers, such as dressage horses, close evaluation of soft tissue structures such as tendons and suspensory ligament is important.

As part of the initial exam, the horse should be observed from 360° degrees in a walk-around exam. The veterinarian should note posturing, particularly foot placement, attitude, character of respiration, muscle symmetry, and body conformation. Conformation should be regarded as two components: the body's form as it relates to its potential athletic use and the body's form as it relates to specific breed characteristics or style.

The exam continues with hands-on evaluation of the horse's body. The head is evaluated for symmetry. The neck is evaluated for muscle symmetry and any sign of cervical vertebrae or muscle pain. The neck is moved to demonstrate left and right flexion as well as dorsal extension and ventral flexion.

The front limbs should be palpated from the withers to the foot, observing for pain response and muscle symmetry with the legs. All joints are examined for evidence of enlargement, excessive synovial effusion, pain, and range of motion. Tendons and ligaments are palpated both weighted and unweighted, noting any enlargement or pain. The foot is carefully evaluated for shape, wall angle, toe length, frog size, and comparison to the opposite foot. Hoof tester evaluation is performed after the horse has been examined in motion. The hindlimbs are then palpated and evaluated similar to the forelimbs. Palpation of the back and pelvis often are best done after the legs are examined, giving the horse time to relax and become confident with the process. In some breeds the tail function should be evaluated for evidence of surgery or previous injections to limit tail function. Gentle anal stimulation of a normal horse will cause it to raise its tail. From the side view the tail should rise well above the horizontal plane with an arched profile.

As the exam proceeds, any abnormalities are noted in the report for further consideration. The collection of these facts and their relative importance to the entire context of the exam are usually evaluated at the end of the exam. Certainly there may be a point anywhere in the exam at which a condition or combination of conditions is serious enough to warrant stopping the exam immediately.

Flexion tests are then performed on all four legs. This usually includes distal limb, fetlock, carpal, and upper limb flexion tests in the forelimb, and distal limb, hock, and stifle flexions in the hindlimb. Any changes in gait are noted. In some cases the veterinarian may feel it is necessary to perform other stress tests based on the observations during the exam. These could include shoulder range of motion, hindlimb abduction and adduction tests, or the wedge navicular test.

The feet should be examined with the hoof testers, often after evaluation in motion. If there are pads present, removal of the shoes is required. If there are considerations for not removing the shoes and pads, the buyer must be told of this compromise. The foot is squeezed from side to side, each heel to opposite toe quarter, frog from the lateral sulci to the opposite wall and the medial sulci to the opposite wall, each bar to respective the hoof wall, and perimeter of the sole as well as the central sole area. Any abnormalities and the degree of pain response are noted.

The other body systems are usually evaluated after the musculoskeletal system. These may include the cardiovascular, respiratory with consideration of the upper airway, ophthalmic, digestive tract including dental age, and reproductive systems if indicated. Neurological issues should surface during the lameness exam, and if needed, further diagnostics or referral may be indicated.

At this point a review of the examination information is discussed with the buyer. If there are no concerns, this may be the end of the prepurchase examination. Often, further diagnostics are indicated and often are performed. It is the veterinarian's responsibility to suggest any further diagnostics that are deemed important to fully assess the health status of the horse. These procedures may include radiographs, ultrasound, upper airway endoscopy, gastroscopy, rectal exam, blood chemistry and complete blood count, and testing for medications. Any procedures that carry potential physical risk to the horse require permission from the seller to proceed.

Radiographs of specific suspicious areas noted in the examination often are offered to the buyer. For some disciplines, radiographs of the front feet and hocks are routine in a prepurchase exam. These include hunters and jumpers and other horses that require consistent and regular work. Radiographs help to establish the current radiographic status as well as provide a baseline for future evaluations. In many cases previous radiographs of the horse can be evaluated in light of the current exam before deciding to take new radiographs.

### *Reporting the Results of the Examination*

The summary of the findings and the prognosis are reported to the buyer. The American Association of Equine Practitioners has a suggested form for reporting the findings of the prepurchase exam (Table 2.2). This information should be written down as well as discussed so everyone has the same report about the examination findings. The prognosis is reported relative to the task the horse will be performing. The prognosis can be expressed in percentages if

**Table 2.2.** Guidelines for reporting prepurchase examinations.

The AAEP recognizes that for practical reasons, not all examinations permit or require veterinarians to adhere to each of the following guidelines.

1. All reports should be included in the medical record.

2. The report should contain:
a. A description of the horse with sufficient specificity to fully identify it.
b. The time, date, and place of the examination.

3. The veterinarian should list all abnormal or undesirable findings discovered during the examination and give his or her qualified opinions as to the functional effect of these findings.

4. The veterinarian should make no determination and express no opinions as to the suitability of the animal for the purpose intended. This issue is a business judgment that is solely the responsibility of the buyer that he or she should make on the basis of a variety of factors, only one of which is the report provided by the veterinarian.

5. The veterinarian should record and retain in the medical record a description of all the procedures performed in connection with the purchase examination, but the examination procedures need not be listed in detail in the report.

6. The veterinarian should qualify any findings and opinions expressed to the buyer with specific references to tests that were recommended but not performed on the horse (X-rays, endoscopy, blood, drug, EKG, rectal, nerve blocks, laboratory studies, etc.) at the request of the person for whom the examination was performed.

7. The veterinarian should record and retain the name and address of parties involved with the examination (buyer, seller, agent, witness, etc.).

8. A copy of the report and copies of all documents relevant to the examination should be retained by the veterinarian for a period of years not less than the statute of limitations applicable for the state in which the service was rendered.

**Taken from the American Association of Equine Practitioners: www.aaep.org/purchase_exams.htm.**

desired. This is especially helpful for the inexperienced horse buyer. For example, a horse with minor problems could have an 80% chance of fulfilling the use as previously stated. Another case with a more serious problem could have a 50% chance of performing the intended use. This system provides the buyer with some relative risk factor, rather than using broad terms such as fair or good. The terms "pass" and "fail" are not appropriate for the veterinarian to use. The acceptance or rejection of the horse is the buyer's decision, based on the information provided by the veterinary examination.

The information from the examination is the property of the buyer and cannot be released to another party without permission of the buyer. The veterinarian must respect this level of confidentiality. If the horse is not purchased by the buyer it is reasonable to ask the buyer to release the details to the seller for her use as a courtesy for allowing the detailed evaluation of the horse.

In summary, the prepurchase exam is performed to provide the buyer with all available information regarding the health of the horse in question. The veterinarian's responsibility is to examine the horse and report the findings to the buyer as a prognosis for the horse to be serviceable with respect to the use intended.

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**Revised from "Examination for Lameness" in *Adams and Stashak's Lameness in Horses, Sixth Edition,* by Gary M. Baxter and Ted S. Stashak; "Lameness in Foals" by Robert J. Hunt; and "Guidelines for Prepurchase Examination" by Terry D. Swanson Robert J. Hunt, and Terry D. Swanson.**

3

CHAPTER

# Assessment of the Lame Horse

## HISTORY

A detailed medical history should be obtained on every horse. Records should include specific information regarding the duration and intensity of the lameness, the specific signs observed by the owner or trainer, the activity immediately preceding the lameness, and any previous treatments or therapies employed. The questions that should be asked in most cases include:

1. How long has the horse been lame?
2. Has the horse been rested since the initial lameness?
3. Has the lameness worsened, improved, or stayed the same?
4. Was the cause of the lameness observed?
5. Does the lameness worsen with exercise or does the horse warm out of the lameness?
6. What treatments have been initiated and have they helped?

## SIGNALMENT AND USE

Patient age and use are important considerations when determining potential lameness conditions (see Table 2.1 in Chapter 2). For example, an aged crossbred horse used for ranch work and occasional rodeo performance would most likely have problems associated with the fore feet and low-motion joints such as the pastern and distal tarsal joints. In contrast, a young racehorse often will have lameness problems associated with high-motion joints (i.e., carpus and fetlock), sprain/strain of flexor support structures, and stress-related fractures. Horses used for competitive trail or endurance riding often sustain sprain/strain injuries, tendinitis, and foot/hoof bruising. Young cutting horses appear to be prone to stifle issues, and any young horse just beginning training may become lame from developmental orthopedic-related problems. Additionally, foals less than four months of age are much more likely to have lameness associated with infection, and older foals are particularly prone to traumatic injuries causing fractures.

## VISUAL EXAMINATION AT REST

Often the initial part of a lameness evaluation is a visual examination of the horse standing squarely on a flat surface at rest. This should be done at a distance and then up close, viewing the horse from all directions. From a distance, the body type is characterized (stocky vs. slender) and conformation is noted; alterations in posture, weight shifting, and pointing also should be noted. It is important to look for changes in the contour of the limbs and asymmetry between limbs.

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

Under normal circumstances, the forelimbs bear equal weight and should be placed squarely under the horse. With bilateral forelimb involvement the weight may be shifted from one foot to the other, or both limbs may be placed too far out in front of the horse. In the hindlimbs, it is normal for the horse to shift its weight from one limb to the other. If the horse consistently rests one hindlimb and refuses to bear weight on it for any length of time, lameness in that hindlimb should be considered.

At close observation, each limb and muscle group should be observed and compared to its opposing member for symmetry. Feet are observed for abnormal wear, hoof cracks, imbalance, size, and heel bulb contraction (Figure 3.1). All joints and tendons and their sheaths are visually inspected for swelling and the muscles of the limbs, back, and rump are observed for swelling and atrophy. Comparing one side to the other is most important. Each abnormal finding should be ruled out as a cause of lameness during exercise and palpation examination. In the forelimbs, the limb with the narrowest (smallest) foot and highest heel is usually the lame or lamest (if the problem is bilateral) limb. The foot is smaller due to chronic alteration in weight-bearing, and extensor muscle atrophy may occur from a reluctance to extend that limb. For the hindlimb, atrophy of the middle gluteal and/or gracilis muscles usually indicates the lame limb (Figures 3.2, 3.3). Generally, if one tuber sacrale is higher than the other and/or the pelvis appears tilted, the horse will likely have an asymmetrical gait (Figure 3.4).

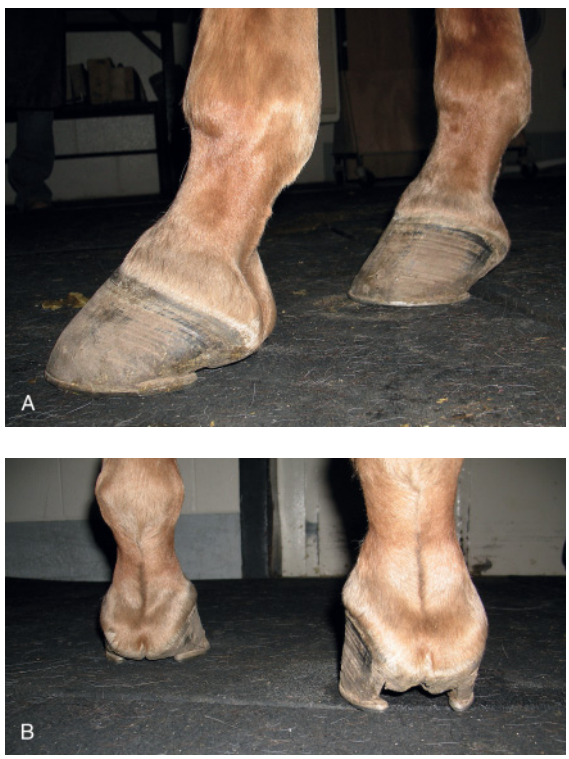


**Figure 3.1.** Lateral view (A) and dorsopalmar (B) views of both front feet in a horse with forelimb lameness. The front feet have markedly different hoof angles. The right foot has contracted heels resulting in a very upright conformation compared to a low under-run heel with a long toe on the left foot resulting in a low hoof conformation. The medial wall of the right foot is concave with the coronary pushed proximally, suggesting excessive concussion.

## PALPATION AND MANIPULATION

Palpation of the musculoskeletal system is a very important aspect of the lameness evaluation. With experience, subtle abnormalities can be detected that are often indicative of the site of the problem. The author usually performs a thorough palpation of the patient prior to observing it at exercise. Most manipulative tests are performed after exercise. A systematic approach of palpation is recommended to avoid missing abnormalities. The author usually palpates the forelimb proximally to distally with it bearing weight, and then distally to proximally with the limb picked up or unweighted. Palpation

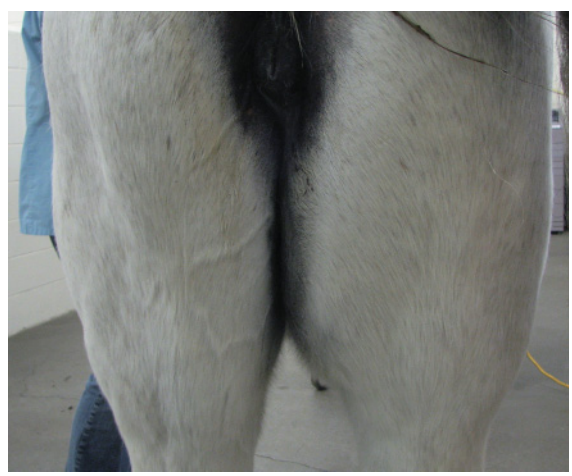

**Figure 3.2.** Example of atrophy of the inner and outer thigh muscles of the left hindlimb that occurred secondary to an upper hindlimb lameness.

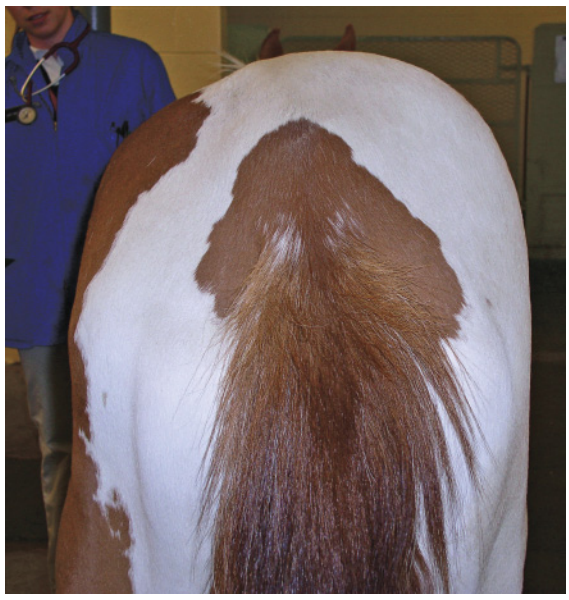

**Figure 3.3.** Example of atrophy of the gluteal muscles (left hindlimb) that often accompanies pelvic fractures in horses.

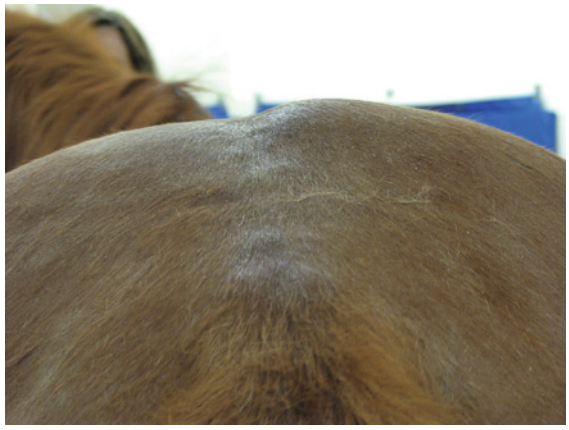

**Figure 3.4.** Rear view of the pelvis of a horse with a history of an acute onset of right hind lameness. Asymmetry to the sacroiliac region was visible and there was pain on firm palpation of the right tuber sacrale.

of the hindlimbs is performed in the same manner, paying close attention to the medial aspects of the stifle and tarsus. The back and axial skeleton are palpated last because some horses become agitated with manipulation of the back. Hoof tester examination of the feet is usually performed after the entire musculoskeletal system has been palpated or after watching the horse go.

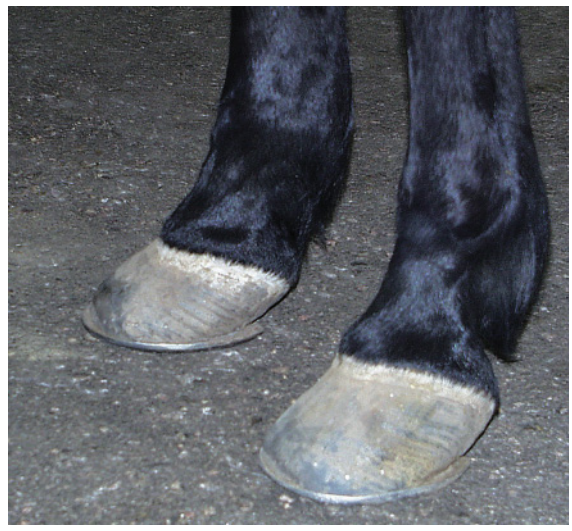

**Figure 3.5.** Concavity of the left front foot in a horse with chronic laminitis. This horse was most lame in the left forelimb.

The following discussion briefly describes how to visually examine and palpate the different anatomic regions of the equine musculoskeletal system. See the accompanying DVD for examples of palpating the musculoskeletal system.

### *Foot*

The size and shape of the foot on the lame limb should be compared to its opposite member. The examiner should look for asymmetry in foot size, abnormal hoof wear, hoof ring formation, heel bulb contraction, shearing of the heels and quarters, hoof wall cracks, coronet swellings, and foot imbalances. Asymmetry in foot size may be a result of trauma, lack of weight-bearing leading to contraction, and congenital or developmental defects.

In general, the limb with the smallest foot is usually the lame limb. Hoof wall ring formation can be unilateral (trauma) or bilateral (selenium toxicosis, laminitis, or systemic disease) and is not always associated with lameness (Figure 3.5). Heel contraction often results from decreased weight-bearing of the affected limb and is usually a symptom rather than the cause of the lameness (Figure 3.1). Visual examination of heel bulb contraction is best performed with the examiner standing or squatting near the flank and looking at both right and left heel bulbs at once (Figure 3.1B). Asymmetry in heel

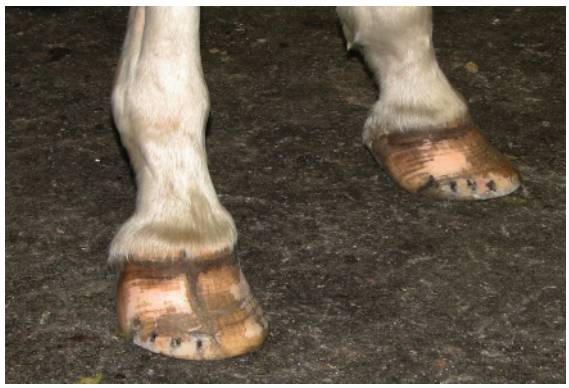

**Figure 3.6.** Partial thickness dorsal hoof crack associated with a long toe and a concavity of the dorsal hoof wall. Both factors most likely contributed to the development of the crack in this horse.

**Figure 3.7.** This horse presented for an acute onset hindlimb lameness. A nail was found protruding from the apex of the frog. Based on the location, entry into the navicular bursa would be unlikely.

bulb height (sheared heels) is most frequently associated with improper trimming and shoeing. Hoof wall cracks may or may not be associated with lameness but should be ruled out with hoof tester examination and in some cases nerve blocks (Figure 3.6). Swellings at the coronary band can result from superficial scar formation from wire cuts, constant bruising during exercise, or deeper involvement (e.g., gravel, keratoma, or quittor). Foot imbalances can be dorsopalmar/plantar (DP), lateral medial (LM), or a combination of the two (Figure 3.1). These imbalances often alter the shape of the hoof wall and can result in abnormal stresses applied to the foot and other support structures.

After superficial cleaning of the sole, abnormal wear of the shoe and/or sole, collapsed heels, heel bulb contraction, and frog atrophy should be noted. Secondary frog atrophy may accompany heel contraction in chronic cases. The shape of the sole should be observed. A slightly concave shape is normal. Some horses are flat-footed and, therefore, predisposed to sole bruising. Convexity dorsal to the apex of the frog ("dropped soles" in front of the frog) is considered abnormal and often is associated with rotation of the distal phalanx. In some cases, the offending cause of lameness may be identified immediately such as a nail wedged in the frog (Figure 3.7). However, the clefts of the frog may need to be opened with a knife to properly evaluate the depths of the sulci for evidence of thrush or canker.

A hoof tester is an instrument that permits deep palpation of the sole, frog, and wall of the hoof (Figure 3.8). When applied properly, the examiner tries to identify and localize hoof sensitivity. Most normal horses should be able to withstand a fair bit of hoof tester pressure without showing signs of discomfort.

The order of hoof tester application is less important than being systematic and complete. One method is to begin at the lateral or medial angle of the sole and continue hoof tester pressure at 2- to 3-cm intervals until the entire surface of the sole is checked. This is followed by applying pressure to the frog (caudal, central, and cranial) from both the medial and lateral heel. Finally, the hoof tester is applied to the hoof wall across the heels; it can then be applied diagonally from the medial heel to the dorsolateral hoof and then from the lateral heel to the dorsolateral hoof.

If sensitivity is encountered, it is necessary to confirm whether the response is from pain and not just a whimsical reaction by the horse. Repeatability is the key to confidence with the

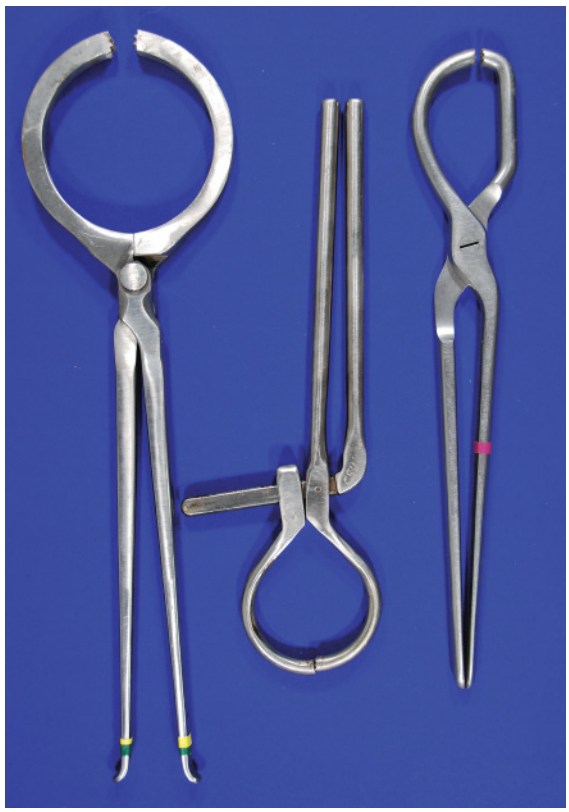

**Figure 3.8.** Examples of several types of hoof testers. Left, GE Forge and Tool Works, 959 Highland Way, Grover Beach, CA, 93433. Middle, Ryding Hoof Tester, Jorgenson Labs, 2198 W 15th St., Loveland, CO, 80537. Right, Kane Enterprises, AG-TEK Division, P.O. Box 1043, Sioux Falls, SD 57101.

findings. True sensitivity is identified by repeated intermittent hoof tester pressure that results in persistent reflexive withdrawal (flexing the shoulder) with hoof tester pressure. Varying amounts of hoof tester pressure are applied to elicit a response, and this depends on sole thickness and the painfulness of the condition. Hoof tester responses should be compared to those obtained from the opposite foot.

In general, diffuse sole sensitivity may suggest a sagittal fracture of the distal phalanx, diffuse pedal osteitis, or chronic laminitis. More localized hoof tester sensitivity usually is obtained with corns, sole bruising, puncture wounds, close or hot nail, and localized subsolar abscesses. Hoof tester sensitivity over the central third of the frog usually suggests navicular syndrome and/or sheared heels and quarters (Figure 3.9). Pain across the heels without pain over the frog

**Figure 3.9.** Hoof testers are applied over the central third of the frog of the fore foot to produce direct pressure over the navicular region. The author prefers the Ryding Hoof Tester.

may suggest bruised heels, sheared heels, contracted heels, or other problems related to shoeing or hoof imbalances. A hoof tester or a hammer also can be used to strike (percuss) the hoof wall. A painful response may suggest laminitis, gravel, or a painful hoof crack (most common in the toe and quarter).

The coronary band should be palpated for heat, swelling, and pain on pressure. A generalized increase in the temperature of the coronary band of both limbs is consistent with laminitis, whereas selective swelling with or without pain on deep palpation just dorsal and proximal to the coronary band may suggest effusion of the distal interphalangeal (DIP) joint. Firm, often nonpainful swelling in this region also may be evidence of low ringbone. Point swelling and pain with or without drainage at the coronet in the mid-quarter region may indicate an abscess along the white line or infection of the collateral cartilage of the distal phalanx. Heat, pain, and swelling with or without drainage of one of the heel bulbs often are found in horses with subsolar abscesses (Figure 3.10). Most penetrating wounds not involving the white line or navicular bursa that develop into an abscess eventually break out in the heel bulb region. In situations in which a small puncture hole in the sole has been identified, hoof tester pressure adjacent to the hole may force pus out of the hole, confirming the presence of a subsolar abscess.

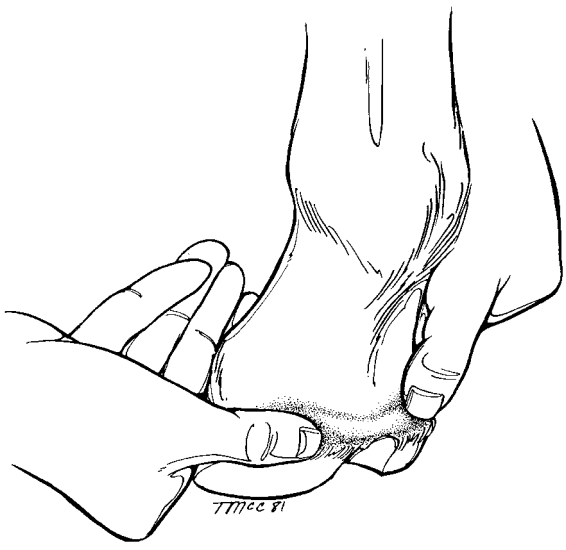

**Figure 3.10.** Palpation of the heel bulbs to identify heat, pain, and swelling that may be associated with subsolar abscesses.

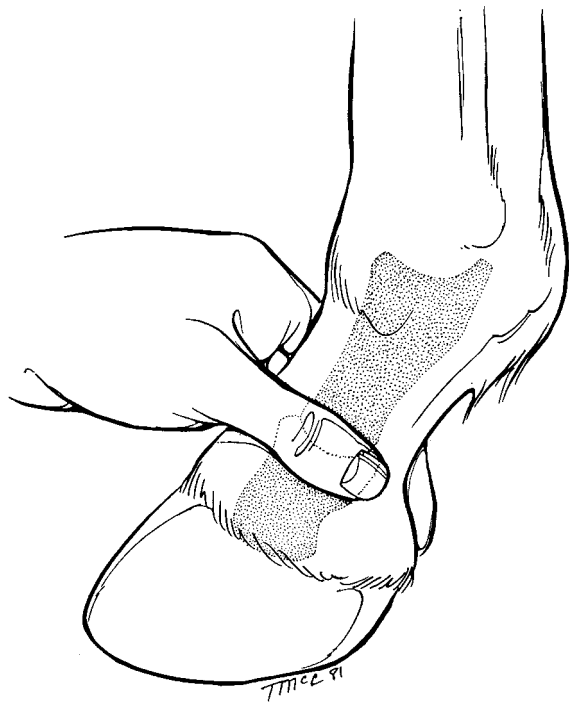

**Figure 3.11.** Palpation of the pastern. Thickening in this region may suggest the presence of ringbone.

### *Pastern*

The dorsal, medial, and lateral surfaces of the proximal interphalangeal (PIP) joint should be palpated for enlargement and heat, which may suggest high ringbone (Figure 3.11). Comparison to the opposite pastern is always recommended, but it is not uncommon for the lateral to medial dimensions of one pastern to be slightly larger than the opposite pastern in normal horses. With the limb off the ground, the distal sesamoidean ligaments and flexor tendons (superficial and deep digital flexors) are palpated deeply for pain, heat, and swelling (Figure 3.12). Particular attention is paid to the lateral and medial branches of the superficial digital flexor tendon (SDFT) as they attach to the middle phalanx. Tendinitis of the deep digital flexor tendon (DDFT) and/or or tenosynovitis of digital flexor tendon sheath (DFTS) often are identified by swelling, effusion, and sometimes pain in this region. Deep palpation of the lateral and medial eminences (wings) of the middle phalanx may elicit pain if a fracture is present. With the hands placed on the hoof wall, the phalangeal joints should be rotated medially and laterally (Figure 3.13). Pain often can be elicited in horses with osteoarthritis (OA) of the PIP joint or with proximal and middle phalangeal fractures.

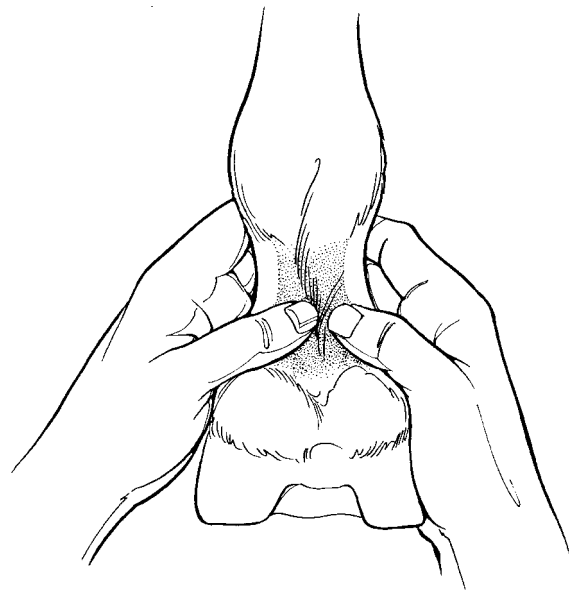

**Figure 3.12.** Palpation of the distal sesamoidean ligaments, branches of the SDFT, and the DDFT in the palmar/plantar aspect of the pastern.

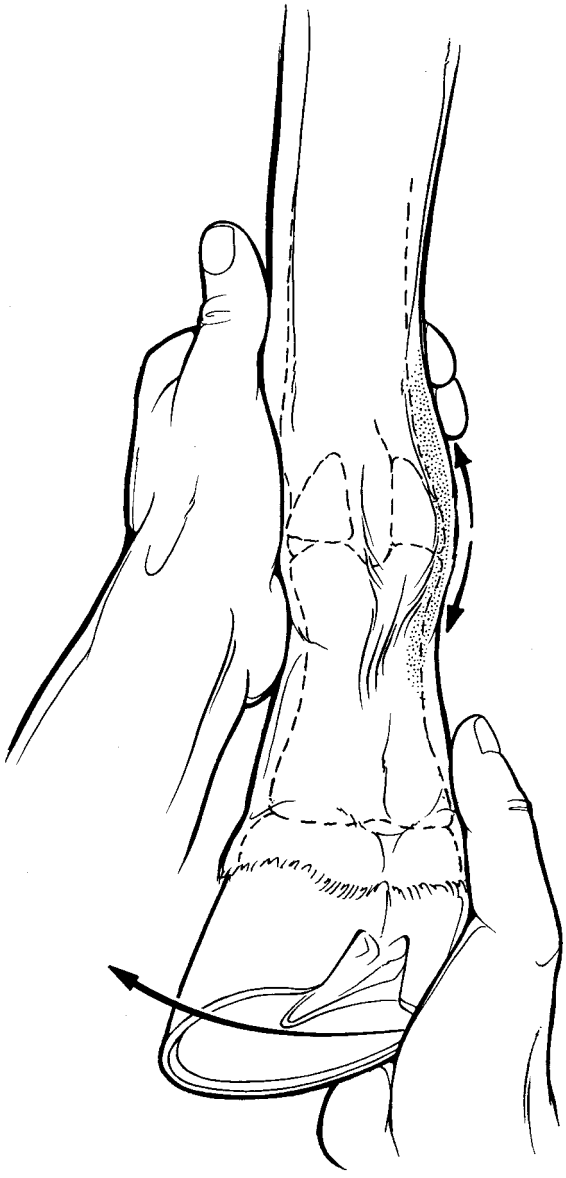

**Figure 3.13.** Tension is applied to the collateral ligaments supporting the fetlock and interphalangeal joints (pastern and coffin) to identify pain.

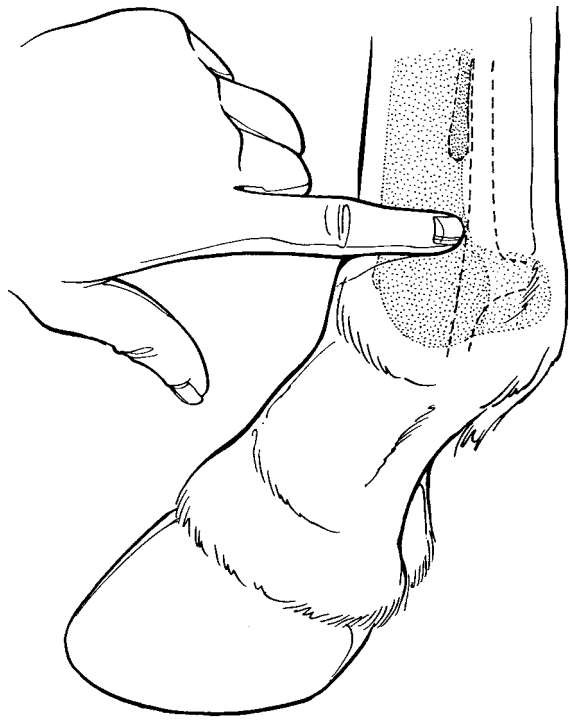

**Figure 3.14.** The finger marks the palmar recesses of the fetlock joint capsule. Distention at the site results from synovial effusion.

## *Fetlock*

The dorsal and palmar/plantar joint pouches of the metacarpophalangeal/metatarsophalangeal (MCP/MTP) joint should be palpated for swelling, effusion, or thickening of the joint capsule. These abnormalities may indicate idiopathic synovitis, chronic synovitis/capsulitis secondary to OA, or any type of articular fracture (Figure 3.14). Pressure should be applied to the lateral and medial branches of the suspensory ligament just above their attachments to the proximal sesamoid bones. Pain and swelling may indicate desmitis, sesamoiditis, or apical/abaxial fractures of the sesamoid bone. The SDFT, DDFT, and digital sheath should be palpated for heat, pain, swelling, or effusion (Figure 3.15). Some distention of the DFTS of all four limbs is not uncommon in performance horses. Often this is referred to as "wind puffs."

With the limb off the ground, thumb or finger pressure is applied to the basilar, body, and apical portions of the proximal sesamoid bones (Figure 3.16). Sensitivity and pain may indicate a sesamoid fracture or desmitis of the suspensory ligament. The fetlock is rotated and the collateral ligaments are checked in a manner similar to that of the pastern joint (Figure 3.13). Additionally, the fetlock joint should be passively flexed to identify pain and assess the range of motion. This is done by extending the carpus as much as possible with one hand, and then

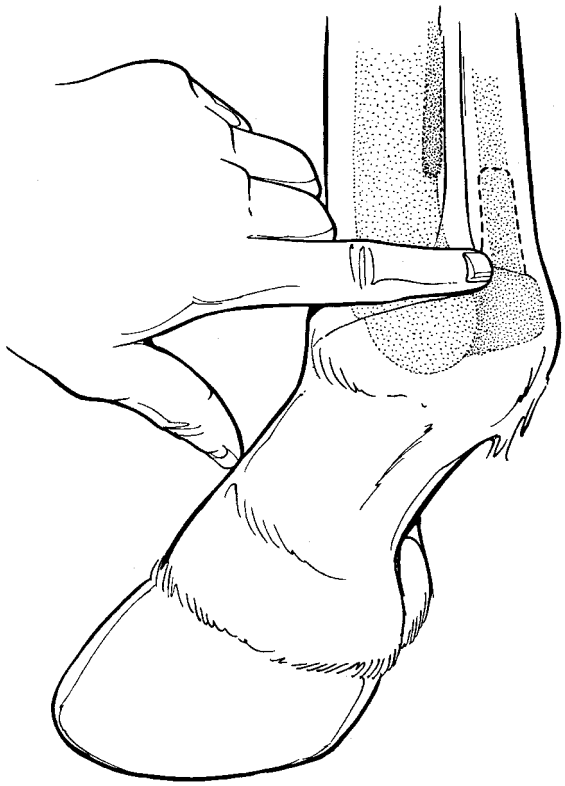

**Figure 3.15.** Palpation of the digital flexor synovial sheath around the superficial and deep digital flexor tendons is performed behind the branch of the suspensory ligament.

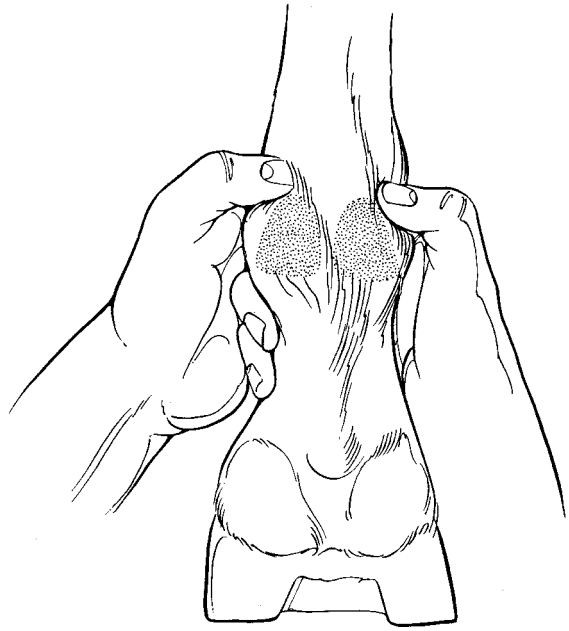

**Figure 3.16.** Digital pressure applied to the apical sesamoid region to detect pain, heat, and asymmetry. Palpation of the midbody and basilar aspects of the bones also should be performed.

flexing the fetlock with the opposite hand (Figure 3.17). This technique flexes the fetlock joint separate from the phalangeal joints in contrast to all of the joints of the distal limb (Figure 3.18).

### *Metacarpus/Metatarsus (MC/MT)*

Palpation of the dorsal aspect of the metacarpus should be performed, especially in racehorses. Firm pressure applied with the fingertips often elicits a painful response in horses with dorsal metacarpal disease (buck shins). Heat and swelling over the dorsal middle third of the metacarpus also may be present. (Figure 3.19). The extensor tendons on the dorsal surface of the MC/MT should be palpated for swelling, thickness, and pain, especially in horses with a history of trauma or laceration to this region. Scarring of the extensor tendon is more common in the MT than the MC and is often an incidental finding.

The entire length of each small MC/MT bone (splint bone) should be palpated with the limb weighted and unweighted. With the limb elevated, the palmar/plantar and axial surfaces of the splint bones can be palpated by pushing the suspensory ligament toward the opposite side (Figure 3.20). The splint bone can be palpated with the thumb, applying pressure as needed. Heat, pain, and swelling may indicate a fracture or a condition referred to as "splints" if located in the proximal aspect of MCII. Splint bone fractures most commonly involve the medial splint bone in the forelimb and the lateral splint bone of the hindlimb. A chronic splint bone fracture associated with excessive swelling and pain presenting with a history of recurrent drainage usually indicates infection or sequestration. It is not uncommon to palpate nonpainful enlargements of the splint bones, which are often incidental findings. However, many middle and proximal splint bone fractures heal with excessive callus that may contribute to lameness.

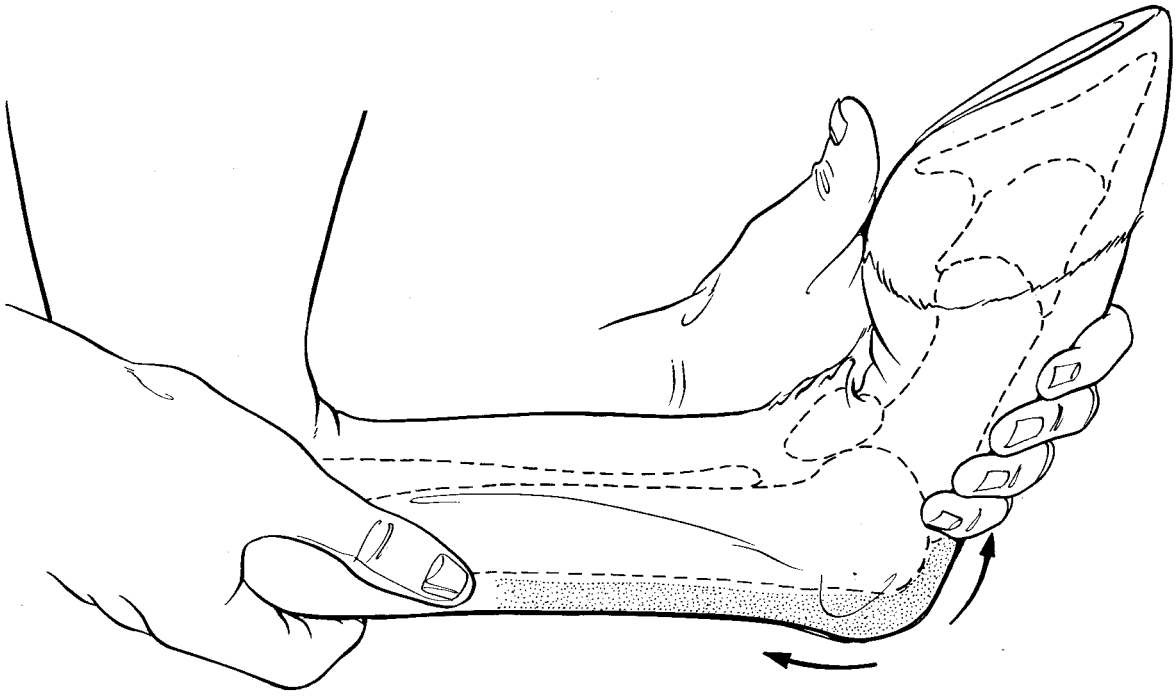

**Figure 3.17.** The fetlock flexion test is performed by extending the carpus and flexing the fetlock joint. One hand is placed on the dorsal aspect of the pastern to flex the fetlock without flexing the interphalangeal joints.

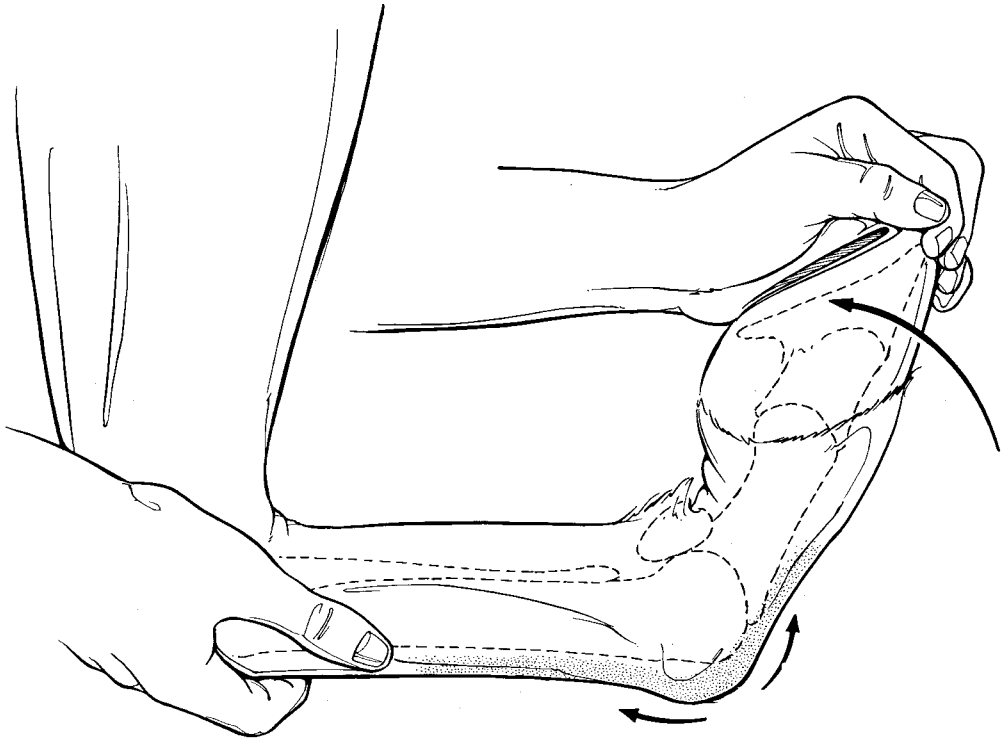

**Figure 3.18.** The distal limb flexion test, in which the interphalangeal (pastern and coffin) and fetlock joints are flexed simultaneously.

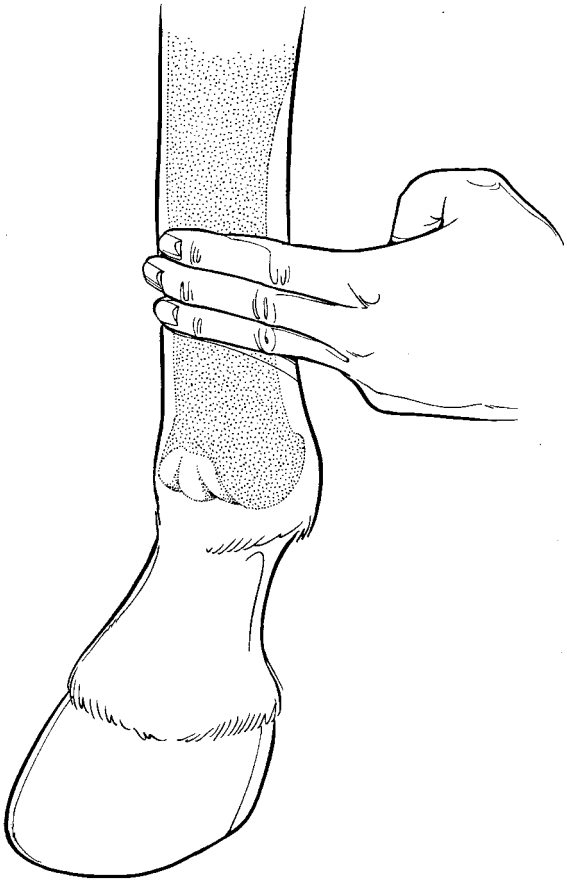

**Figure 3.19.** Palpation over the dorsal middle third of the metacarpus to identify heat, pain, and swelling associated with dorsal metacarpal disease.

### *Suspensory Ligament*

The suspensory ligament (interosseus medius muscle) lies just palmar/plantar to the splint bones in the MC/MT groove. It should be palpated with the limb bearing weight and with the limb flexed. Deep palpation often is needed to identify swelling and pain, and comparison of the lateral and medial branches may be helpful. Damage to the suspensory tends to occur distally within the branches of the suspensory ligament or at its proximal attachment to the MC/MT. However, secondary suspensory desmitis may be associated with a healing splint fracture anywhere along its length.

With the limb held in a flexed position, the proximal attachment of the suspensory ligament can be palpated by pushing the flexor tendons to the side and applying pressure with the thumb (Figure 3.21). Alternatively, pressure can be applied to this region by placing the palm of the hand on the dorsal MC and wrapping the fingers around the medial side of the MC. The fingertips are used to "squeeze" the limb and apply pressure to the proximal palmar MC region (Figure 3.22). Many horses may react initially by withdrawing (flexing) the limb. However, with constant pressure, this response often fatigues. A painful withdrawal that persists (does not fatigue) may indicate a problem at the origin of the suspensory ligament, inferior check ligament desmitis, or a fissure fracture of the proximal palmar metacarpus. Palpation of the proximal suspensory is more difficult in the hindlimbs than the forelimbs because the small metatarsal bones closely surround the ligament and the SDFT is less easily pushed to the side (Figure 3.23).

### *Inferior (Carpal) Check Ligament*

The inferior check ligament (accessory ligament of the DDFT) originates from the palmar carpal ligaments and attaches in a cup-like manner to the DDFT at about the middle of the metacarpus. It lies directly palmar to the suspensory ligament and can be palpated by holding the ligament between the index finger and the thumb or by applying pressure from the palmar aspect with the thumb.

### *Flexor Tendons*

The SDFT and DDFT are located palmar to the suspensory ligament and are intimately associated with each other. The proximal one-third of the flexor tendons (associated with the carpus) and distal one-third (associated with the fetlock) are encased in tendon sheaths, whereas the central one third is covered by a paratenon only. Each region should be palpated carefully for heat, pain, and swelling with the tendons weighted and relaxed. With the limb held in one hand, an attempt should be made to roll or separate the SDFT from the DDFT with the thumb and forefinger (Figure 3.24). If normal, they can be easily separated and differentiated. With pathology such as tendinitis, varying degrees of adhesions between the two as well as thickening will result in an inability to separate them. Most horses respond slightly to "pinching" the SDFT between the thumb and index finger. Pain will be elicited easily with palpation in most horses with tendinitis.

**Figure 3.20.** Palpation of the medial (axial) surfaces of the small metacarpal bones. The fetlock can be flexed to relax the suspensory ligament to permit easier palpation.

### *Carpus*

The carpus is visualized for swelling/effusion on the dorsal and palmar surfaces. Point swelling associated with the radiocarpal and middle carpal joints medial to the extensor carpi radialis tendon often is present in horses with osteochondral chip fractures and/or OA. More diffuse swelling of these joints may indicate more severe articular pathology such as slab fractures, advanced OA, and proliferative exostosis (carpitis). Distention of the tendon sheaths of the extensor tendons overlying the carpus (common digital and extensor carpi radialis) may indicate tenosynovitis and/or rupture, particularly of the common digital extensor tendon in foals (Figure 3.25). A diffuse fluctuant, subcutaneous swelling over the dorsal surface of the carpus, is consistent with acute hematoma/seroma or chronic hygroma (Figure 3.26). Swelling/effusion of the palmar carpal canal may be found with accessory carpal bone fractures, tenosynovitis (carpal tunnel syndrome), or osteochondroma formation of the caudal distal aspect of the radius.

Palpation of the carpal joints and bones, including the accessory carpal bone, are best done with the carpus flexed. The degree of carpal flexion or range of motion also should be evaluated. In the normal horse the flexor surface of the metacarpal region can approximate that of the forearm when the carpus is flexed (Figure 3.27). Carpal flexion should be performed slowly in horses with severe lameness with suspected carpal pathology to avoid a severely painful response. Reduced degrees of flexion with a painful response are consistent with most any intra-articular (IA) carpal problem and possibly desmitis of the proximal attachment of the suspensory ligament. After flexion, the carpus should be rotated by swinging the metacarpus laterally and medially. With the carpus held in flexion, the individual carpal bones are evaluated

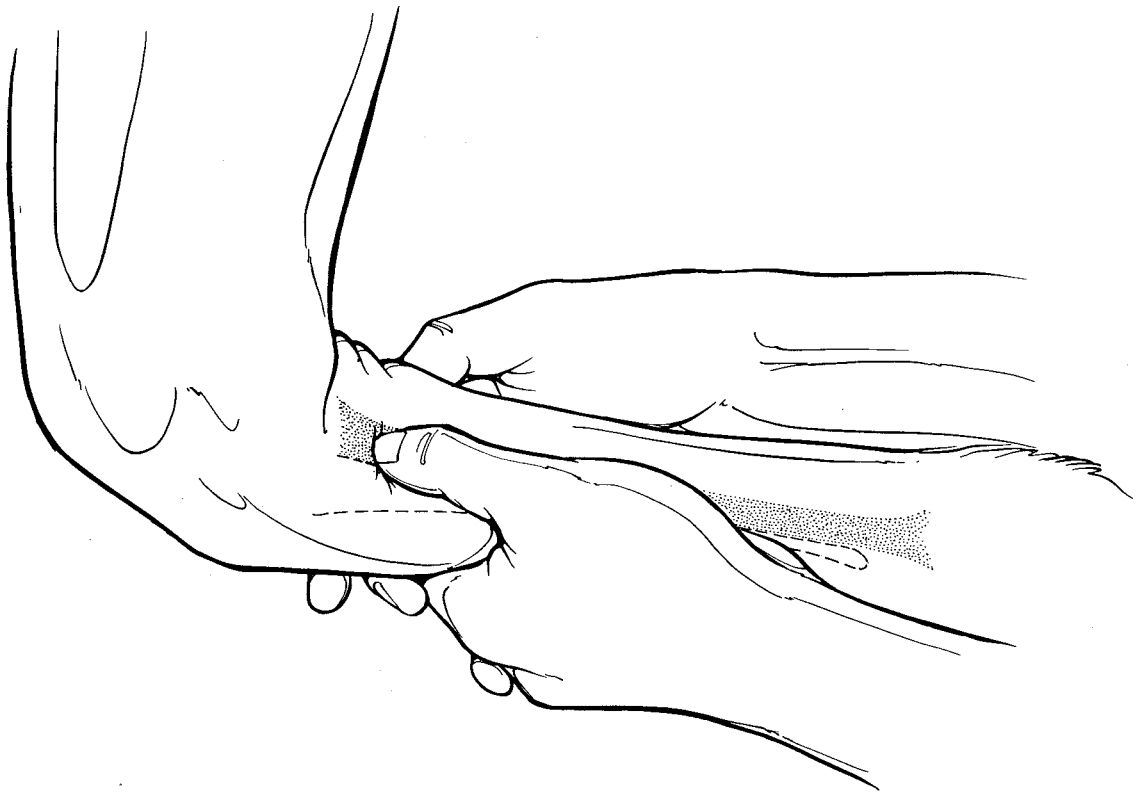

**Figure 3.21.** Palpation of the origin of the suspensory ligament in the proximal aspect of the metacarpal region. A repeatable painful response may suggest proximal suspensory desmitis.

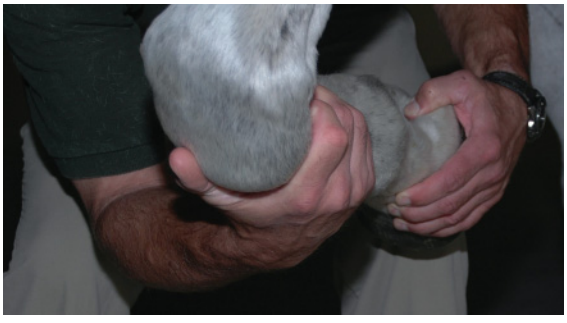

**Figure 3.22.** Method used to apply digital pressure to the suspensory ligament in the proximal palmar metacarpal region.

by deep digital pressure along the dorsal articular surfaces (Figure 3.28). With the carpus flexed, and the tension of the ulnaris lateralis and flexor carpi ulnaris reduced, the accessory carpal bone can be manipulated, potentially identifying a fracture. In some cases an osteochondroma on the caudodistal aspect of the radius may be palpable if effusion of the carpal canal is present.

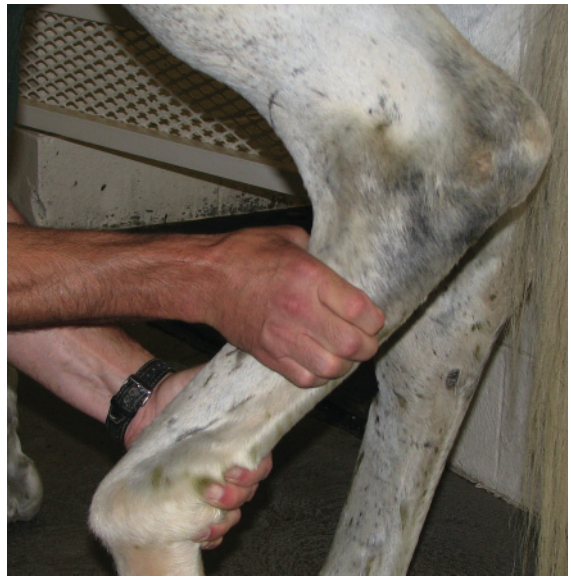

**Figure 3.23.** Method used to apply digital pressure to the suspensory ligament in the proximal plantar metatarsal region.

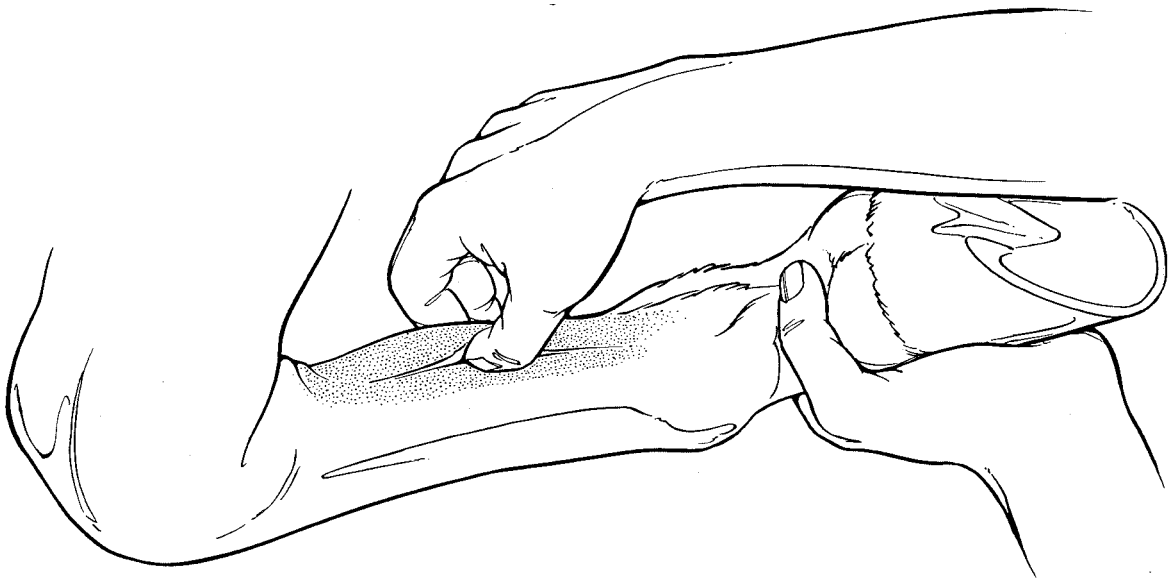

**Figure 3.24.** Palpation of the flexor tendons with the fetlock flexed to permit separation of the superficial and deep digital flexor tendons. Inability to separate the tendons usually suggests tendinitis.

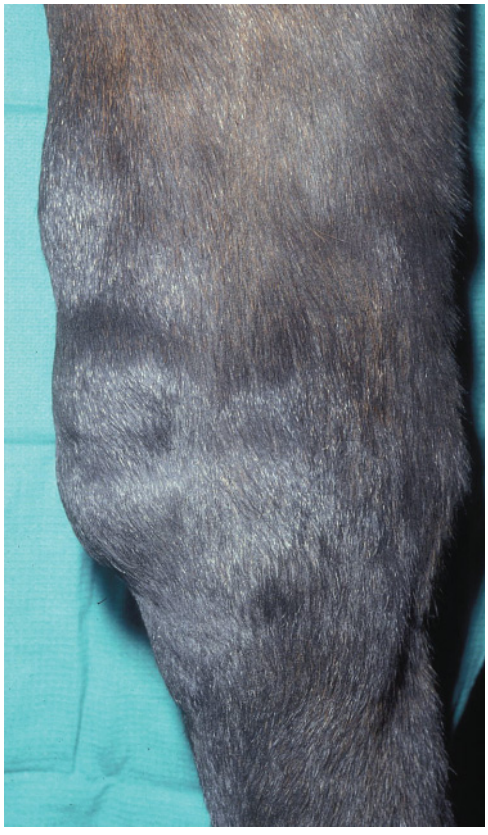

**Figure 3.25.** Effusion of the extensor carpi radialis tendon sheath is usually characterized by swelling that courses up and down the cranial aspect of the carpus.

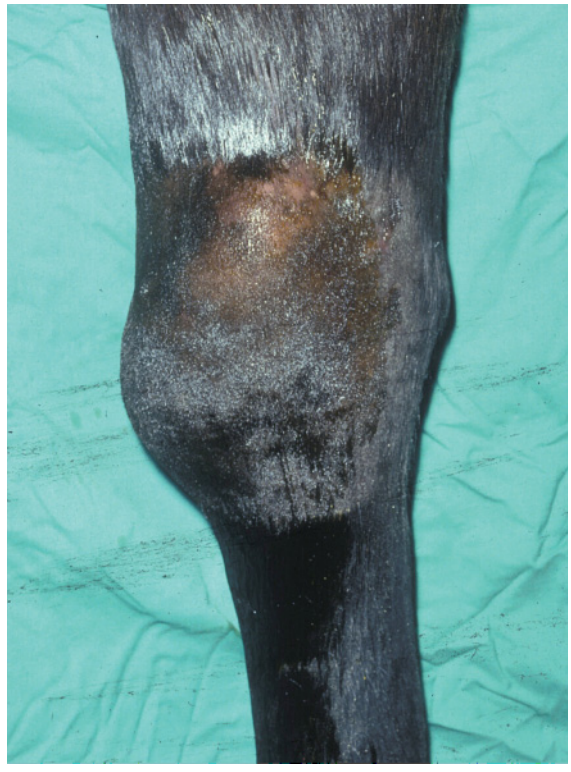

**Figure 3.26.** A hygroma of the carpus is a fluid-filled swelling located under the skin on the cranial aspect of the carpus. It is usually confined to the cranial aspect of the carpus and does not extend up the limb.

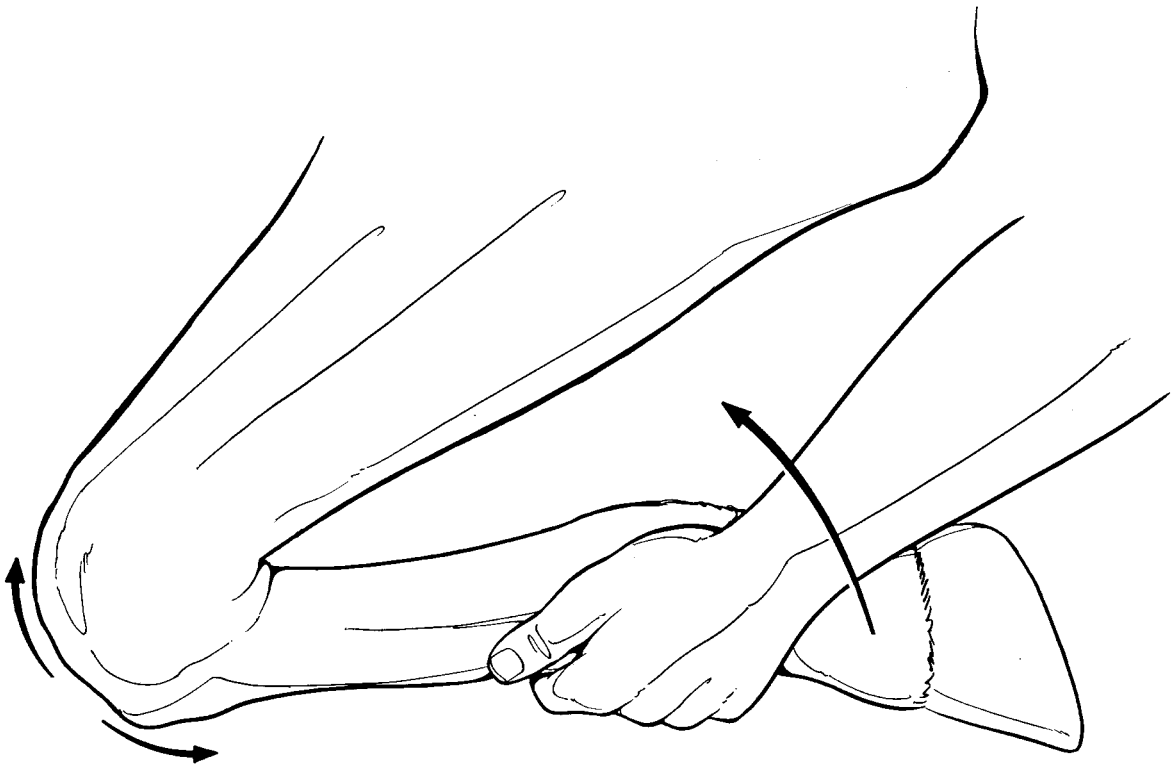

**Figure 3.27.** Flexion of the carpus to identify a painful response. In the normal horse, the flexor surface of the metacarpus approximates that of the forearm.

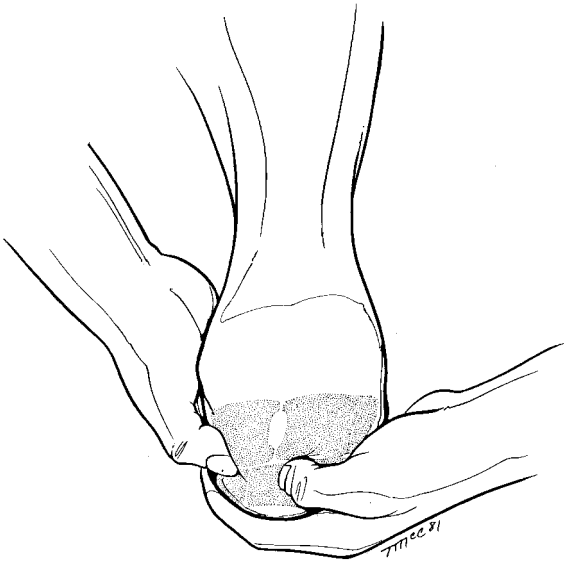

**Figure 3.28.** The dorsal articular margins of the carpal bones can be palpated after the carpus is flexed to identify pain within the individual carpal bones.

### *Forearm (Antebrachium) and Elbow*

The soft tissues of the forearm and elbow joint (cubital joint) should be palpated for signs of inflammation, particularly swelling. A firm swelling associated with the flexor muscles may be consistent with a myositis or a fibrotic or ossifying myopathy of these structures. The distal aspect of the radius should be palpated for swelling, heat, and pain. A firm, usually non-painful but fluctuant swelling at the point of the elbow is consistent with an elbow hygroma, also known as olecranon bursitis (Figure 3.29). Severe swelling and an inability to extend the limb (dropped elbow) are consistent with a fracture of the olecranon. In cases of non-displaced or chronic olecranon fractures, palpation of the caudal aspect of the olecranon may reveal variable degrees of swelling and pain with digital pressure. Elevation of the limb into extension also will often cause pain (Figure 3.30). The collateral ligaments of the elbow joint can be evaluated by abducting and adducting the elbow. This is not selective, however, because the carpal and shoulder joints are manipulated as well. Severe

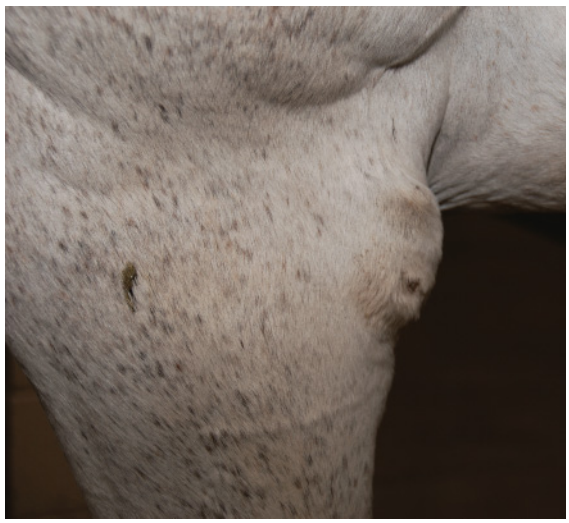

**Figure 3.29.** A small, fluid-filled swelling was present on the point of the elbow in this horse, consistent with olecranon bursitis. The skin was also thickened in the region but no lameness was present.

swelling, crepitation, and pain may be observed in horses with humeral fractures and can be confused with fractures of the olecranon.

### *Shoulder and Scapula*

The soft tissues around the shoulder joint (scapulohumeral joint) are observed and palpated for swelling or atrophy. Pressure applied directly over the joint may sometimes elicit a painful response (Figure 3.31). Particular attention must be paid to the bicipital bursa region (cranial aspect of shoulder), and deep digital palpation may suggest pain in the region. The muscle and tendon should be grasped with the fingers and thumb and pulled laterad. Another test to evaluate this region is to flex the shoulder joint by placing one hand on the olecranon process and pull the limb caudad. Young horses exhibiting obvious shoulder lameness at exercise and variable degrees of pain on manipulation often have osteochondrosis of the shoulder joint.

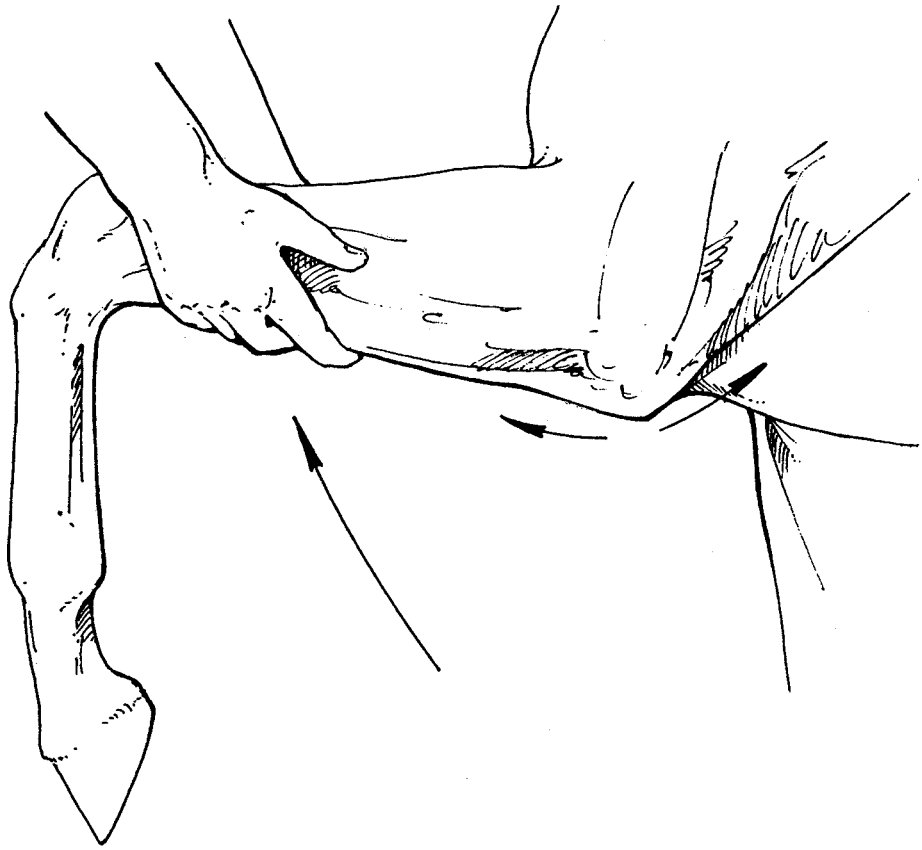

**Figure 3.30.** Elevating the limb into extension to flex the elbow joint extends the shoulder and increases the tension on the triceps brachii tendon at its insertion on the olecranon process.

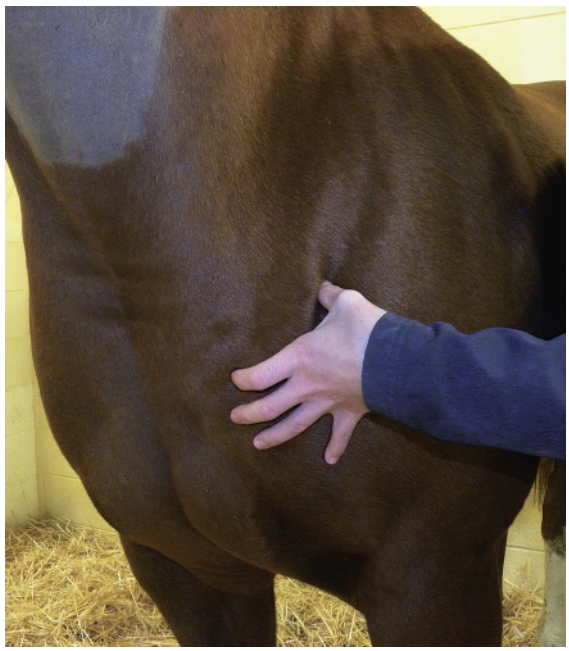

**Figure 3.31.** Thumb pressure applied just cranial to the infraspinatous tendon may elicit a painful response in horses with shoulder pain.

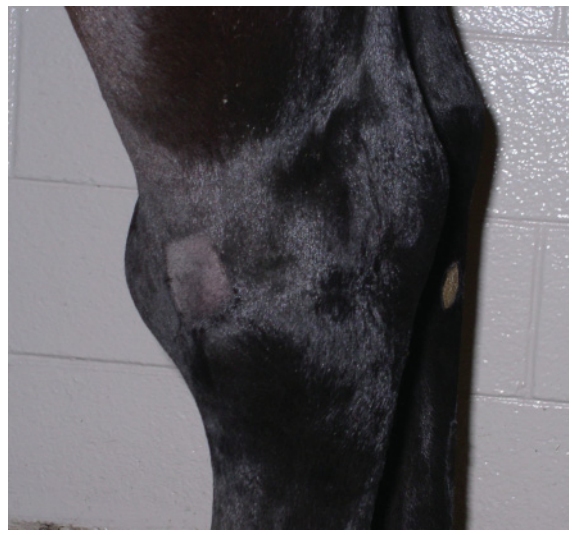

**Figure 3.32.** Young horse with effusion of the tarsocrural joint that is easily compressible and nonpainful.

Elevation of the limb as described for the elbow joint may also result in a painful response, particularly if a fracture or a lesion within the joint is present (Figure 3.30).

### *Tarsus (Hock)*

The tarsus should be examined for:

1. Tarsocrural joint effusion (synovitis, bog spavin, osteochondrosis)
2. Thickening of the fibrous joint capsule (chronic OA, previous trauma)
3. Bone proliferation of the distal tarsal joints (bone spavin)
4. Distension of the tarsal sheath (thoroughpin)
5. Inflammation of the long plantar ligament or SDFT (curb)
6. Luxation of the SDFT over the calcaneus
7. Capped hock
8. Subtendinous bursal effusion (calcaneal bursitis)

In general, there are three different types of soft tissue swelling that may be seen within the tarsus. The first is a fluctuant fluid distension of the tarsocrural joint, often referred to as "bog spavin." The synovial effusion can be easily compressed from the dorsal medial pouch to distend the plantar pouches of the joint capsule and vice versa (Figure 3.32). The second type of swelling is a firm distension of the tarsocrural joint capsule, and the synovial fluid is difficult to compress from one pouch to the other. This "firmness" is due to synovial inflammation of the fibrous layer of the joint capsule (capsulitis) and often suggests a chronic problem such as OA or trauma to the fibrous joint capsule. The third type of swelling is a firm, diffuse swelling of the entire tarsal joint region (Figure 3.33). It is usually due to a severe sprain to the fibrous joint capsule and surrounding ligamentous support structures associated with trauma.

The medial aspect of the distal tarsal joint region (distal DIT and TMT joints) should be closely examined visually and with palpation (Figure 3.34). In the normal horse there is a smooth contour that tapers to the distal tarsal bones as they join the proximal metatarsus. This is easily visualized from the rear and palpated from the side. If this region appears boxy with obvious enlargement, OA of the DIT and TMT joints should be suspected. These medial enlargements also have been referred to as "tarsal shelves" (Figure 3.35). Applying pressure over the medial aspect of the distal tarsus has been

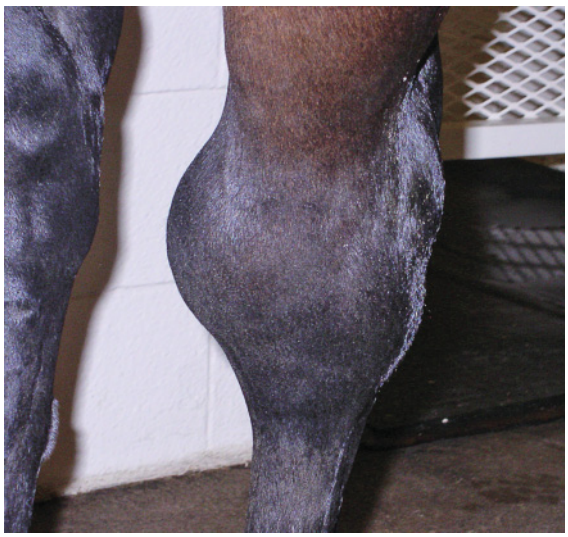

**Figure 3.33.** This young horse had a history of previous trauma to the tarsus. Part of the swelling was firm and painful to palpation, but there was also effusion within the tarsocrural joint.

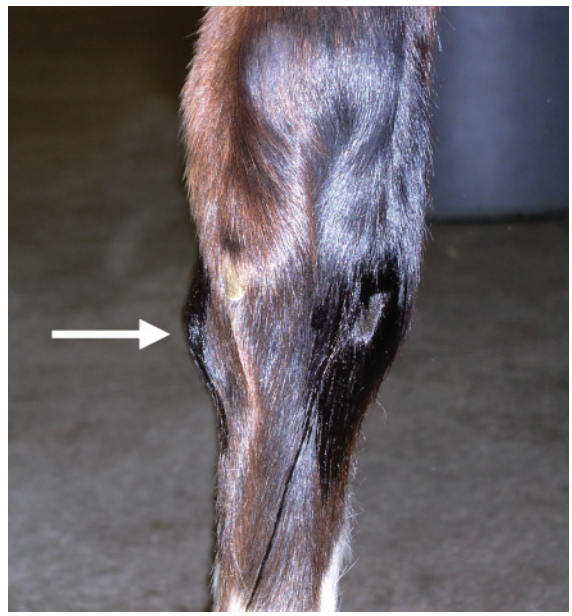

**Figure 3.35.** Enlargement of the medial aspect of the distal tarsus (arrow) consistent with distal tarsal OA (bone spavin).

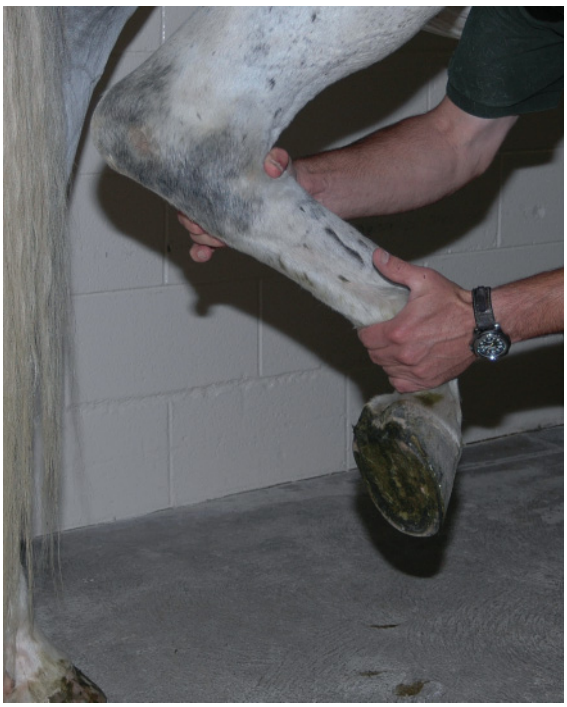

**Figure 3.34.** Limb and hand positioning to perform the "Churchill" test to detect pain on the medial aspect of the distal tarsal joints.

referred to as the Churchill pressure test. Using the index and middle fingers, firm pressure is applied to the plantar aspect of the head of the second metatarsal (splint) bone (Figure 3.34). The test is considered positive if the horse flexes and abducts the limb away from the pressure. A positive Churchill test may indicate distal tarsal OA or cunean bursitis, especially if there is a marked difference in the response between the two tarsi.

Effusion/swelling of the tarsal sheath often is referred to as "thoroughpin," and usually can be observed and palpated on the medial aspect of the tarsus. Unlike effusion of the tarsocrural joint, the effusion is asymmetrical on the limb (medially) and runs in a distal to proximal direction (Figure 3.36). Tarsocrural joint effusion is symmetrical and courses circumferentially around the tarsus. Tarsal sheath effusion may be a cosmetic blemish but can be indicative of problems of the DDFT or the sustentaculum tali of the talus (Figure 3.36). The plantar aspect of the tuber calcis should be palpated for inflammation of the plantar ligament (curb), tendinitis of the SDFT (Figure 3.37), lateral displacement of the SDFT (Figure 3.38), and a fluid swelling at its proximal limits referred to as "capped" hock. Swelling associated with a capped hock is

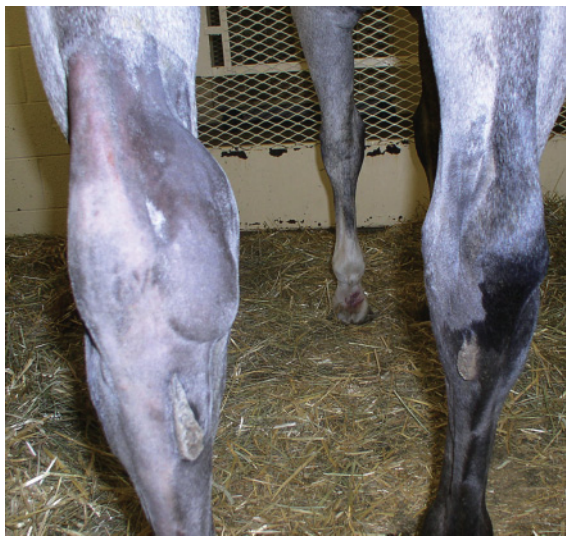

**Figure 3.36.** Effusion of the tarsal sheath on the medial aspect of the tarsus that was associated with fragmentation of the sustentaculum tali.

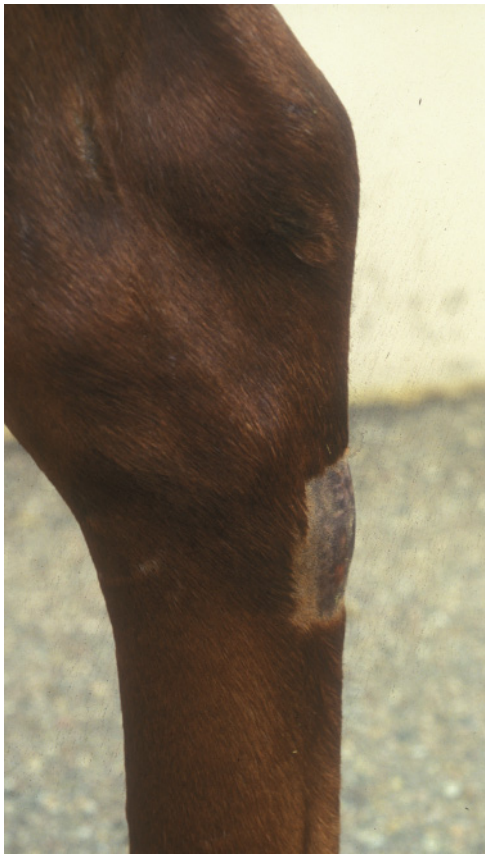

**Figure 3.37.** Tendinitis of the superficial digital flexor tendon in the proximal metatarsal region, which can be misdiagnosed as a curb in some horses.

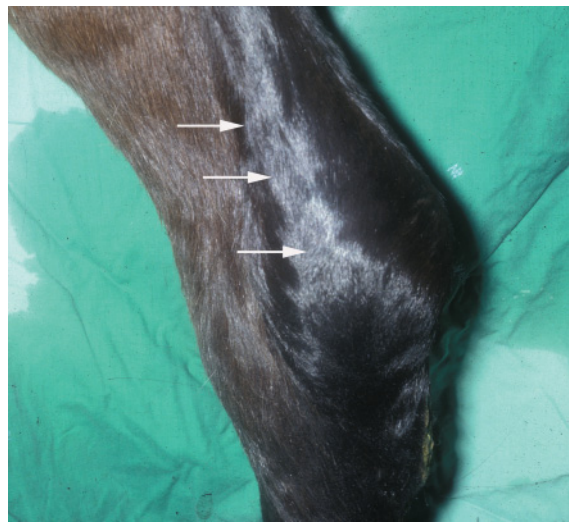

**Figure 3.38.** Lateral displacement of the superficial digital flexor tendon from the point of the calcaneus (arrows). Usually effusion of the calcaneal bursa is also present in these horses.

subcutaneous, whereas effusion within the calcaneal bursa is beneath the SDFT. Effusion of the calcaneal bursa often is characterized by small pockets of fluid on each side of the tendon above and below the point of the hock (Figure 3.39).

### *Tibia*

It can be difficult to detect abnormalities in the tibial region, both visually and with palpation. Swelling in the caudal tibial region may indicate myositis of the semimembranosus and semitendinosus muscles or gastrocnemius tendinitis. Focal swelling of the distal medial epicondyle of the tibia could be associated with a fracture or sprain to the medial collateral ligament of the tarsus. Severe pain with deep digital palpation of the distal third of the tibia, together with a severe lameness and a positive spavin test, may suggest the possibility of an incomplete tibial fracture. A complete fracture of the tibia is associated with non-weight-bearing lameness, severe swelling, limb deviation, and crepitus on palpation and manipulation.

The semimembranosus and semitendinosus muscles should be palpated for any evidence of pain and swelling indicative of myositis (hamstring pull) and for firm scarring/fibrosis that is often present with fibrotic myopathy (Figure 3.40). Although it is an uncommon site for problems, the gastrocnemius tendon should be pal-

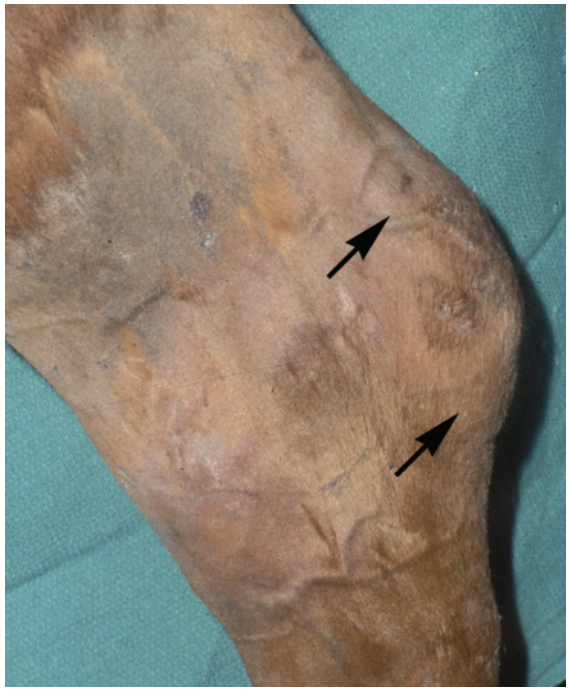

**Figure 3.39.** Effusion within the calcaneal bursa often can be palpated as fluid outpouchings above and below the retinaculum of the SDFT (black arrows).

pated for swelling and pain. Additionally, an attempt should be made to extend the hock joint if clinical signs consistent with rupture of the peroneus tertius muscle are present during exercise. With the stifle flexed, the hock can be extended and a characteristic dimpling of the gastrocnemius tendon occurs (Figure 3.41).

### *Stifle*

The stifle should be observed and palpated for swelling and/or atrophy of the associated muscle groups and for fluid distention of the joints. Distension of the femoropatellar joint is best seen from the lateral view (Figure 3.42), and distention of the medial femorotibial (MFT) is best observed from the cranial aspect (Figure 3.43). However, palpation is the preferred method to detect effusion within the stifle joints.

The femoropatellar joint pouch is located on the cranial aspect of the stifle beneath the patella ligaments. In general, effusion of the femoropatellar joint will make palpation of the three distal patellar ligaments more difficult. These ligaments should be easily palpable across the dorsal aspect of the stifle and are the landmarks to

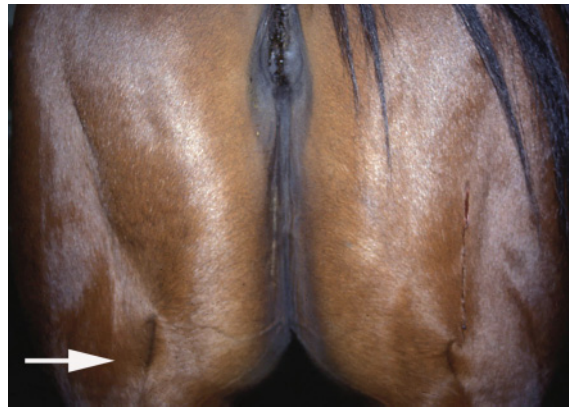

**Figure 3.40.** Horse with fibrotic myopathy of the left hindlimb. There is atrophy of the semitendinosus muscle and firm scar tissue palpable in the caudal tibial region (arrow).

locate the three synovial pouches of the stifle. The patella ligaments should be palpated for evidence of desmitis, and the medial patellar ligament should be evaluated for scarring that may suggest previous surgery for upward fixation of the patella. Fluid distension of the femoropatellar pouch may indicate pathology within the femoropatellar joint, or within the MFT joint as they communicate with each other. The MFT joint pouch is located medial to the medial patella ligament directly above the tibial plateau. The lateral femorotibial joint is located lateral to the lateral patella ligament and effusion within this joint is rarely detected. Palpation of the stifle joints for effusion should always be compared to the opposite limb. In general, abnormalities within the stifle joint are usually accompanied with synovial effusion. Effusion of the femoropatellar and MFT joints can be associated with a variety of IA abnormalities.

The patella should be palpated for parapatellar inflammation and pain, crepitus, and displacement. The patella displacement test can be performed by grasping the base of the patella between the thumb and forefinger and pushing proximally (upward) and laterally (outward) in an attempt to engage the medial patellar ligament over the medial trochlea. Most horses object to this manipulation and will attempt to flex the stifle to prevent the forced upward displacement of the patella. With complete upward fixation the horse will be unable to flex its stifle or hock and may drag its limb behind in extension (Figure 3.44).

Manipulative tests also may be performed on the stifle to assess the cruciate ligaments and

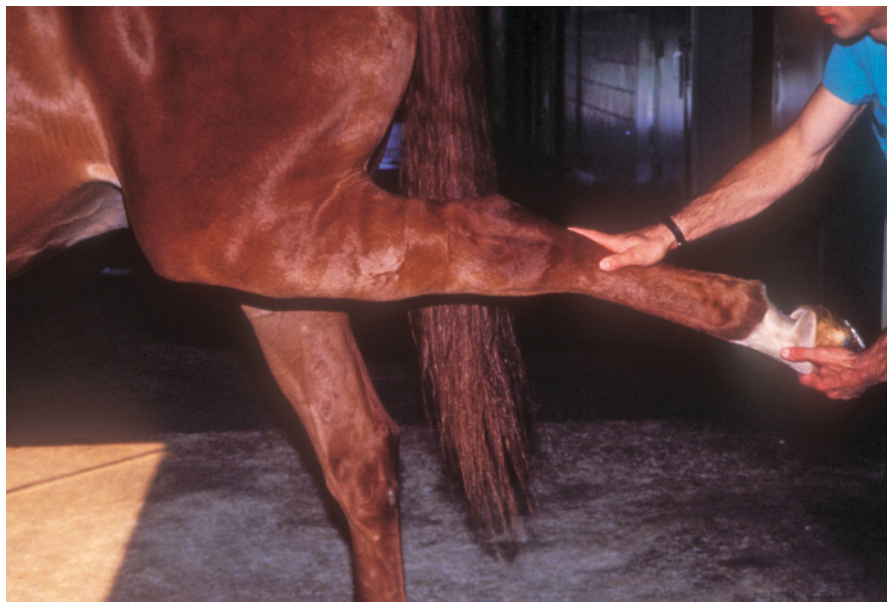

**Figure 3.41.** Horse with rupture of the peroneus tertius as suggested by the ability to simultaneously extend the hock and flex the stifle.

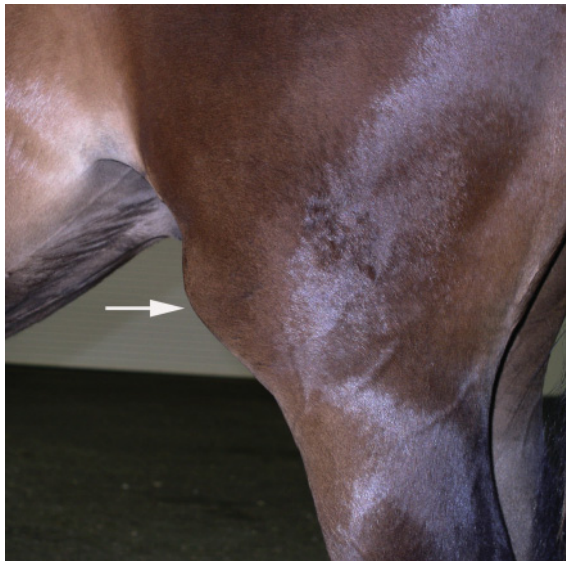

**Figure 3.42.** Effusion can be seen and palpated within the femoropatellar joint cranial to the patella ligaments (arrow).

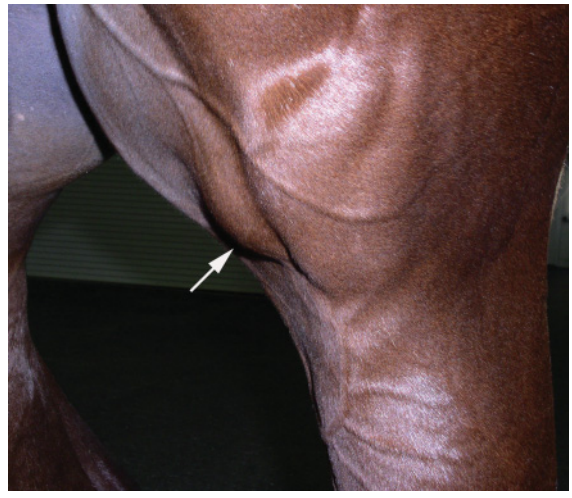

**Figure 3.43.** Visible and palpable effusion within the medial femorotibial joint is present just behind the medial patella ligament (arrow).

medial collateral ligament. These tests are very subjective and only used in those horses in which other clinical signs suggest injury to these structures. The cruciate test can be performed from either the caudal or cranial aspect of the limb.

With the caudal approach, the examiner stands behind the horse with their arms brought around the limb and the hands clasped together at the proximal end of the tibia (Figure 3.45). The examiner's knees or knee should be in close contact with the plantar aspect of the calcaneus, and the examiner's toe placed between the bulbs

**Figure 3.44.** The horse is experiencing upward fixation of the patella. The limb is locked in extension and extended caudally and a bit laterally. The digit also is fixed in the flexed position. Courtesy of Ken Sullins.

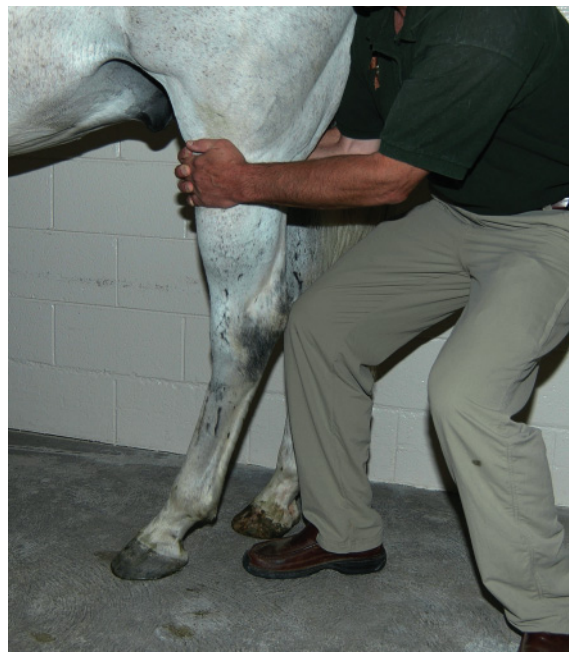

**Figure 3.45.** The caudal cruciate test is performed by grasping the tibia from behind and quickly pulling the tibia caudally. Increased movement of the tibia or a painful response may suggest damage to one of the cruciate ligaments.

of the heels. In this position the examiner pulls the tibia sharply caudally and releases it cranially, feeling for looseness and crepitation. A generalized looseness within the stifle often is the only definitive finding because it is difficult to identify the phase (caudal or cranial) in which the movement occurs.

With the cranial approach, the examiner stands in front of the affected limb with one hand placed on the proximal tibial tuberosity. The other hand is used to pull the tail to that side to force the horse into weight bearing. The tibia is pushed caudally as quickly and forcibly as possible, which is thought to stress the cranial and caudal cruciate ligaments (Figure 3.46). This may be repeated multiple times, after which the horse may be trotted off and the degree of lameness observed.

The medial collateral ligament test is performed by placing the shoulder or outside hand over the lateral aspect of the stifle and abducting the distal limb with the other hand (Figure 3.47). Increased lateral movement of the distal limb indicates complete rupture of the medial collateral ligament. The opposite approach may be performed to test the integrity of the lateral collateral ligament, but injuries to this ligament are uncommon. If a sprain to the ligament is suspected, the limb may be abducted five to 10 times, after which the horse is trotted off and the degree of lameness assessed.

### *Femur*

The muscles surrounding the femur are primarily examined for swelling and/or atrophy. The femoral artery should be palpated for the quality of pulsations on the medial side of the thigh in the groove between the sartorius muscle cranially and the pectineus muscle caudally. If the pulse is weak or nonexistent, thrombosis of the iliac artery may be contributing to the lameness. Pressure can be applied to the greater trochanter, and if painful, middle gluteal muscle strain or trochanteric bursitis (whirlbone disease)

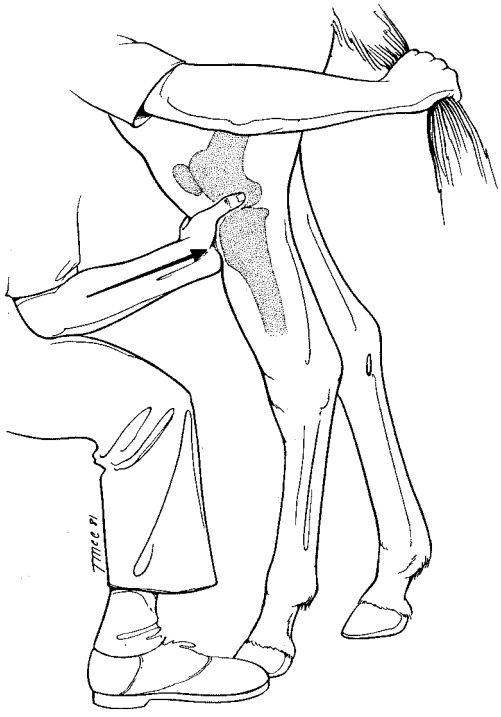

**Figure 3.46.** Positioning to check for problems with the cranial cruciate ligament. The examiner places one hand on the proximal tibia and forces it caudally (arrow) to check for increased movement (cranial drawer) of the tibia in relation to the femur.

should be suspected. Complete fractures of the femur usually result in non-weight-bearing lameness with severe swelling and limb shortening due to overriding of the fracture. Femoral neck fractures are more difficult to diagnose because they typically cause less swelling and lameness than diaphyseal femoral fractures. With time the swelling may migrate distally on the medial side of the thigh, giving the impression that the distal femoral region is involved.

### *Hip*

The hip should be examined for asymmetry, swelling, and atrophy of associated muscle groups. With hip problems, swelling over the coxofemoral joint may be visually apparent and pain often can be elicited with deep palpation directly over the joint using the palm of the hand. Frequently a stifle-out, hock-in, toe-out gait is observed at a walk, with an apparent shortening of the limb length (Figure 3.48). From the side, the affected limb may appear to be straighter than the contralateral limb. With the metatarsus held in hand, the coxofemoral joint can be manipulated into extension, flexion, and abduction to check for evidence of pain and crepitation. Additionally, the hip can be intermittently flexed and auscultated with a stethoscope at the same time to identify crepitus. Limb abduction often is painful to horses with hip conditions, and repeated limb abduction will often exacerbate the lameness.

### *Pelvis*

Visual identification of asymmetry of the bones and musculature of the pelvis is an important aspect of examination of the pelvis. This includes the tuber coxae, the tuber ischium, the tuber sacrale, and the gluteal muscles on each side. Asymmetry of the bony pelvis often suggests a pelvic fracture, subluxation of the sacroiliac region, or fracture of the specific bony prominence. Gluteal muscle atrophy often accompanies chronic pelvic fractures but can be seen with any chronic hindlimb lameness (Figure 3.49). Crepitus associated with pelvic fractures usually can be elicited by swaying the horse from side to side, or sometimes can be picked up by rectal examination.

### *Back*

Visual assessment of the horse's back includes observing the muscle contour from the side and axial alignment from the rear. The dorsal spinous processes should be palpated for axial alignment, protrusion or depression, and interspinous distance (Figure 3.50). Any muscle swelling, atrophy, or asymmetry in the epaxial musculature should be noted.

Palpation is usually best performed with firm fingertip pressure using both hands simultaneously (Figure 3.51). Alternatively, the palm of the hand can be used to apply downward pressure to the epaxial muscles. Palpation of the epaxial muscles lateral to the dorsal spinous processes along the entire length of the back should be performed. Many horses may respond to downward pressure in the lumbar region by ventroflexing their backs, but this response often causes fatigue, and withdrawal is less prominent. In horses that have clinically significant back pain, ventroflexion of the back often is severe and any increase in finger- or palm-applied pressure greatly increases this response. The

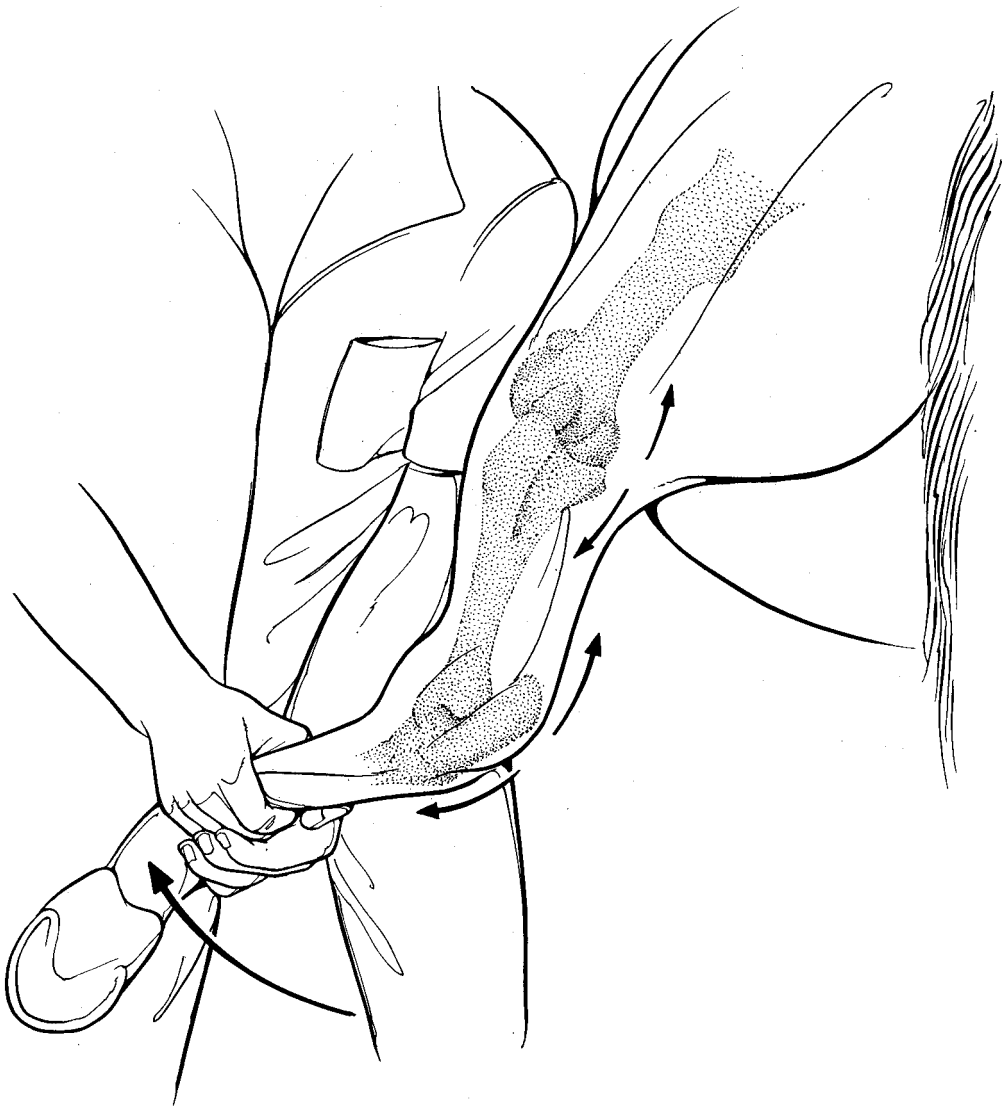

**Figure 3.47.** Test to stress the medial collateral ligaments of the hock and stifle. Alternatively, one hand can be placed on the medial aspect of the distal tibia to selectively stress the medial collateral ligament of the femorotibial joint. The examiner's shoulder can be placed over the middle of the tibia and both hands on the distal metatarsus to selectively stress the medial aspect of the hock.

horse attempts to "drop down" to get away from hand pressure. Palpation also may cause the horse to vocalize, swish its tail, or actually kick out behind. Back palpation is somewhat subjective; therefore, the assessment requires clinical experience. In some cases, tightening of the longissimus dorsi muscle may be felt with palpation rather than a painful withdrawal response. This usually signifies that the horse is attempting to fix the vertebral column because ventroflexion is painful.

The examiner also may wish to assess the horse's willingness to ventroflex, dorsiflex, and lateroflex its thoracic and lumbar vertebrae. Assessment of the horse's ability to ventroflex the back is obtained by exerting downward pressure to the muscles in the thoracolumbar region. A blunted instrument (needle cap) run over the

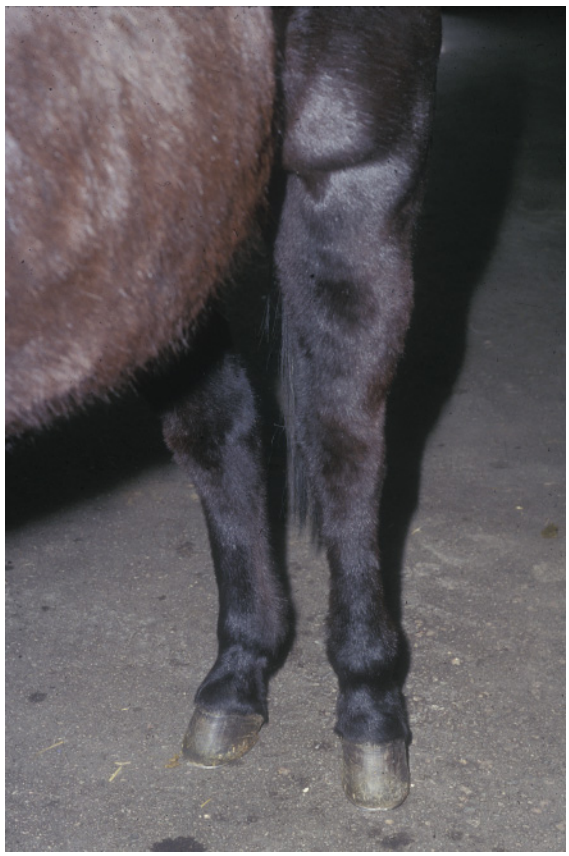

**Figure 3.48.** Typical toe-out, hock-in stance that often accompanies problems within the hip and pelvic region.

croup usually causes dorsiflexion or arching of the back. Lateral flexion usually can be assessed by firmly stroking either side of the thoracolumbar region with a blunted instrument. Reluctance to flex the back in any of these directions may suggest muscle tightening and back rigidity within the thoracolumbar region.

### *Neck*

The neck should be examined for contour from the side and axial alignment from the front and rear. Excessive ventral arching of the neck in the mid-cervical region may be seen in some cases of cervical vertebral malformation. A straight (extended) poll can be seen with atlanto-occipital and atlanto-axial malformations. Axial deviations of the neck are most commonly due to developmental problems (e.g., hemivertebrae) or trauma. Splinting and spastic contraction of the neck muscles with or without signs of spinal

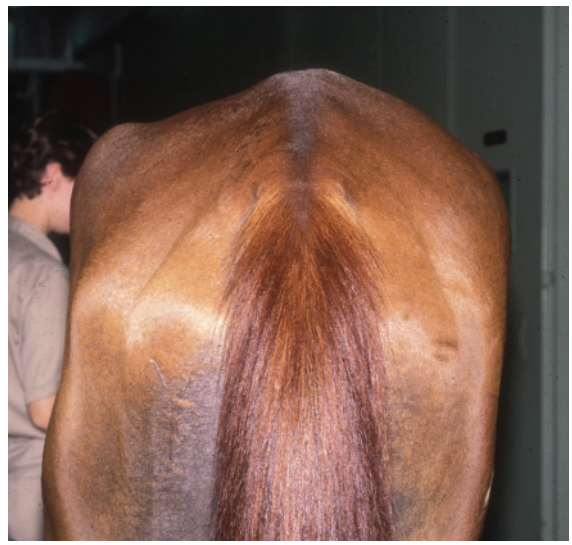

**Figure 3.49.** Severe atrophy of the left gluteal muscles secondary to a pelvic fracture. Disuse gluteal atrophy tends to occur more quickly and more profoundly with pelvic fractures than other lameness problems more distal in the limb.

ataxia is often consistent with vertebral fracture. Generally these horses are very painful.

Palpation should be done to identify muscle atrophy or swelling and document the alignment of the vertebrae. The transverse processes should be palpated for alignment and symmetry. Muscle atrophy is most often observed in the caudal neck region dorsal to the cervical vertebrae and may be symmetric or asymmetric. Potential causes for muscle atrophy include cervical vertebral malformation, articular facet joint OA, and neurologic problems such as equine protozoal myelopathy. Swelling of the neck, either lateral or ventral, generally is a sign of trauma and/or infection.

The neck should be flexed laterally and ventrally and extended to assess flexibility, range of motion, and pain. Lateral flexing can be initiated by pulling the horse's head to one side and then the other. Alternatively, lateral neck flexion can be encouraged by holding a treat at the horse's shoulder. Most horses should be able to flex their neck laterally enough that the muzzle almost contacts the craniolateral shoulder region. Ventroflexion is assessed by feeding the horse from the ground level, and extension is evaluated by elevating the head and neck. Resistance to neck movement in any direction usually is due to pain, and can be from many potential causes.

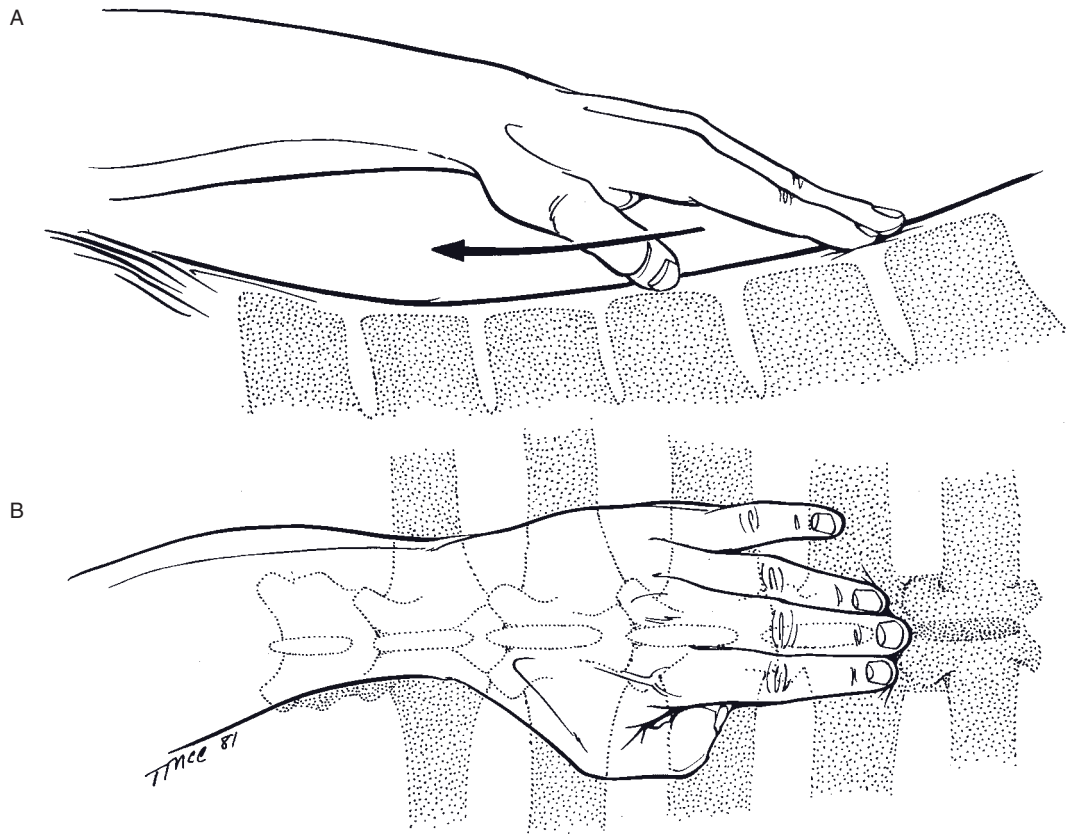


**Figure 3.50.** (A) Palpation of the summits of the dorsal spinous processes to identify depressions or protrusions that may indicate subluxation or fracture. (B) Palpation of the axial alignment of the dorsal spinous processes.

## VISUAL EXAM AT EXERCISE

Visual examination of the horse at exercise focuses on the gait characteristics of all limbs and is usually performed from a distance. In most cases, it is best to observe the forelimbs first, and then the hindlimbs. In most cases, the horse is observed at exercise without removing the shoes, although the shoes can be removed if necessary.

The main objective of exercising the horse is to identify the limb or limbs involved and the degree of lameness and/or incoordination in movement. The horse should be observed at a walk and trot in a straight line, then while lunging in circles. Foot placement usually is best observed from the side with the horse at a walk, and from the front or rear with the horse walked toward or away from the observer, respectively. In general, forelimb lameness is best viewed from the front and side, and hindlimb lameness is best observed from the side and rear. The examiner is looking for abnormal head movement; gait asymmetry; alterations in height of the foot flight arc; alterations in foot flight; length of stride; joint flexion angle; foot placement; degree of fetlock extension with weight-bearing; action of the shoulder musculature; symmetry in gluteal rise and use; and movement of the pelvis, hemipelvis, and croup.

The two most important concepts in lameness detection in the horse are the head nod for forelimb lameness and the pelvic hike or rise for hindlimb lameness. Kinematic gait analysis indicates that maximal vertical acceleration of the head and displacement amplitude of the tuber sacrale are the best indicators to quantify a forelimb and hindlimb lameness, respectively. In both scenarios the horse tries to unweight the lame limb by elevating the head and neck for a forelimb problem and by elevating the pelvis or hemi-pelvis and croup for a hindlimb problem. However, objective analysis of lame horses suggests that elevation of the head and neck and

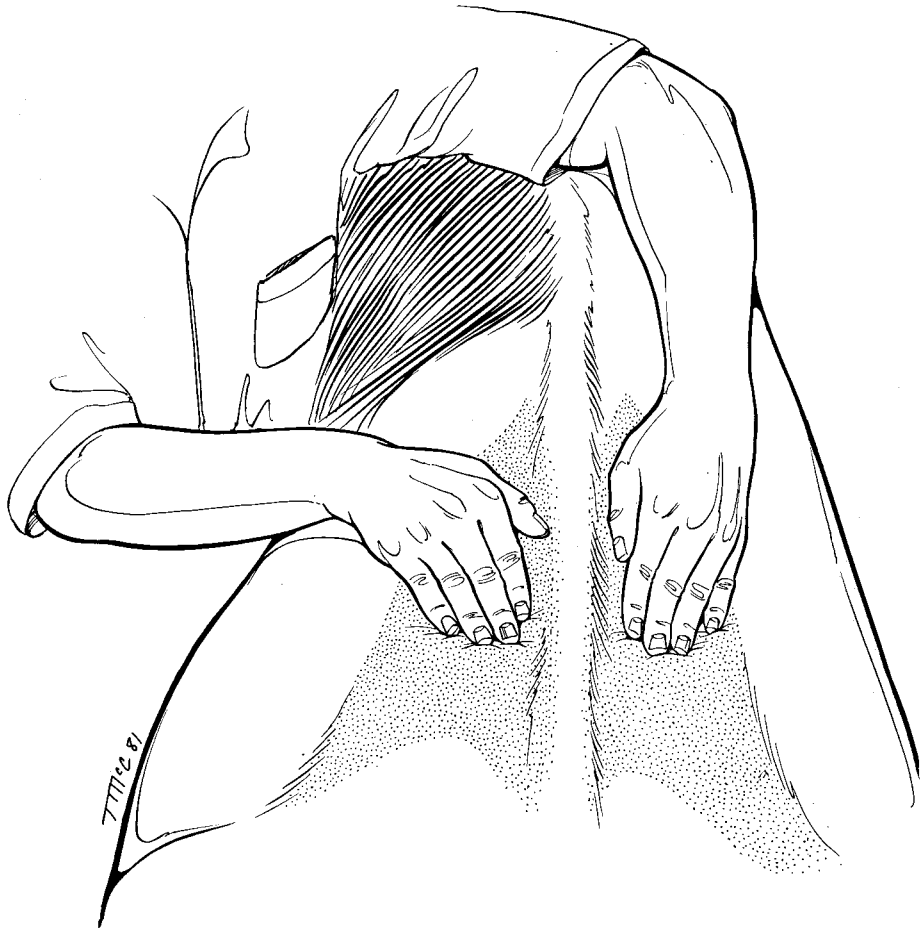

**Figure 3.51.** Firm pressure applied to the back musculature from the withers to the tuber sacrale to identify a painful response. The fingers should be held flat to prevent "digging in" with the fingertips.

pelvic hike may only occur in horses with substantial forelimb and hindlimb lameness.

Abnormal movement of the head and pelvis and other gait abnormalities are best viewed at a distance from the front, side, or rear of the horse. An overall impression of how the horse moves should be obtained initially, paying close attention to movement of the head and neck and pelvis and croup. The action of all four limbs should then be observed followed by the limb in question. It is important to observe the opposite limb for a comparison. Observation of subtle gait changes may require visual shifting from one limb to the other and back again. In some cases, it is helpful to observe the horse while being ridden or driven, or at speed on a treadmill.

## FLEXION TESTS/MANIPULATION

Most flexion tests, regardless of the location, are usually performed for 30 to 60 seconds and are a subjective method to further isolate the site of the lameness. However, the response to flexion tests must be interpreted in light of clinical findings because many otherwise normal horses may demonstrate positive responses. These positive responses were thought to be directly related to the force applied to the limb as 20 of 50 horses responded to a "normal" distal limb flexion and 49 of 50 horses responded to a "firm" distal limb flexion in one study. Another study revealed that more than 60% of 100 sound horses had a positive response to distal limb flexion and that the

positive outcome increased significantly with age. Both of these studies question the validity of distal limb flexion tests to predict future joint-related problems. In addition, false-positive responses to flexion seem to occur more commonly in horses in active work than those that have been rested or turned out to pasture. In general, there are more false-positive results to flexion at any location than false-negative results, but both can occur. False-positive responses are most common in the front fetlock.

Because both the amount of force and the duration it is applied affect the response to flexion, the procedure should be standardized as much as possible to minimize variability. For instance, the same person should flex the right and left limbs at any location for the same period of time so they can be accurately compared. Different people may have slight differences in the way they hold the limb or apply pressure to the limb, which can alter the responses. Despite this potential for variability, the flexion techniques performed by experienced veterinarians usually are sufficient to objectively assess responses to flexion.

Passive flexion usually refers to manipulation of a joint during routine palpation of the horse, and pain detected with passive flexion often predicts a significant response to a 30- to 60-second flexion test. However, flexion tests also can be used to subjectively assess the severity of damage within an affected joint(s). In general, the severity of damage often is proportional to the severity of the response to the flexion test. For instance, horses with severe responses to carpal, fetlock, or stifle flexion typically have significant IA or extra-articular pathology. However, flexion tests are not specific for the joint because it is nearly impossible to flex a single joint without affecting other nearby joints and soft tissues. Flexion of a joint not only increases the IA and subchondral bone intra-osseous pressures within the joint but also compresses and stretches the joint capsule and surrounding soft tissues. The numerous other "structures" that are being manipulated with any flexion test should always be considered when interpreting the clinical significance of flexion tests.

The responses to flexion should be graded in some manner and included in the records. This is most important when re-evaluating the lameness to more accurately determine if improvement is being made. The author uses a grading scale of negative, mild response, moderate response, and severe response to assess the flexion tests. Alternatively, a plus-minus system may be used with "–" being no response, 1+ equating to mild, 2+ to moderate, and 3+ to severe. Regardless of the system used, the responses to flexion are a very important aspect of the lameness examination, and should be recorded. Additionally, changes in lameness in the limb not being flexed (weight-bearing limb) also should be recorded because this is often an important clinical finding. This contralateral response to flexion tends to occur most commonly in horses with bilateral hock and carpal problems.

### *Distal Limb/Phalangeal/Fetlock Flexion*

Attempts can be made to isolate the fetlock joint from the pastern and coffin joints during flexion of the distal limb. However, it is nearly impossible to only flex the fetlock or only flex the pastern or coffin joints. Flexion of the fetlock joint can be performed by placing one hand on the dorsal MC and pulling up on the pastern with the opposite hand (Figure 3.17). Flexion of just the phalangeal joints is performed by maintaining fetlock extension by placing one hand on the pastern while flexing the phalanges by pulling up on the toe with the opposite hand (Figure 3.52). All three joints can be flexed together (distal limb flexion) by pulling up on the toe and

**Figure 3.52.** Hand and limb positioning to perform flexion of the phalangeal joints without flexing the fetlock.

**Figure 3.53.** Hand and limb positioning to perform distal limb flexion (phalangeal and fetlock joints) of the hindlimb.

phalanges with both hands while facing toward the back of the horse (Figure 3.18). In general, it is much more difficult to isolate the phalanges from the fetlock region in the hindlimb; therefore, most distal limb flexion tests performed in the hindlimb include all three joints (distal limb flexion test; Figure 3.53).

Regardless of how the flexion test is performed, the fetlock/phalanges are held in position for 30 seconds, after which the horse is trotted off and lameness is observed. Differences in the severity of the responses may be used to suggest whether there may be a problem in the fetlock vs. the pastern or coffin joints. A painful response to phalangeal flexion and a negative response to fetlock flexion may suggest a problem in the coffin or pastern joint or any soft tissue structure in the area. A negative response to phalangeal flexion together with positive responses to both the fetlock and distal limb flexion would suggest a fetlock problem. Any positive signs should be checked with the opposite limb; a marked asymmetry in responses to distal limb flexion tests is an important clinical finding, and further indicates a potential problem in the area. However, false positive fetlock flexion tests do occur, especially in horses in work, and many horses may show a positive response if a large amount of force is applied to the fetlock/distal limb. One study in normal horses evaluating the force applied for the fetlock and phalangeal flexion test by different examiners found that the force varied considerably and was frequently too high. Methods to standardize the fetlock flexion test have been recommended (calibrated measuring device) but are not being used clinically by most clinicians.

### *Carpal Flexion*

The carpal flexion test is very useful to help isolate a problem to the carpus. A negative response does not rule out a problem in the carpus (many horses with osteochondral fragmentation are not positive to carpal flexion), but a positive response is suggestive of a carpal problem (there are few false-positive responses). This is in contrast to the fetlock, where false-positive responses are much more common. Carpal flexion is performed by grasping the metacarpus with the outside hand while facing the horse, and pulling up on the distal limb (Figure 3.27). The foot should be able to contact the caudal aspect of the olecranon in normal horses. The carpus is held in this position for 60 seconds, after which the horse is jogged away and observed for increased lameness.

### *Elbow Flexion*

It is difficult to completely separate the elbow from the shoulder when performing upper limb flexion tests in the forelimb. This is analogous to the tarsus and stifle in the hindlimb, as flexion of one area often affects the other. However, flexion of the elbow can be performed by lifting the antebrachium (forearm) so that it is parallel to the ground and not pulled forward (Figure 3.30). This flexes the elbow and causes the carpus and distal limb to "hang" freely. The limb is held in this position for 60 seconds and the horse is jogged off. Elbow flexion is not part of a routine lameness evaluation; usually it is performed when an abnormality in the elbow region was found on physical examination.

### *Shoulder/Upper Forelimb Flexion*

Manipulation of the upper forelimb can be performed either by pulling the limb cranially and upward or by pulling the limb caudally. The cranial approach is similar to flexing the elbow, except the limb is pulled forward. This will flex the elbow and extend the shoulder. This is performed by standing in front of the limb, grasping the antebrachium, and lifting the limb up and forward (Figure 3.54). This will exacerbate

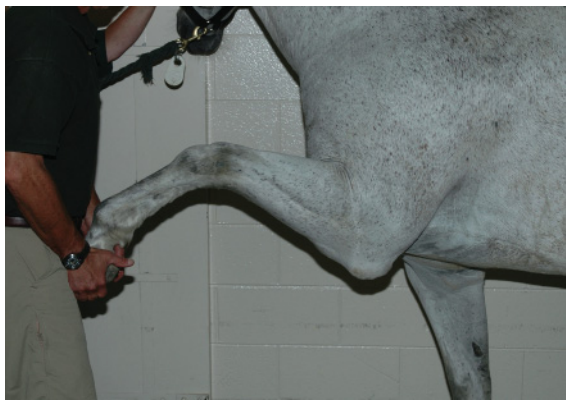

**Figure 3.54.** Upper limb flexion test in which the limb is pulled cranially and upward to "stress" the shoulder region.

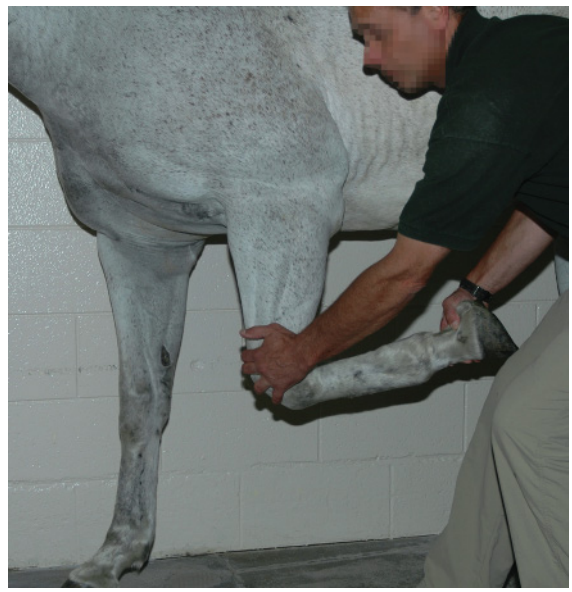

**Figure 3.55.** Flexion of the upper forelimb can be performed by grasping the antebrachium and foot and pulling the limb caudally.

lameness problems in the caudal aspect of the elbow (olecranon, triceps brachii) and the cranial aspect of the shoulder (bicipital bursa, biceps brachii). The more the limb is elevated, the more pressure that is applied to the cranial aspect of the shoulder. The position is maintained for 60 seconds (or as long as the horse tolerates it) and the horse is trotted off. Horses with supraglenoid tubercle fractures of the scapula and horses with bicipital bursitis often respond to this type of shoulder manipulation.

The caudal approach to flex the shoulder joint is performed by placing one hand on the olecranon process and pulling the limb caudally. Alternatively, the cranial antebrachium may be grasped and pulled caudally together with the distal limb instead of applying pressure to the olecranon (Figure 3.55). The position is maintained for 60 seconds (or as long as the horse tolerates it), after which the horse is trotted and the degree of lameness is evaluated.

### *Tarsal/Hock Flexion*

The tarsal flexion test or spavin test is somewhat of a misnomer because it flexes the fetlock, stifle, and hip in addition to the tarsus. A positive response to hock flexion is not synonymous with a tarsal problem, but can be used together with other physical examination findings to suggest a problem in the tarsus. Hock flexion is performed by placing the outside hand (when facing the rear of the horse) on the plantar surface of the distal third of the metatarsus and elevating the limb to flex the hock (Figure 3.56). The opposite hand is then placed around the metatarsus and the limb is held with both hands while facing the back of the horse. The grip should avoid the sesamoid bones and be loose enough to avoid excessive pressure to the flexor tendons and suspensory ligament. The fetlock and phalanges should not be forcibly flexed. Alternatively, the tip of the toe can be held so the pastern and fetlock joints are extended and the hock is flexed (Figure 3.57). It also may be beneficial to gradually flex the tarsus to its fullest extent over a 15-second period to avoid resentment by the horse. In some cases, if the horse tends to lean away from the examiner, it may be helpful to have an assistant provide counterbalance to the tuber coxae of the opposite hip. Once the tarsus is in full flexion, it is held in this position for 60 seconds, the limb is released gradually, and the horse is trotted off.

A positive hock flexion test is indicated by an increase in lameness of the flexed limb. However, increased lameness of the opposite limb (standing limb) is thought to occur with some upper limb lameness problems (sacroiliac problems). The first few steps the horse takes after this test are often the most important. The response to tarsal flexion should nearly always be compared to the opposite limb because an asymmetrical response often is an important clinical finding.

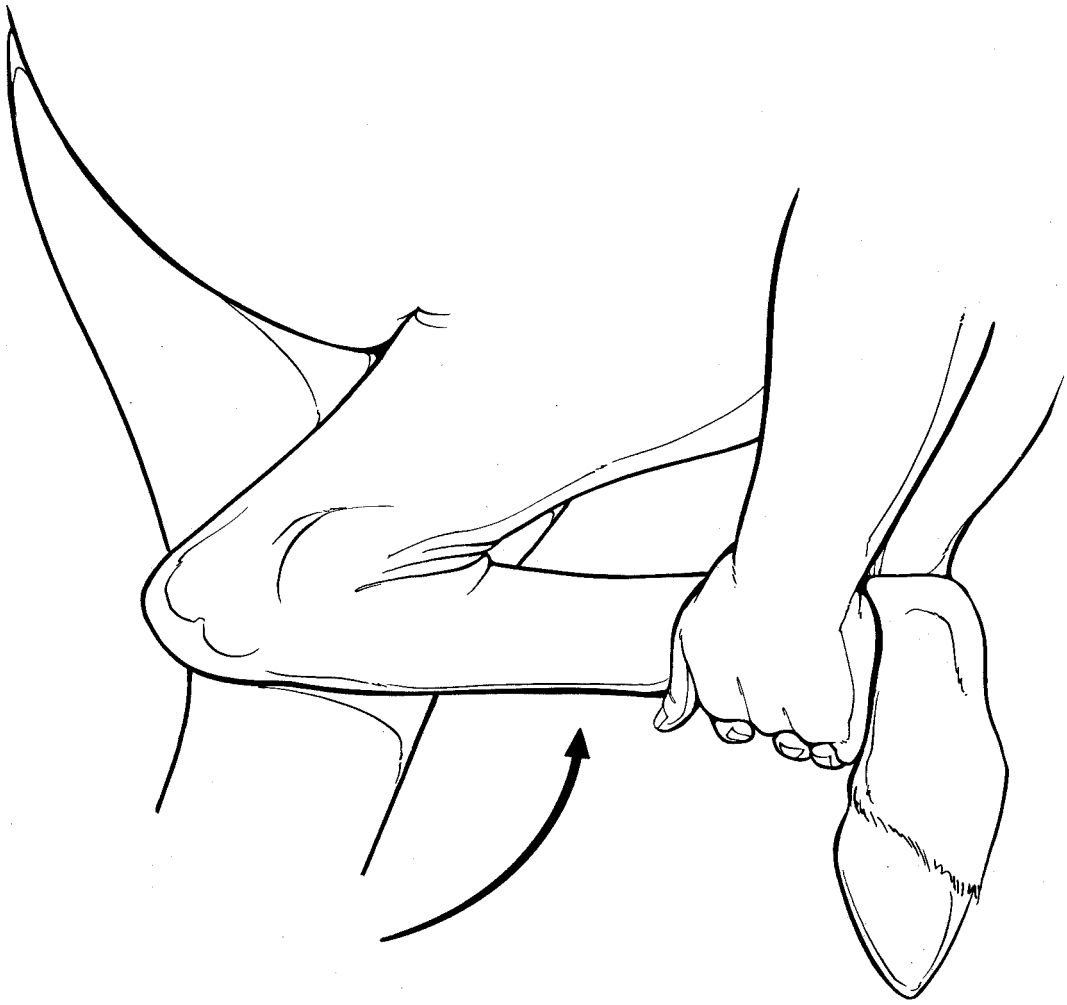

**Figure 3.56.** Hock or tarsal flexion (spavin) test. The hindlimb is flexed so that the metatarsus is approximately parallel to the ground. This test is not specific for the tarsus because it flexes both the stifle and the fetlock to some degree.

### *Stifle Flexion*

Often, the stifle flexion test is used in an attempt to separate stifle pain from tarsal pain in horses that respond to a tarsal flexion test. In most cases the stifle flexion test will flex the hock less than the tarsal flexion test will flex the stifle. In many cases it can be more specific for stifle problems, but this is debatable. It is performed by grasping the distal tibia and pulling the limb backward and upward until maximam stifle flexion is achieved (Figure 3.58). It is best to face the back of the horse with the limb in front of the examiner when performing this test. The limb is held in this position for 60 seconds and the horse is trotted off. Some clinicians prefer to perform the stifle flexion test before the hock flexion test, whereas others do it afterward. A positive tarsal flexion together with a more positive stifle flexion may suggest that the lameness is due to a stifle problem and vice versa.

### *Full Limb Forelimb and Hindlimb Flexion*

Full limb forelimb and hindlimb flexion tests can be used as a quick screening test to determine if more isolated flexion tests may be necessary. If there is no response to flexing all of the joints at one time, the potential for getting positive responses to individual flexion tests is thought to

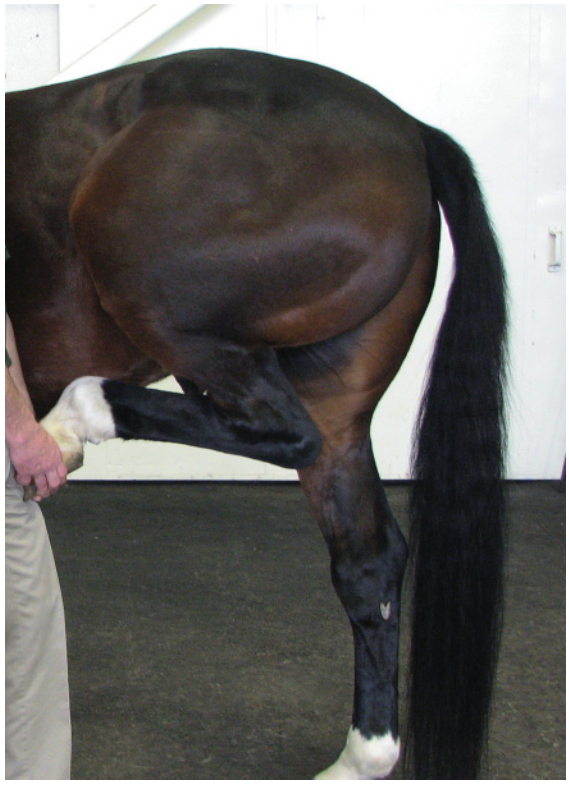

**Figure 3.57.** Positioning to perform hock or tarsal flexion of the hindlimb, with the limb held by the hoof.

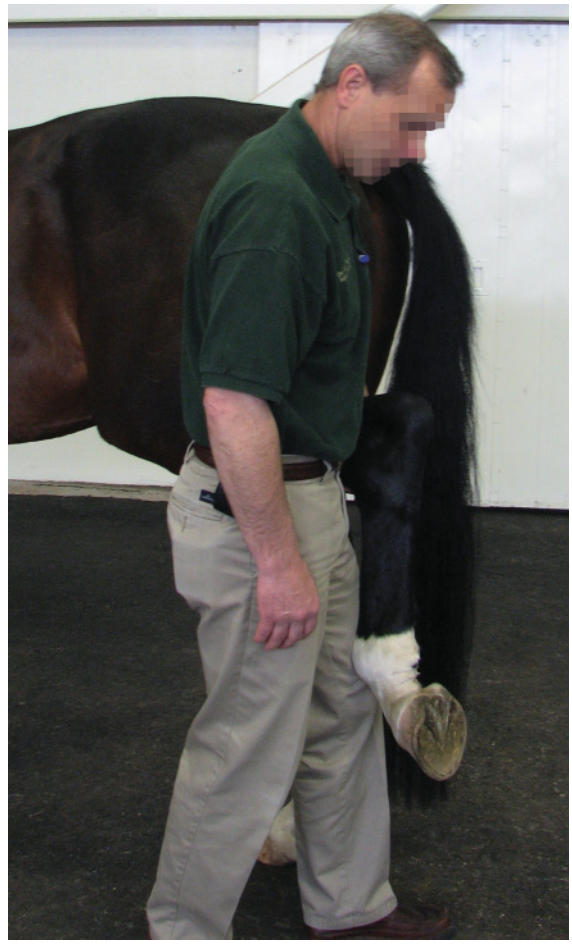

**Figure 3.58.** Flexion of the stifle is performed by pulling the hindlimb caudally and lifting up on the distal tibia.

be unlikely. Full limb forelimb flexion is performed by grasping the foot and lifting the leg to flex the fetlock, carpus, and elbow. The opposite hand is placed on the metacarpus and the limb elevated and pulled forward to extend the shoulder. Full limb hindlimb flexion is performed by grasping the foot and flexing the fetlock, hock, and stifle simultaneously. The hindlimb is pulled out behind the horse to help flex the stifle. The limb is usually held in position for 60 seconds. A negative response is thought to suggest that individual flexion responses will also be negative, but this has not been determined definitively for either the forelimb or hindlimb.

### *Navicular Wedge Test*

The navicular wedge test can be performed in two different ways. A wedge (usually a block of wood) may be placed beneath the frog of the affected foot while the opposite limb is held up. It is thought to apply direct pressure to the frog area, similar to hoof testers. The test is usually performed for one minute, after which the horse is trotted off. Alternatively, the toe is forced into an elevated position in relation to the heel by placing a wooden wedge under the toe (Figure 3.59). This serves to increase the tension on the DDFT and increased pressure over the navicular bone. The wedge also may be applied to the medial or lateral aspect of the foot to manipulate the soft tissues of the digit. The opposite limb is elevated for one minute, and the horse is trotted off. Although these tests are often used for horses with navicular syndrome, any cause of heel/foot pain may be exacerbated with these tests.

### *Direct or Local Pressure Plus Movement*

The premise for applying direct pressure to a specific site and watching the horse trot is to

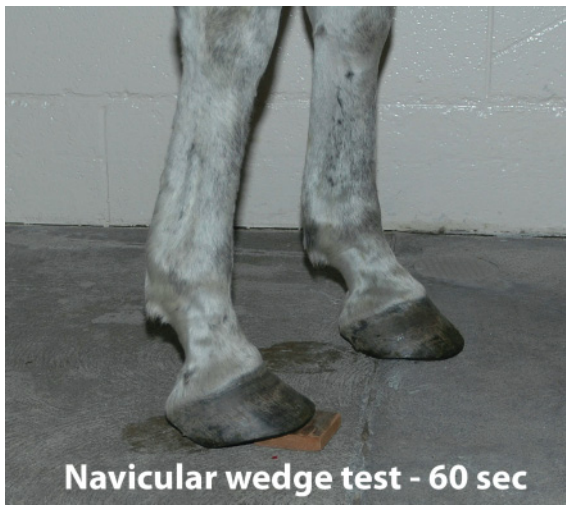


**Figure 3.59.** The navicular wedge test is performed by elevating the toe with a wooden block to increase the tension on the DDFT in the navicular region.

confirm the significance of palpation findings. A positive response to static palpation does not necessarily indicate the site of the problem. However, increasing the baseline lameness by deep palpation of a suspicious area or anatomic structure will often confirm the potential of a problem in the area. Direct pressure usually is applied manually; however, hoof testers can be used to apply pressure to the sole of the foot. The limb is usually elevated, the site is compressed for 15 to 30 seconds, and the horse is trotted off. Exacerbation of the lameness by one or more grades is considered to be a positive response. The direct pressure test is most commonly performed over swellings of the splint bones, dorsal metacarpus, flexor tendons, suspensory body and branches, and medial aspect of the tarsus. It also may be used to assess pain in the proximal suspensory region of both the forelimb and hindlimb as well as several areas of the axial skeleton.

## PERINEURAL ANESTHESIA

### *General Considerations*

Deposition of local anesthetics (i.e. mepivacaine) into synovial structures and perineurally is commonly employed to specifically identify the region(s) causing pain in the lame limb (Table 3.1). Perineural anesthesia also is frequently used to facilitate surgical procedures and reduce postoperative pain.

### *Anesthetics*

The local anesthetics most frequently used are 2% lidocaine hydrochloride (Xylocaine hydrochloride) and 2% mepivacaine hydrochloride (Carbocaine). These solutions are potent and rapidly effective, but can be locally irritating. Mepivacaine is longer lasting and less irritating than lidocaine; therefore, it is used most frequently. Lidocaine is thought to last only 60 minutes, with the maximum effect at 15 minutes. However, a recent study using force plate evaluations indicated that mepivacaine also was only fully effective for 15 to 60 minutes after a palmar digital (PD) nerve block was performed. The effect of the block began to subside between one and two hours, but gait characteristics persisted beyond two hours. This is very important to remember when performing multiple nerve blocks on any given horse over a prolonged period of time. Bupivacaine hydrochloride (marcaine) may be used if the goal is to provide a longer duration of analgesia (four to six hours), such as following surgery. The duration of anesthesia also may be prolonged (up to six hours) by combining local anesthetic with epinephrine. However, swelling is usually more severe and the potential to cause skin necrosis over the site of injection is a serious concern.

### *Skin Preparation*

Most perineural blocks do not require an antiseptic skin preparation. The only skin preparation necessary for most sites of regional anesthesia is scrubbing/wiping the area with $4 \times 4$ gauzes soaked in alcohol until clean. Exceptions to this include the low palmar (four-point) block, the high palmar (four-point) block, the lateral palmar block, and the high plantar (four-point) block. These sites have the potential to enter synovial cavities; therefore, an antiseptic skin preparation should be performed.

### *Restraint*

The type of physical restraint depends on the disposition of the horse and the skill of the veterinarian. Most blocks are performed out of the stocks, but in selected instances stocks can be helpful. When performing local anesthesia, the horse should be haltered and restrained by an attendant who is standing on the same side of the horse. Most perineural blocks of the distal limb can be performed with minimal restraint, depending on the nature of the horse.

**Table 3.1.** Guidelines for perineural local anesthesia.

| Specific block | Needle size | Volume of anesthetic | Skin prep recommended (yes or no) | Location |
|---|---|---|---|---|
| Palmar/plantar digital (PD) | 25 g, 5/8 inch | 1 to 1.5 mL | No | Just above collateral cartilages |
| Basisesamoid (high PD) | 25 g, 5/8 inch | 1.5 to 2 mL | No | Base of the proximal sesamoid bone |
| Pastern ring block | 22 g, 1-1/2 inches | 2 to 3 mL | No | Above collateral cartilages and directed dorsally |
| Abaxial sesamoid | 25 g, 5/8 inch | 1.5 to 2 mL | No | Abaxial surface of proximal sesamoid bone |
| Low palmar or four-point | 22 to 25 g, 5/8 to 1 inch | 2 to 3 mL/site | Yes | Distal metacarpus (above buttons of splint bones) |
| High palmar or four-point | 25 g, 5/8 inch and 20 to 22 g, 1-1/2 inches | 3 to 5 mL/site | Yes | Proximal metacarpus |
| Lateral palmar (lateral approach) | 20 to 22 g, 1 inch | 5 to 8 mL | Yes | Distal to accessory carpal bone |
| Lateral palmar (medial approach) | 25 g, 5/8 inch or 22 g, 1 inch | 2 to 4 mL | No | Medial aspect of accessory carpal bone |
| Ulnar | 20 g, 1-1/2 inches | 10 mL | No | 4 inches above accessory carpal bone |
| Median | 20 to 22 g, 1-1/2 to 2-1/2 inches | 10 mL | No | Caudal to radius below pectoralis muscle |
| Medial cutaneous antebrachial | 22 to 25 g, 1 to 1-1/2 inches | 5 to 10 mL | No | Mid-radius near cephalic and accessory cephalic veins |
| Low plantar or six-point | 25 g, 5/8 inch or 22 g, 1 inch | 2 to 3 mL/site | Yes | Distal metatarsus and each side of long digital extensor tendon |
| High plantar, high four-point, or subtarsal | 25 g, 5/8 inch and 20 to 22 g, 1-1/2 inches | 3 to 5 mL/site | Yes | Proximal metatarsus |
| Deep branch of lateral plantar | 20 to 22 g, 1-1/2 inches | 5 to 7 mL/site | Yes | Lateral aspect of proximal metatarsus |
| Tibial/peroneal | 20 to 22 g, 1-1/2 inches | 10 to 20 mL/site | No | 4 inches above point of hock on lateral and medial aspects of limb |

However, twitch restraint is often very helpful, especially with the more proximal blocks. When using local anesthesia in the hindlimb, the practitioner should always be in a position so that minimal bodily harm will result if rapid movement occurs.

### *Assessment of Response to Blocks*

Skin sensation often is used to assess the success of perineural blocks in the distal limb. This can be performed with a blunt object, such as a pen, hemostat, or needle cap. These objects

should not be jabbed into the skin, but applied gently at first with a gradual increase in pressure. Most horses are receptive to this technique, and will quietly respond if the nerves are not totally desensitized. However, some horses are difficult to read and skin sensation may persist even with an effective block. This is especially true for blocks performed more proximally in the limb (above the fetlock). Other manipulative tests that previously caused pain (such as hoof tester examination, deep palpation, and flexion) may need to be repeated to accurately determine if the block worked. In general, the higher the perineural block, the less accurate skin sensation can be to evaluate the success of the block. This is because deeper tissues are targeted, which may not desensitize the skin surface. For instance, the lateral palmar nerve block and the deep branch of the lateral plantar nerve block target the proximal suspensory region and do not necessarily block the overlying skin.

## *Perineural Anesthesia: Forelimb*

### Palmar Digital (PD) Block (Figure 3.60)

**Quantity of Local Anesthetic:** 1 to 1.5 mL
**Needle Size:** 5/8 inch, 25 gauge
**Injection Technique:** The injection is performed with the foot elevated in most cases. Some prefer to stand with their back toward the animal's hind end while holding the hoof between their knees. Others prefer holding the pastern with one hand while injecting with the other, and assume either a lateral or frontal position in relation to the limb. The PD nerves should be anesthetized just distal to or at the proximal border of the collateral cartilages. Blocking the nerves at this location will reduce the risk of anesthetizing the dorsal branches of the PD nerve. If the PD block is performed 2 to 3 cm above the collateral cartilages, the pastern joint can be desensitized in addition to the foot. The PD nerve and neurovascular bundle are easily palpable at the level of the collateral cartilage just behind the DDFT. A 25-gauge, 5/8-inch needle is inserted in a proximal to distal direction over the nerve, and local anesthetic is injected perineurally.

**Pitfalls:**

1. Blocking too high in the pastern
2. Using too much anesthetic—diffusion decreases specificity
3. Assuming that the PD block only desensitizes the palmar aspect of the foot

### Basisesamoid (High PD) Block (Figure 3.61)

**Quantity of Local Anesthetic:** 1.5 to 2 mL
**Needle Size:** 5/8 inch, 25 gauge
**Injection Technique:** This block is performed similarly to the PD block, except it is more prox-


**Figure 3.60.** This image illustrates the positioning to perform a PD nerve block when facing the back of the horse and holding the limb with one hand. The needle is directed toward the hoof and inserted at or below the level of the collateral cartilages.

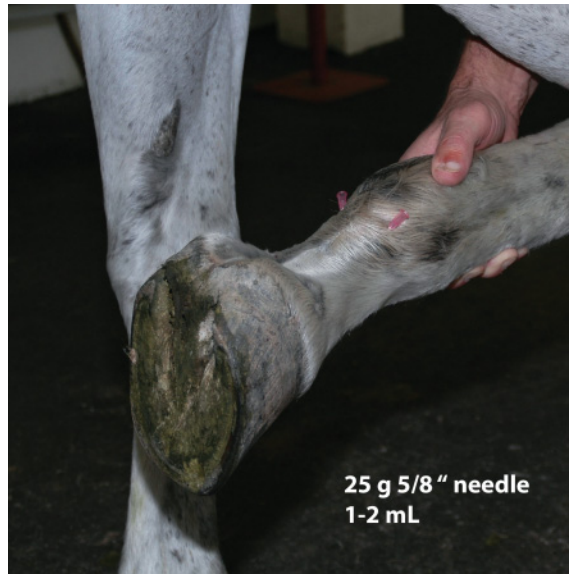


**Figure 3.61.** Needle location to perform a basisesamoid or high PD block.

imal on the limb at the base of the proximal sesamoid bones (often referred to as a high PD block). The PD nerves can be palpated at this location and 1.5 to 2 mL of anesthetic is deposited directly over the nerves. The basi-sesamoid block will desensitize the dorsal branch and the PD nerve at a more proximal location in the pastern. This block will desensitize the palmar/plantar soft tissue structures of the pastern, the PIP joint, and all structures of the foot. Because it is performed at the base of the sesamoid bones, it is unlikely to desensitize any of the fetlock joint.

**Pitfalls:**

1. Using too much anesthetic—diffusion decreases specificity
2. Difficulty in palpating the PD nerves—they are not as superficial in this location compared to more distally

### Abaxial Sesamoid Block (Figure 3.62)

**Quantity of Local Anesthetic:** 1.5 to 2 mL
**Needle Size:** 5/8 inch, 25 gauge
**Injection Technique:** With the limb elevated by holding the fetlock in the palm of the hand, the palmar nerve is isolated by rolling it away from the artery and vein with the thumb or forefinger. A 5/8-inch, 25-gauge needle is used to inject 2 mL of anesthetic perineurally. It is best to use a small volume of anesthetic and direct the needle distally to avoid partial desensitization of the fetlock joint. The biaxial block desensitizes the foot, middle phalanx, PIP joint, distopalmar aspects of the proximal phalanx, distal portions of the SDFT and DDFT, distal sesamoidean ligaments, and the digital annular ligament.

**Pitfalls:**

1. Using too much anesthetic—diffusion can desensitize the fetlock joint or the sesamoid bones
2. May not completely desensitize the skin over the dorsal aspect of the pastern region

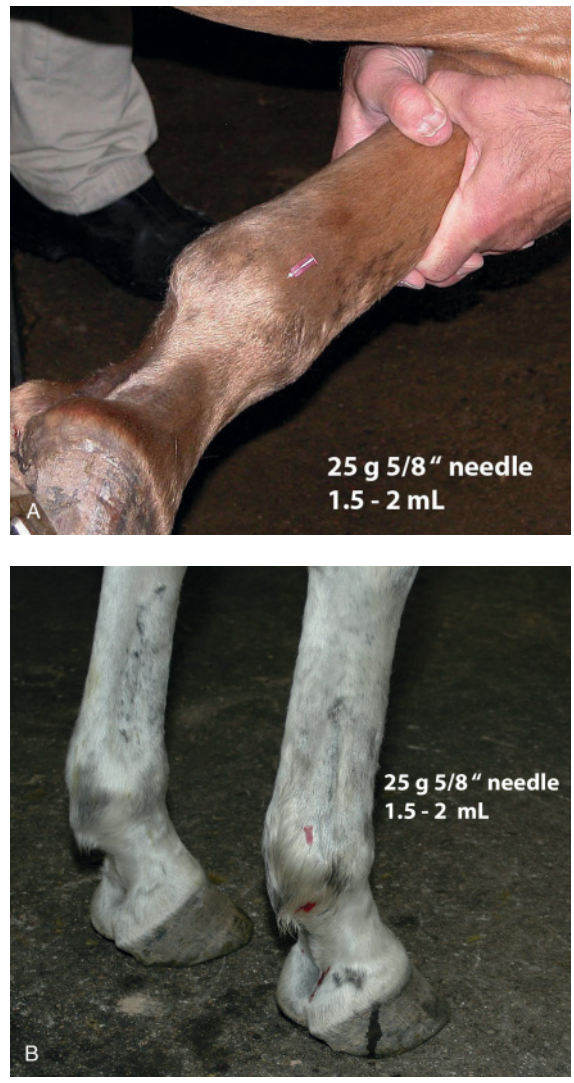


**Figure 3.62.** Needle location to perform an abaxial sesamoid nerve block in the forelimb with (A) the limb held or (B) in the standing horse.

### Low Palmar or Four-point Block (Figure 3.63)

**Quantity of Local Anesthetic:** 2 to 3 mL/site
**Needle Size:** 5/8 or 1 inch, 22 to 25 gauge
**Injection Technique:** The lateral and medial palmar nerves lie between the suspensory ligament and the deep digital flexor tendon. These nerves are relatively deep but can be reached in most cases with a 5/8-inch, 25-gauge needle (a 1-inch, 22-gauge needle also may be used), after which 2 to 3 mL of local anesthetic is deposited. It is best to perform these blocks 1 cm proximal to the distal ends of the splint bones to avoid injection into the digital flexor tendon sheath. The medial and lateral palmar metacarpal nerves innervate the deep structures of the fetlock and course parallel and axial to the second and fourth metacarpal bones. A 5/8-inch, 25-gauge needle or a 1-inch, 22-gauge needle is used to inject 2 to 3 mL of anesthetic around these nerves

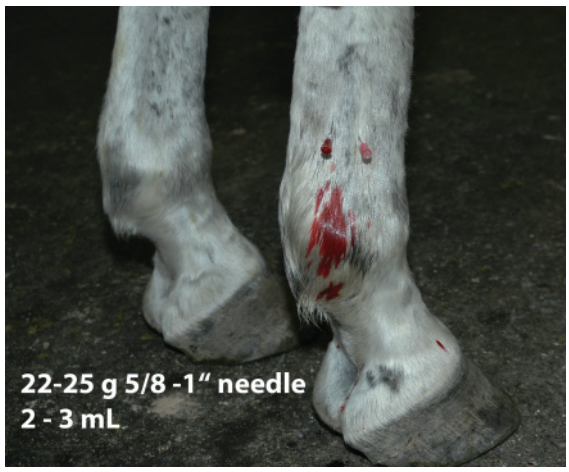


**Figure 3.63.** A low palmar or four-point block in the standing horse.

as they emerge distal to the ends of the second and fourth metacarpal bones. However, because the palmar pouches of the fetlock joint can be inadvertently entered at this location, these nerves can also be anesthetized more proximally.

**Pitfalls:**

1. Inadvertent injection of the fetlock joint or digital flexor tendon sheath (Figure 3.64).
2. Proximal diffusion of anesthetic that may desensitize the body of the suspensory or other more proximal structures
3. Difficulty in assessing whether the palmar metacarpal nerves are desensitized

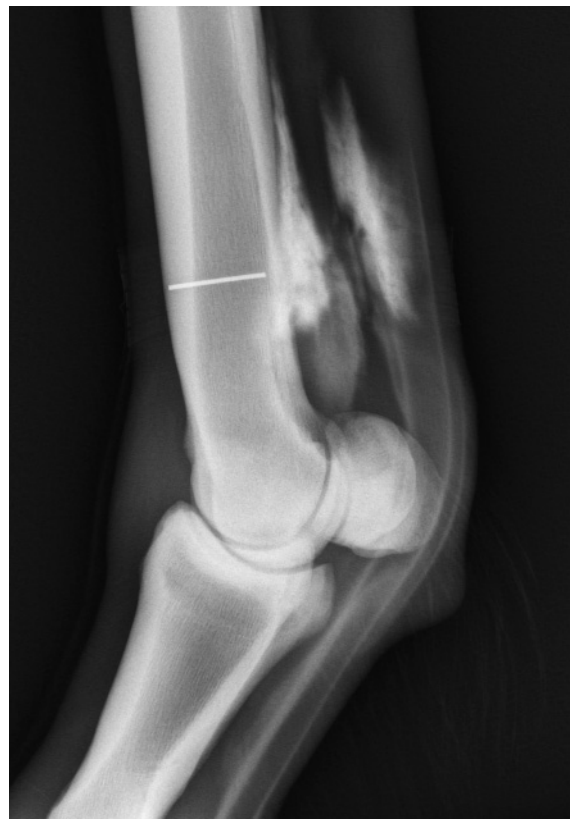

**Figure 3.64.** Contrast material within the DFTS after a low palmar nerve block.

### High Palmar or High Four-point Block (Figure 3.65)

**Quantity of Local Anesthetic:** 3 to 4 mL/site

**Needle Size:** 5/8 inch, 25 gauge and 1.5 inch, 22 gauge

**Injection Technique:** The high four-point or high palmar block is analogous to the low four-point block because the same four nerves are anesthetized in the proximal aspect of the metacarpus just below the carpometacarpal joint. However, the high palmar block is more difficult to perform because the soft tissue structures are more closely confined to the metacarpus and the palmar metacarpal nerves are located deeper within the axial borders of the second and fourth metacarpal bones. The proximal palmar nerves are anesthetized in the groove between the suspensory ligament and the DDFT. A 5/8-inch, 25-gauge needle is inserted through the heavy fascia and 3 to 4 mL of anesthetic is deposited. Blocking just the palmar nerves will not completely desensitize the deep structures of the metacarpus. The palmar metacarpal nerves run parallel and axial to the second and fourth metacarpal bones and each can be desensitized by infiltration of 3 to 4 mL of local anesthetic along the axial surfaces of the metacarpal bones. A 1.5-inch needle is directed toward the palmar metacarpus along the axial borders of the splint bones until bone is contacted. The needle is withdrawn slightly and aspirated to be certain that the needle is not within the carpometacarpal joint before the anesthetic is deposited. Blocking the palmar metacarpal nerves usually is performed with the limb held, whereas anesthesia of the palmar nerves often is easier with the limb bearing weight. These four nerve blocks will

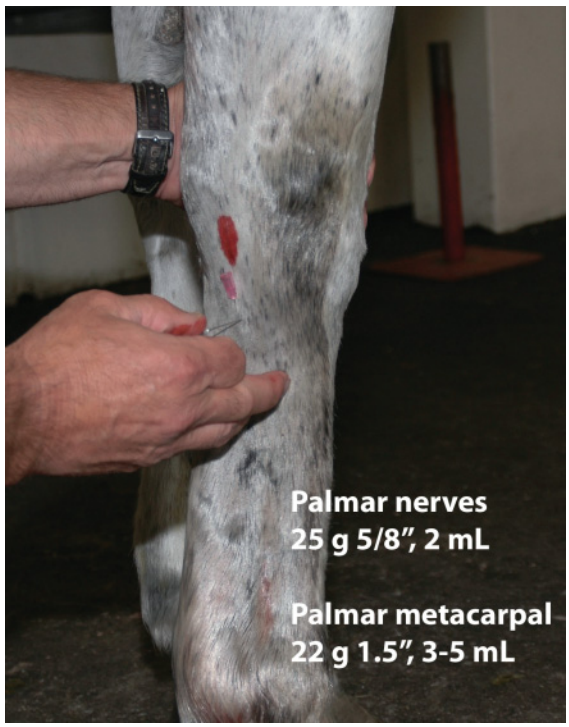


**Figure 3.65.** High palmar or four-point block.

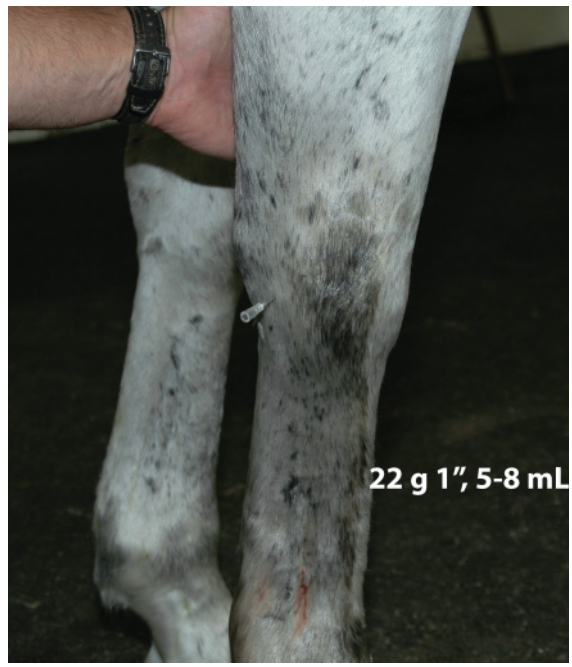


**Figure 3.66.** Lateral approach to block the lateral palmar nerve.

effectively desensitize the deep structures of the metacarpus with the exception of the origin of the suspensory ligament.

**Pitfalls:**

1. Inadvertent injection of the distal outpouchings of the carpometacarpal joint (and therefore the middle carpal joint).
2. Resentment by the horse when blocking the palmar metacarpal nerves axial to the splint bones
3. Difficulty in assessing whether the palmar metacarpal nerves are desensitized
4. Swelling of the proximal metacarpal region that may interfere with a subsequent ultrasound evaluation

### Lateral Palmar Block (Lateral Approach; Figure 3.66)

**Quantity of Local Anesthetic:** 5 to 8 mL
**Needle Size:** 5/8 or 1 inch, 22 to 25 gauge
**Injection Technique:** At the proximal end of the fourth metacarpus, the lateral palmar nerve gives off its deep branch that detaches branches to the origin of the suspensory ligament and divides into the lateral and medial palmar metacarpal nerves. The lateral palmar nerve can be anesthetized just below the accessory carpal bone (lateral approach) or axial to the accessory carpal bone in a more proximal location (medial approach). This block desensitizes the origin of the suspensory ligament and other deep structures of the palmar metacarpus. With the lateral approach, the nerve is anesthetized midway between the distal border of the accessory carpal bone and the proximal end of the fourth metacarpal bone. The needle is directed in a palmarolateral-to-dorsomedial direction and must penetrate the 2- to 3-mm thickness of the flexor retinaculum of the carpus. This block may be performed with the horse standing or with the carpus slightly flexed.

**Pitfalls:**

1. Inadvertent injection into the carpal sheath or middle carpal joint
2. Difficulty in injecting—needle has not penetrated the fascia below accessory carpal bone
3. Difficulty in assessing success of the block. Best done by palpating the absence of pain in the suspensory ligament

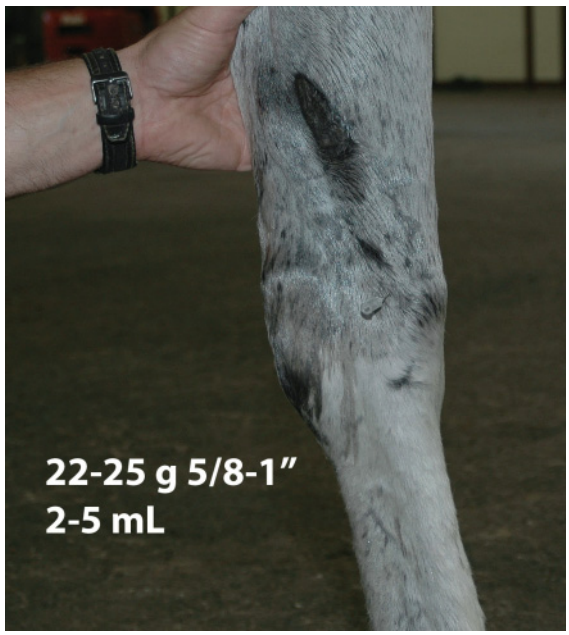


**Figure 3.67.** The medial approach to desensitize the lateral palmar nerve is located on the axial border of the accessory carpal bone.

### Lateral Palmar Block (Medial Approach; Figure 3.67)

**Quantity of Local Anesthetic:** 2 to 5 mL
**Needle Size:** 5/8 or 1 inch, 22 to 25 gauge
**Injection Technique:** With the medial approach, the lateral palmar nerve is blocked medial to the accessory carpal bone. This medial technique is thought to reduce the risk of inadvertent injection into the carpal sheath. The site of injection is a longitudinal groove in the fascia palpable over the medial aspect of the accessory carpal bone, palmar to the insertion of the flexor retinaculum that forms the palmaromedial aspect of the carpal canal. With the limb bearing weight, the needle is inserted into the distal third of the groove in a mediolateral direction perpendicular to the limb.

**Pitfalls:**

1. Difficulty in injecting—needle in fascia or against the medial aspect of the accessory carpal bone
2. Difficulty in assessing success of the block. Best done by palpating the absence of pain in the suspensory ligament

### High Two-point Block

**Quantity of Local Anesthetic:** 2 to 8 mL
**Needle Size:** 5/8 or 1 inch, 22 to 25 gauge
**Injection Technique:** The high two-point block is a combination of the lateral palmar block and the high medial palmar block (one nerve of the high four-point block). When performed, all deep and superficial structures on the palmar aspect of the metacarpus distal to the block will be desensitized. This includes the proximal aspects of the second and fourth metacarpal bones and the origin of the suspensory ligament. This block can be used instead of the high four-point block, and is easier to perform with less risk of complications.

**Pitfalls:**

1. Similar to those of the lateral palmar block and the high palmar block
2. It may be necessary to block the medial palmar nerve in conjunction with blocking the lateral palmar nerve.
3. Difficulty in assessing success of the block. Best done by palpating the absence of pain in the suspensory ligament and metacarpal region.

### Ulnar Nerve Block (Figure 3.68)

**Quantity of Local Anesthetic:** 10 to 12 mL
**Needle Size:** 1-1/2 inch, 20 to 22 gauge
**Injection Technique:** The ulnar nerve is anesthetized approximately four inches (10 cm) proximal to the accessory carpal bone on the caudal aspect of the forearm. Careful palpation will reveal a groove between the flexor carpi ulnaris and ulnaris lateralis muscles. A 1-1/2-inch, 20-gauge needle is inserted through the skin and fascia perpendicular to the limb. Although the depth of this nerve varies, it is usually about 1/4 to 1/2 inch (1 to 1.5 cm) below the skin surface. The local anesthetic (10 to 12 mL) is infused both superficially and deeply in this region. The ulna block will partially desensitize the accessory carpal bone and surrounding structures, palmar carpal region, carpal canal, proximal metacarpus, superficial digital flexor tendon (SDFT), and suspensory ligament.

**Pitfalls:**

1. Injecting too proximally or distally on the limb
2. Injecting the anesthetic too superficially
3. Difficulty in assessing success of the block

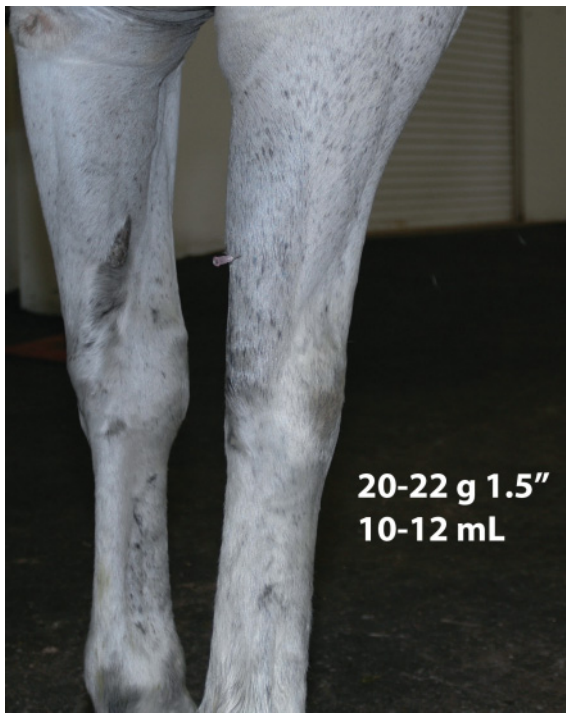


**Figure 3.68.** Ulnar nerve block.


**Figure 3.69.** Median and medial cutaneous antebrachial nerve blocks.

### Median and Medial Cutaneous Antebrachial Blocks (Figure 3.69)

**Quantity of Local Anesthetic:** 10 to 12 mL/site
**Needle Size:** 1 inch, 22 gauge and 1-1/2 to 2-1/2 inch, 20 gauge
**Injection Technique:** The median nerve is anesthetized on the caudomedial aspect of the radius, cranial to the origin of the flexor carpi radialis muscle. The injection site is located just below the elbow joint where the ventral edge of the posterior superficial pectoral muscle inserts in the radius. At this point the nerve is superficial and lies directly on the caudal surface of the radius. A 1-1/2- to 2-1/2-inch, 20-gauge needle is inserted obliquely through the skin and fascia to a depth of 1 to 2 inches. The needle should be kept as close to the radius as possible to avoid the median artery and vein, which lie caudal to the nerve. At least 10 to 12 mL of anesthetic is usually used. Blocking this nerve alone accomplishes little more than a medial and lateral palmar nerve block. However, blocking the median nerve in conjunction with the ulnar nerve will effectively anesthetize most important areas of lameness distal to the blocks.

The two branches of the medial cutaneous antebrachial nerve are blocked on the medial aspect of the forearm halfway between the elbow and the carpus, just cranial to the accessory cephalic vein. The nerve is usually just below the skin; however, its location can vary. It is best to block the subcutaneous tissues both cranial and caudal to the cephalic vein. A 1-inch, 22-gauge needle is used to deposit 5 ml of anesthetic in both locations.

**Pitfalls:**

1. Hitting the median artery or vein or the cephalic vein
2. Injecting too proximally or distally on the limb
3. Injecting the anesthetic too superficially for the median nerve
4. Difficulty in assessing the success of the block.

## *Perineural Anesthesia—Hindlimb*

The plantar digital, basisesamoid, and abaxial sesamoid nerve blocks in the hindlimb are performed in a similar manner to those in the forelimb. However, the PD and basisesamoid blocks are more difficult to perform in the hindlimb because the distal limb flexes when the limb is


**Figure 3.70.** Low six-point block in the hindlimb. The nerves on either side of the long digital extensor tendon also are blocked (arrow) to completely desensitize the fetlock region.

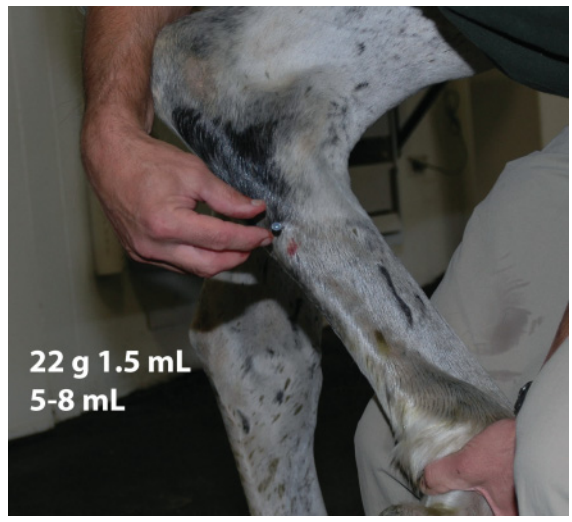


**Figure 3.71.** The high plantar or subtarsal nerve block can be performed in the proximal metatarsal region in a similar manner as the high palmar block. The same location can be used to directly infiltrate the proximal suspensory region.

held off the ground due to the reciprocal apparatus.

### Low plantar (Six-point) Block (Figure 3.70)

**Quantity of Local Anesthetic:** 2 to 4 mL
**Needle Size:** 5/8 or 1 inch, 22 to 25 gauge
**Injection Technique:** The plantar and plantar metatarsal nerves in the distal metatarsus are blocked in a similar manner to the corresponding nerves in the forelimb. One difference in the neuroanatomy between the forelimb and the hindlimb is that lateral and medial dorsal metatarsal nerves from the deep peroneal (fibular) nerve course over the dorsolateral and dorsomedial surfaces of MTIII and digits. Anesthesia of the dorsal metatarsal nerves is performed by injecting 2 to 3 mL of local anesthetic subcutaneously, lateral and medial to the long digital extensor tendon using a 5/8-inch, 25-gauge needle. Blocking the dorsal metatarsal nerves together with the plantar and plantar metatarsal nerves will effectively anesthetize all structures innervated by the nerves distal to the block.

**Pitfalls:**

1. Resentment by the horse if the periosteum of the metatarsus is contacted with the needle.
2. Inadvertent injection into the fetlock joint or digital flexor tendon sheath
3. Difficulty in assessing the success of the block

### High Plantar (Subtarsal) Blocks (Figure 3.71)

**Quantity of Local Anesthetic:** 3 to 5 mL
**Needle Size:** 5/8 or 1-1/2 inche, 20 to 25 gauge
**Injection Technique:** The high plantar block anesthetizes the medial and lateral plantar and plantar metatarsal nerves just below the tarsus analogous to the high palmar block of the forelimb. The plantar metatarsal nerves can be blocked using a 1.5-inch, 20-gauge needle inserted axial to the second and fourth metatarsal bones and directed dorsally toward the plantar aspect of the metatarsus.

Inadvertent administration of anesthetic into the tarsal sheath or the tarsometatarsal joint can occur when performing anesthesia of the plantar metatarsal nerves. The medial and lateral plantar nerves can be anesthetized by placing 3 to 5 mL of anesthetic through the heavy fascia adjacent to the dorsal surface of the DDFT in the proximal metatarsal region using a 5/8-inch, 25-gauge needle. The high plantar block will effectively desensitize the second and fourth metatarsal bones, the suspensory ligament and its origin, and the flexor tendons in the metatarsal region.

One study concluded that the high plantar nerve block cannot be used to differentiate between flexor tendon and suspensory ligament lesions as horses with both conditions improved after the block.

**Pitfalls:**

1. Inadvertently injecting into the tarsal sheath or tarsometatarsal joint
2. Difficulty in performing the blocks because of the anatomic configuration of the splint bones and the resentment of the horse
3. Difficulty in assessing the success of the block

### Deep Branch of the Lateral Plantar Nerve (DBLPN) Block (Figure 3.72)

**Quantity of Local Anesthetic:** 5 to 8 mL
**Needle Size:** 1 to 1-1/2 inches, 20 to 23 gauge
**Injection Technique:** The deep branch of the lateral plantar nerve (DBLPN) innervates the proximal suspensory in the hindlimb and is removed to treat some horses with hindlimb proximal suspensory desmitis. Two different techniques have been described. With the first approach, a 1-inch, 23-gauge needle is inserted 15 mm distal to the head of the fourth metatarsus and directed perpendicular to skin between the axial border of the fourth metatarsus and the SDFT to a depth of approximately 25 mm. Alternatively, a 1.5-inch, 20-gauge needle is inserted 20 mm distal and plantar to the head of the fourth metatarsus and directed proximodorsally and axial to the bone. The needle is advanced to a depth of 1 to 2 cm and 5 to 7 mL of anesthetic is deposited. It is usually best to hold the limb to perform either of these techniques. The single injection technique for the DBLPN is thought to provide a reliable method for perineural analgesia of the deep branch of the lateral plantar nerve (and therefore the proximal suspensory region) with minimal risk of inadvertently desensitizing other tarsal structures.

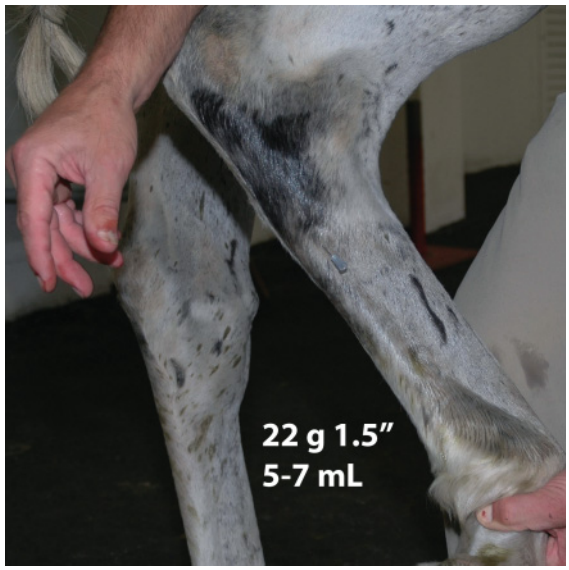


**Figure 3.72.** The deep branch of the lateral plantar nerve (DBLPN) can be desensitized by inserting a needle 20 mm below the head of the lateral splint and directing it proximodorsally and axial to the bone.

**Pitfalls:**

1. Difficulty in injecting—needle in origin of suspensory or overlying fascia. Most likely the needle is too deep.
2. Difficulty in assessing success of the block—best done by palpating absence of pain in the suspensory ligament
3. Inadvertent desensitization of distal tarsal joints

### Tibial and Peroneal Block (Figure 3.73)

**Quantity of Local Anesthetic:** 10 to 20 mL/site
**Needle Size:** 1-1/2 to 2 inches, 20 to 22 gauge
**Injection Technique:** Anesthetizing the tibial and deep and superficial peroneal nerves above the point of the hock desensitizes the entire distal limb. These blocks can be helpful to diagnosis some horses with hock lameness, or can be used to rule out whether the pain causing the lameness is located within the hock or distal limb. The site for injection of the tibial nerve is approximately 4 inches above the point of the hock on the medial aspect of the limb, between the Achilles tendon and the deep digital flexor muscle. The block may be performed by standing on the lateral side of the limb to be blocked or by reaching across from the opposite limb to access the medial aspect of the limb. A small amount of anesthetic placed in the skin and subcutaneous tissues may minimize the horse's reaction to the block. A 1-1/2–inch, 20- to 22-gauge needle is used to deposit 15 to 20 mL of anesthetic in several tissue planes in the fascia that overlies the deep digital flexor muscle. Blocking the tibial nerve provides anesthesia to the plantar tarsus, metatarsus, distal Achilles tendon, calcaneus, suspensory ligament, and most of the foot.

To completely desensitize the hock and limb distal to the hock, the deep and superficial

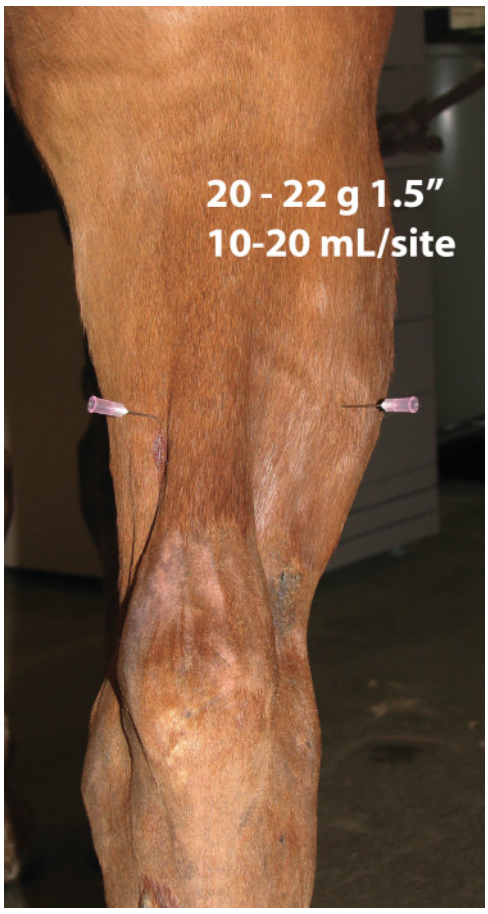


**Figure 3.73.** Image illustrating the locations to block the tibial and peroneal nerves. The site for injection of the tibial nerve is approximately 4 inches (10 cm) above the point of the hock on the medial aspect of the limb, between the Achilles tendon and the deep digital flexor muscle. The location to block the peroneal nerves is approximately 4 inches (10 cm) above the point of the hock on the lateral aspect of the limb in the groove formed by the muscle bellies of the lateral and long digital extensor muscles.

peroneal (fibular) nerves must be anesthetized. The location of injection is approximately 4 inches above the point of the hock on the lateral aspect of the limb in the groove formed by the muscle bellies of the lateral and long digital extensor muscle. A 1-1/2- to 2-inch, 20-gauge needle is inserted in a slightly caudal direction until the needle contacts the caudal edge of the tibia. Ten to 15 mL of anesthetic is injected on the lateral border of the cranial tibial muscle close to the tibia. The needle is then retracted and another 10 to 15 mL of local anesthetic is injected more superficially in several planes to be sure that the superficial peroneal nerve is blocked. The depth of the superficial peroneal nerve can vary, so the more superficial injection should include a region from 0.6 cm to 2.5 cm deep.

**Pitfalls:**

1. Difficulty in palpating the tibial nerve
2. Placing the anesthetic too proximal or distal on the leg
3. Placing the anesthetic too superficially to block both branches of the peroneal nerves
4. Difficulty in assessing the success of the block

## INTRASYNOVIAL ANESTHESIA

### *General Considerations*

Deposition of local anesthetics (i.e., mepivacaine) into synovial structures is commonly employed to specifically identify the region(s) causing pain in the lame limb (Table 3.2). Intrasynovial injections of medications are also commonly employed to treat a variety of joint, tendon sheath, and bursal conditions. Injection of saline into synovial cavities also can be helpful to determine whether traumatic injuries have penetrated the synovial structure. Knowing several different approaches to these synovial cavities can be very helpful, depending on the location of the injury.

### *Anesthetics*

The three commonly used local anesthetics differ in their time of onset of anesthesia and their duration of action. Lidocaine and mepivacaine typically have an onset of less than 10 minutes, while bupivacaine is generally longer than 10 minutes. Mepivacaine has a longer duration of analgesia (2 to 3 hours) compared to lidocaine (1 to 1-1/2 hours). Bupivacaine is thought to last anywhere from 3 to 8 hours. Mepivacaine is the local anesthetic preferred by most clinicians because it is thought to be less irritating and has a more rapid onset than lidocaine.

### *Skin Preparation*

Techniques for preparing the skin for intrasynovial injections vary among clinicians. Many clip the hair over the injection site, although this has been shown to be unnecessary if an adequate sterile preparation of the site is performed.

**Table 3.2.** Guidelines for intrasynovial anesthesia.

| Synovial cavity | Needle size | Volume of anesthetic | Approaches and limb position (standing or held) |
|---|---|---|---|
| Coffin joint | 20 to 22 g, 1 to 1-1/2 inches | 4 to 6 mL | Dorsal approaches: standing<br>Lateral approach: standing or held |
| Pastern joint | 20 to 22 g, 1-1/2 inches | 4 to 6 mL | Dorsolateral approach: standing<br>Palmar/plantar approach: held |
| Fetlock joint | 20 to 22 g,<br>1 to 1-1/2 inches | 8 to 12 mL | Proximal palmar/plantar approaches: standing or held<br>Collateral sesamoidean approach: held<br>Distal palmar/plantar approach: standing<br>Dorsal approach: standing |
| Carpal joints | 20 to 22 g, 1 to 1-1/2 inches | 8 to 10 mL | Dorsal approaches: held<br>Palmar approaches: standing |
| Elbow | 20 g, 1-1/2 inches or 20 g, 3-1/2 inches | 20 to 30 mL | All approaches: standing |
| Shoulder | 18 to 20 g, 3-1/2 inches | 20 to 40 mL | All approaches: standing |
| Tarsometatarsal joint | 20 g, 1 to 1-1/2 inches | 4 to 6 mL | All approaches: standing |
| Distal intertarsal joint | 25 g, 5/8 inch or 22 g, 1 inch | 3 to 5 mL | All approaches: standing |
| Tarsocrural joint | 20 to 22 g, 1-1/2 inches | 10 to 20 mL | All approaches: standing |
| Femoropatellar joint | 20 g, 1-1/2 to 3-1/2 inches | 30 to 40 mL | All approaches: standing |
| Medial femorotibial joint | 20 g, 1-1/2 inches | 20 to 30 mL | All approaches: standing |
| Lateral femorotibial joint | 20 g, 1-1/2 inches | 20 to 30 mL | All approaches: standing |
| Coxofemoral joint | 16 to 18 g, 6- to 8-inch spinal | 30 to 60 mL | All approaches: standing |
| Sacroiliac joint | 15 to 16 g, 10-inch spinal | 7 to 10 mL | All approaches: standing |
| Digital flexor tendon sheath | 20 to 22 g, 1 to 1-1/2 inches | 8 to 15 mL | Proximal approach: standing<br>All other approaches: held |
| Carpal sheath | 20 g, 1-1/2 to 3-1/2 inches | 15 to 30 mL | Medial approach: standing<br>Lateral approach: held |
| Tarsal sheath | 20 g, 1-1/2 inches | 15 to 20 mL | Medial approach: standing |
| Extensor carpi radialis sheath | 20 g, 1-1/2 inches | 10 to 20 mL | All approaches: standing or held |
| Calcaneal bursa | 20 g, 1-1/2 inches | 10 to 15 mL | Distal approach: standing<br>Proximal approach: standing or held |
| Bicipital bursa | 18 to 20 g, 3-1/2 to 5 inches or 20 g, 1-1/2 inches | 20 to 30 mL | All approaches: standing |
| Trochanteric bursa | 18 to 20 g, 1-1/2 to 3-1/2 inches | 7 to 10 mL | All approaches: standing |
| Cunean bursa | 20 to 22 g, 1 inch | 2 to 3 mL | Medial approach: standing |

Regardless of whether or not the hair is clipped, a minimum 5-minute sterile scrub of the site using an antiseptic (povidone-iodine or chlorhexidine) and alcohol or saline should be performed. Sterile gloves are recommended and are always used by the author.

### *Restraint*

The type of physical restraint depends on the disposition of the horse and the skill of the veterinarian. Twitch restraint is recommended by the author to perform all intrasynovial injections unless it is not tolerated by the horse. Horses being treated by intrasynovial injections often are routinely tranquilized, but this is not usually possible for injections used for diagnostic purposes. In addition, the smallest gauge needle as possible (usually 20 gauge or smaller) should be used to minimize objection by the horse. Most blocks are performed out of the stocks, but in selected instances stocks can be helpful. Examples include the sacoiliac and coxofemoral joints.

### *Assessment of Response to Blocks*

Most intrasynovial blocks take effect quickly and their effects wear off quickly. The response to the block should be assessed no later than 10 minutes after performing the block and then again at 20 to 30 minutes if no improvement in the lameness is observed at the initial re-evaluation. A positive response to an intrasynovial block should be observed within 30 minutes in most cases. In general, at least a 50% improvement in lameness should be observed to suggest that the synovial structure is the primary location of the lameness. Not all intrasynovial blocks are specific for the joint, tendon sheath, or bursae, but they are typically more specific than most perineural nerve blocks. Examples include the distal interphalangeal (DIP) joint, middle carpal joint, tarsometatarsal (TMT) joint, and distal intertarsal (DIT) joint. Diffusion of anesthetic to local structures, inadvertent anesthesia of peripheral nerves closely associated with the synovial cavity outpouchings, and the possibility that the injection was not in the synovial cavity should all be considered when assessing the response to intrasynovial injections.

### *Joint Anesthesia*

#### Distal Interphalangeal (DIP) Joint

**Quantity of Local Anesthetic:** 4 to 6 mL
**Needle Size:** 1 to 1-1/2 inches, 20 or 22 gauge

**Injection Techniques:**

- Dorsolateral approach (Figure 3.74): This approach to the DIP joint is performed with the horse standing. The site of injection for the dorsolateral approach is 1/2 inch above the coronary band and 3/4 to 1 inch lateral (or medial) to midline. A 1- to 1-1/2-inch, 20-gauge needle is inserted from a vertical position and directed distally and medially toward the center of the foot at approximately a 45° angle. The needle should enter the DIP joint capsule at the edge of the extensor process. If entry into the joint is uncertain, the needle can be directed at a more acute angle (more horizontal) to the skin and inserted until the needle contacts the distal end of the second phalanx (P2). It then is "walked" distally until the joint is penetrated.
- Dorsal parallel or perpendicular approaches (Figure 3.75): Some prefer to enter the joint on dorsal midline using the proximal outpouching of the DIP joint above the extensor process. The injection site is just above the coronary band, 1/4 to 1/2 inch above the edge of the hoof wall on the dorsal midline of the foot. With the dorsal perpendicular approach, the needle is directed downward perpendicular to the bearing surface of the foot. With the dorsal parallel approach, the needle is directed parallel or slightly downward (hub of the needle is moved proximally) to the ground to a depth of approximately 1/2 inch. The dorsal parallel approach usually is easier to perform and is recommended by many clinicians.
- Lateral approach (Figure 3.76): The site for injection for the lateral approach is bounded distally by a depression along the proximal border of the collateral cartilage approximately midway between the dorsal and palmar/plantar border of P2. A 1-inch, 20-gauge needle is directed downward at a 45° angle toward the medial weight-bearing hoof surface. Most horses appear to tolerate this technique very well. However, the specificity of the lateral approach is thought to be less than the dorsolateral approach. In one study using the lateral approach, only 65% of the limbs had contrast exclusively in the DIP joint, 20% had contrast in the digital sheath, and 5% had contrast in the subcutaneous tissues.

**Pitfalls:**

1. Using more than 6 mL of anesthetic and blocking the palmar/plantar digital nerves
2. Interpreting a positive DIP joint block as only a coffin joint problem

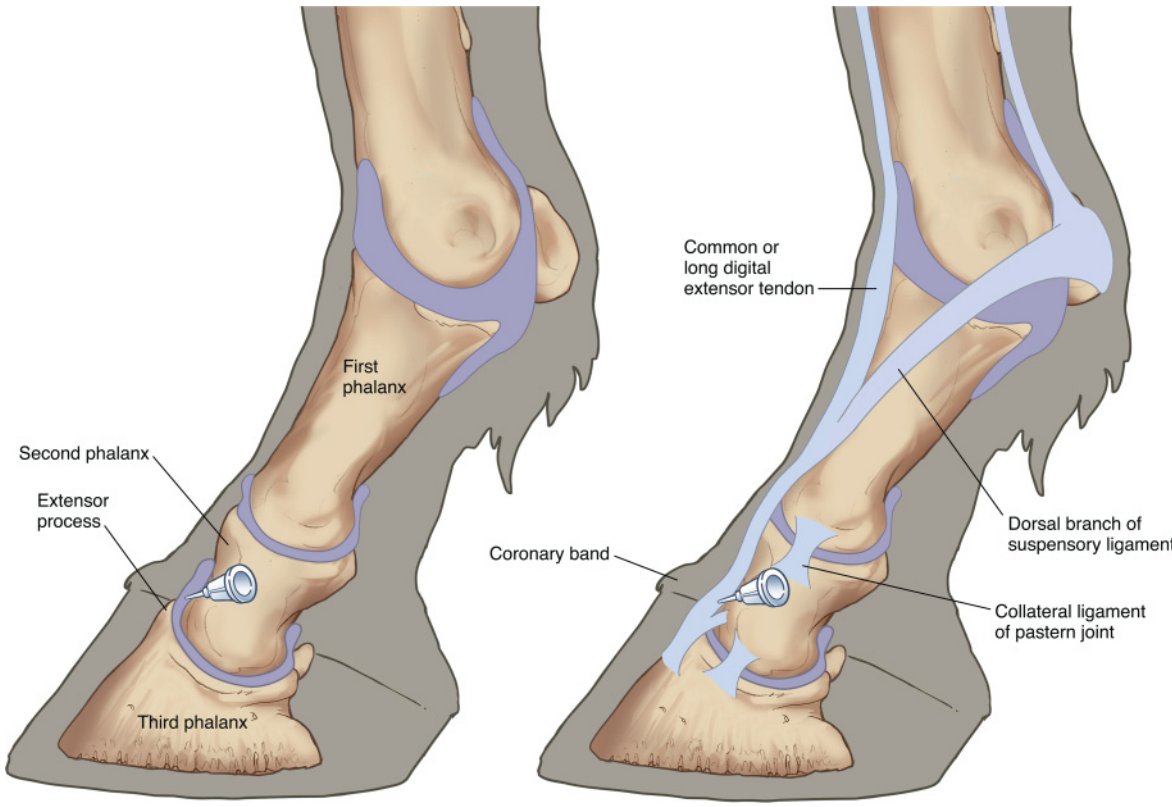


**Figure 3.74.** Dorsolateral approach to the coffin joint.

3. Entering the digital flexor tendon sheath when using the lateral approach
4. Contacting bone due to incorrect angle of needle with the dorsolateral approach
5. Inability to obtain synovial fluid

## Proximal Interphalangeal Joint

**Quantity of Local Anesthetic:** 3 to 5 mL
**Needle Size:** 1 to 1-1/2 inches, 20 or 22 gauge
**Injection Techniques:**

- Dorsolateral approach (Figure 3.77): The dorsolateral approach can be done while the horse is standing or with the limb extended and the sole supported on the knee. The condylar eminences of the distolateral aspect of the first phalanx (P1) are identified and a 1-1/2-inch, 20-gauge needle is inserted parallel to the ground surface 1/2 inch distal to the palpable eminence. The needle is directed underneath the edge of the extensor tendon dorsal to the collateral ligament to enter the joint at a depth of 1/2 inch.
- Palmar/plantar approach (Figure 3.78): The palmaro/plantaroproximal approach is best performed with the distal limb in a flexed position. A 1-1/2-inch, 20-gauge needle is inserted perpendicular to the limb into the palpable V-depression formed by the palmar aspect of P1 dorsally, the distal eminence of P1 distally, and the lateral branch of the SDFT as it inserts on the eminence of P2 palmarodistally. This corresponds to the transverse bony prominence on the proximopalmar/plantar border of P2 that is usually easily palpable. The author prefers to angle the needle slightly dorsally to contact P1, and then direct the needle along the palmar/plantar aspect of the bone. This ensures that the needle is just behind P1, where it will enter the PIP joint capsule at a depth of approximately 1 inch.

**Pitfalls:**

1. No easily palpable joint pouches because of extensor tendon dorsally and the ligaments/tendons on the palmar/plantar aspect

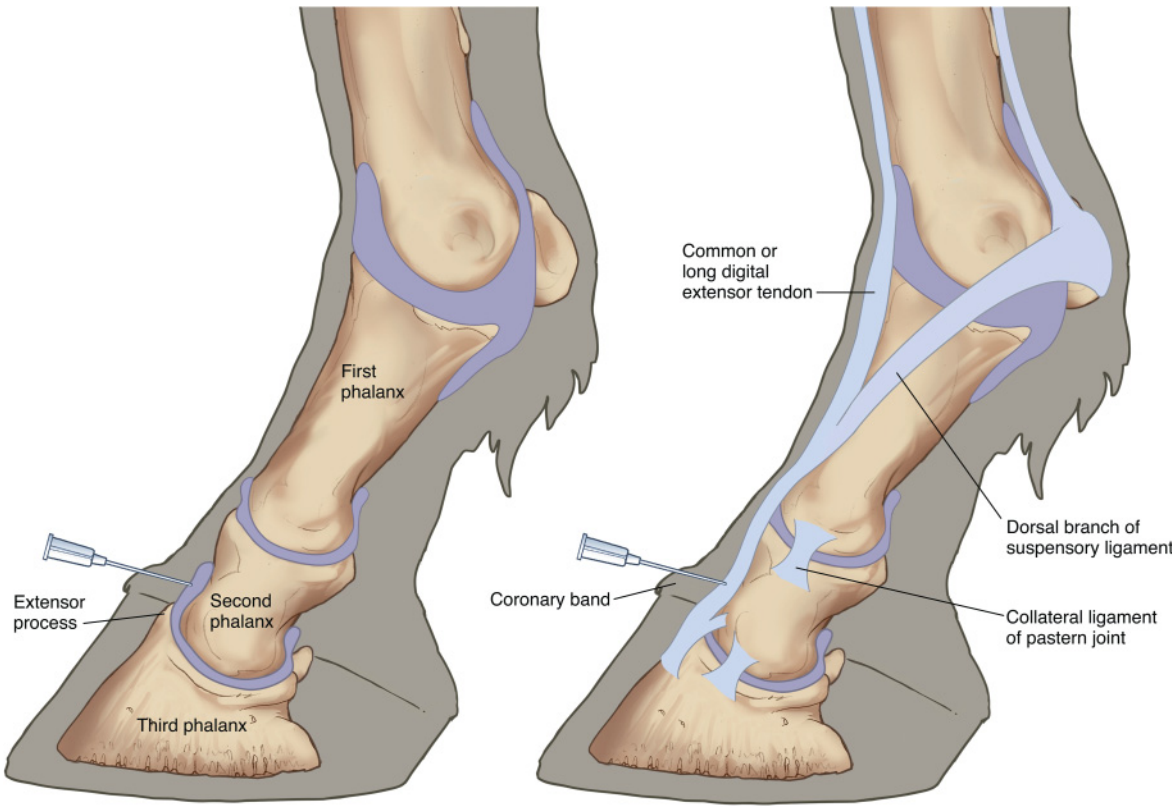


**Figure 3.75.** Dorsal parallel approach to the DIP joint.

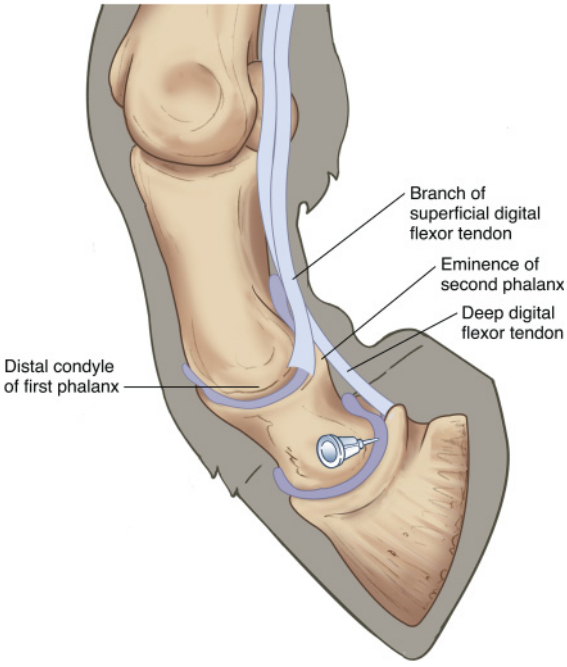


**Figure 3.76.** Lateral approach to the DIP joint.

2. Difficulty to "feel" the needle penetrate the joint space dorsally
3. Placing the needle too distally when using the palmar/plantar approach
4. Injecting the digital flexor tendon sheath when using the palmar/plantar approach

### Metacarpo/Metatarsophalanageal (MCP/MTP; Fetlock) Joint

**Quantity of Local Anesthetic:** 8 to 12 mL
**Needle Size:** 1 to 1-1/2 inches, 20 or 22 gauge
**Injection Techniques:**

- Proximal palmar/plantar approach (Figure 3.79): The boundaries of the palmar/plantar pouches of the fetlock joint are the apical border of the proximal sesamoid bones distally, the distal ends of the splint bones proximally, the third metacarpal/metatarsal bone dorsally, and the branch of the suspensory ligament palmar/plantarly. When performing this approach in the standing patient, a 1- to 1-1/2-inch, 20-gauge needle is inserted from lateral to medial and directed distally at a 45°

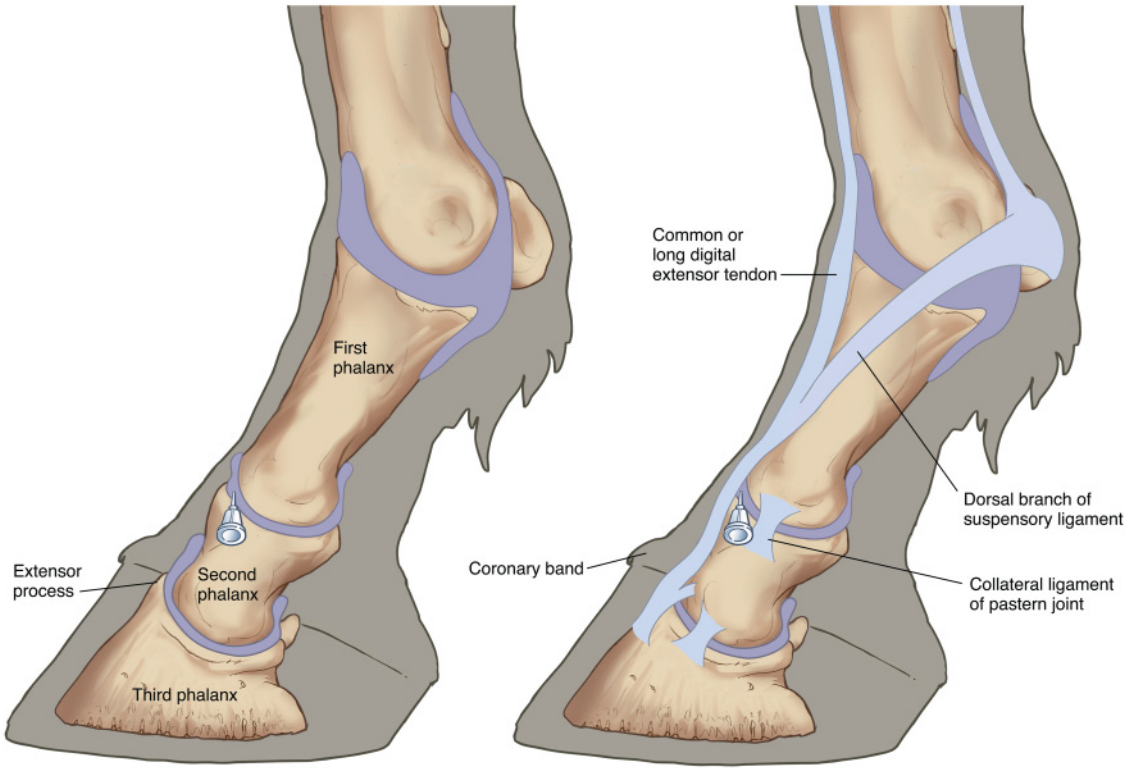


**Figure 3.77.** Dorsolateral approach to the PIP joint.

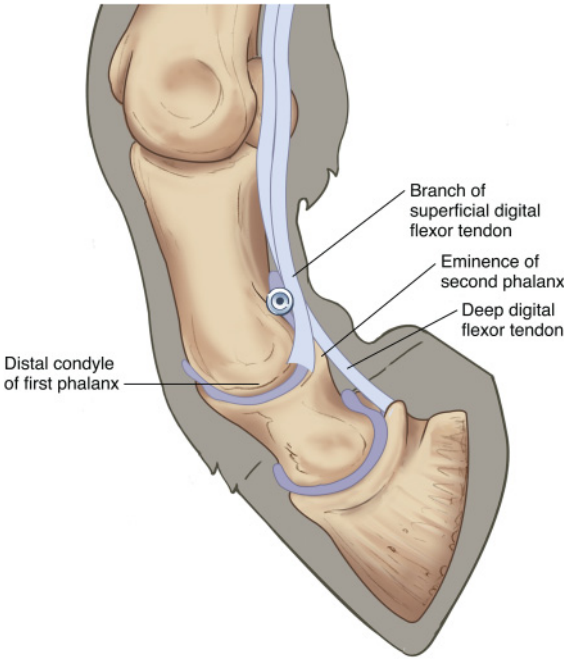


**Figure 3.78.** Palmar/plantar approach to the PIP joint.

angle to the long axis of the limb. The disadvantages of this approach are the possibility of contaminating the synovial fluid sample with blood because of the highly vascular synovial membrane and the inability to aspirate synovial fluid because the synovial villi plug the needle.

Performing the palmar/plantar approach with the fetlock flexed can potentially minimize these complications. With the fetlock flexed there is a very palpable depression at the very distal aspect of the pouch just above the branch of the suspensory ligament. A 1- to 1-1/2-inch, 20-gauge needle is inserted at this location and directed distally at a 45° angle. The more distal location in the palmar/plantar pouch reduces the risk of iatrogenic hemorrhage.

- Collateral sesamoidean approach (Figure 3.80): Arthrocentesis of the fetlock through the lateral collateral sesamoidean ligament is probably the best approach to obtain a hemorrhage-free synovial fluid sample. The fetlock is flexed to increase the space between the articular surfaces of the proximal sesamoid bones and the back of the metacarpus/

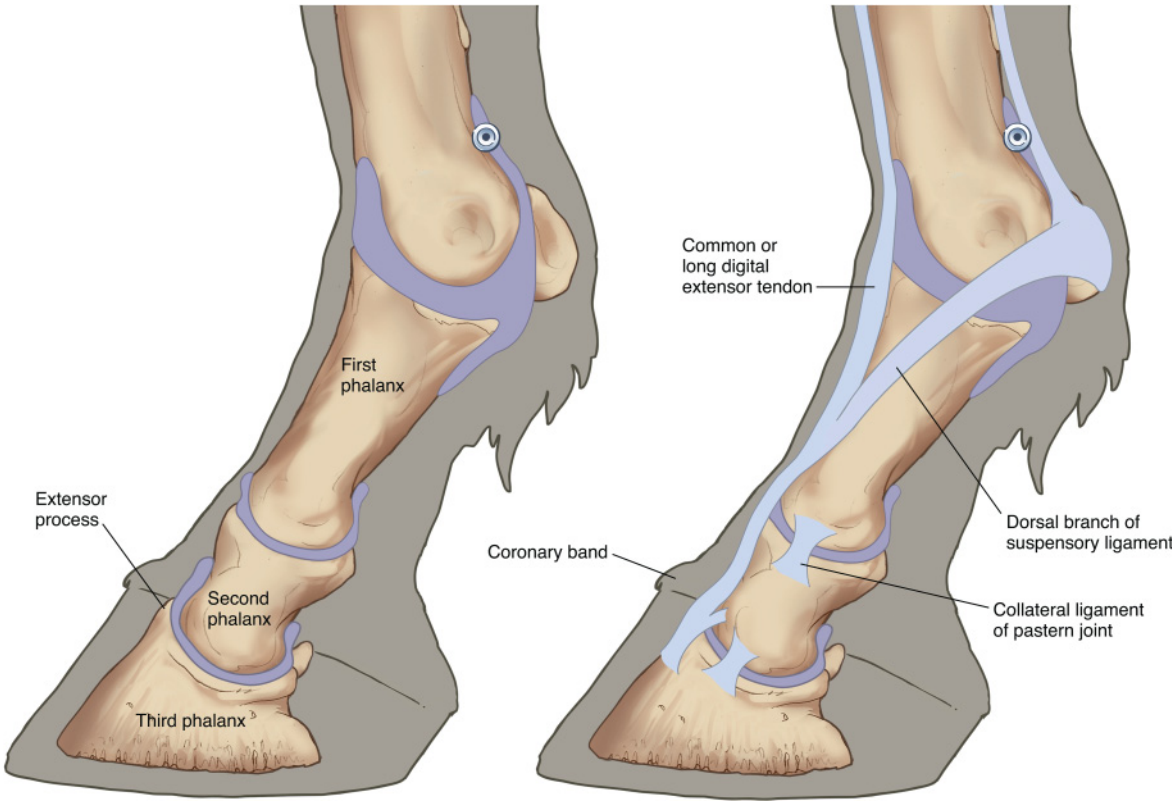


**Figure 3.79.** Proximal palmar/plantar approach to the fetlock joint in the standing horse.

metatarsus. The depression between the bones is palpated and a 1-inch, 20-gauge needle inserted through the collateral sesamoidean ligament perpendicular to the limb. If the needle fails to advance, it is most likely contacting bone and will need to be redirected to enter the joint space.

- Distal palmar/plantar approach (Figure 3.81): The distal palmar/plantar approach is performed in the palpable depression formed by the distal aspect of the proximal sesamoid bone and the proximopalmar/plantar eminence of P1. The landmarks are the distal aspect of the proximal sesamoid bone and collateral sesamoidean ligament proximally; the proximal palmar/plantar eminence of P1 distally; and the digital vein, artery, and nerve palmar/plantarly. A 1-1/2-inch, 20-gauge needle is inserted in the depression and directed slightly dorsally (10° to 20°) and proximally (10°) until the joint is entered. To avoid penetration of the digital sheath it is important that the needle be inserted dorsal to the palmar digital artery, vein, and nerve. The advantages to this approach are that the landmarks are easily palpable, synovial fluid is often obtained, it can be performed in the standing horse.
- Dorsal approach (Figure 3.82): The dorsal approach usually is performed with the limb bearing weight. The needle is inserted proximal to the proximodorsal limits of P1 in the palpable joint space in a slightly oblique manner, either lateral or medial to the extensor tendon. The fetlock joint capsule is thicker in this location than in the palmar/plantar pouch and appears to cause greater discomfort to the horse than the other techniques.

**Pitfalls:**

1. Blood contamination and inability to aspirate synovial fluid with the proximal palmar/plantar approach
2. Contacting bone when using the collateral sesamoidean approach
3. Incorrect needle angle when using the distal palmar/plantar approach
4. Damaging the articular surfaces with the dorsal approach

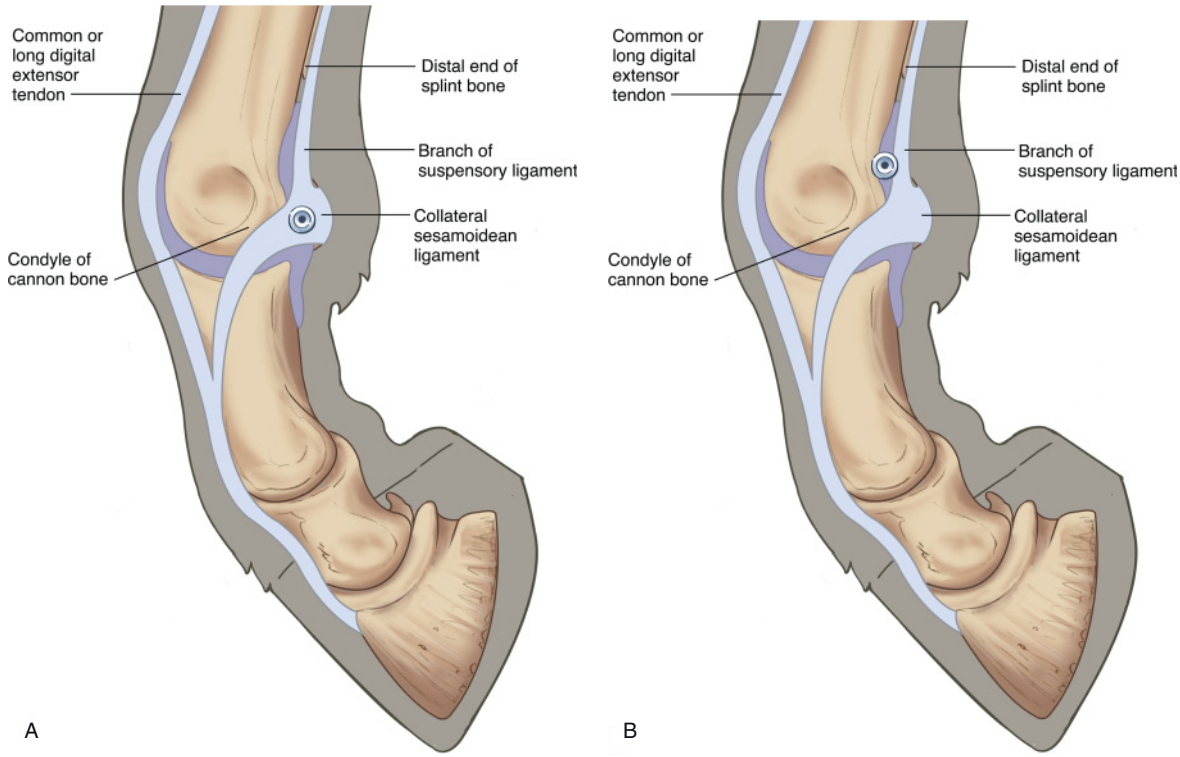


**Figure 3.80.** (A) Lateral view of the collateral sesamoidean ligament approach to the fetlock joint. (B) The needle also may be inserted just above the ligament in a depression made by the collateral sesamoidean ligament and the branch of the suspensory ligament when the limb is flexed.

## Middle Carpal Joint

**Quantity of Local Anesthetic:** 8 to 10 mL
**Needle Size:** 1 to 1-1/2 inches, 20 or 22 gauge
**Injection Techniques:**

- Dorsal approach (Figure 3.83A): The site of injection for the middle carpal joints is located in palpable depressions lateral or medial to the extensor carpi radialis tendon on the dorsal aspect of the carpus. The injection is made with a 1-inch, 20- or 22-gauge needle midway between the proximal and distal rows of the carpal bones. Because the surfaces of the carpal bones are at an angle, the needle should be directed slightly proximally to avoid hitting the articular cartilage.
- Palmarolateral approach (Figure 3.83B): The palmarolateral approach to the middle carpal joint is best used if the joint is distended. With distension the joint capsule is superficial and protrudes palmar and lateral to the ulnar and fourth carpal bones distal to the accessory carpal bone. The injection site is approximately 1 inch distal to the site of injection of the radiocarpal joint. A 1-inch 20-gauge needle is inserted perpendicular to the skin to a depth of about 1/2 inch.

**Pitfalls:**

1. Placing the needle too distally when using the palmar approach
2. Inability to palpate the palmar pouch of the middle carpal joint
3. Anesthetizing the proximal metacarpal region with a middle carpal joint block
4. Damaging the articular cartilage using the dorsal approach

## Radiocarpal Joint

**Quantity of Local Anesthetic:** 8 to 10 mL
**Needle Size:** 1 to 1-1/2 inches, 20 or 22 gauge
**Injection Techniques:**

- Dorsal approach (Figure 3.84A): The site of injection for the radiocarpal joint is located in palpable depressions lateral or medial to the extensor carpi radialis tendon on the dorsal

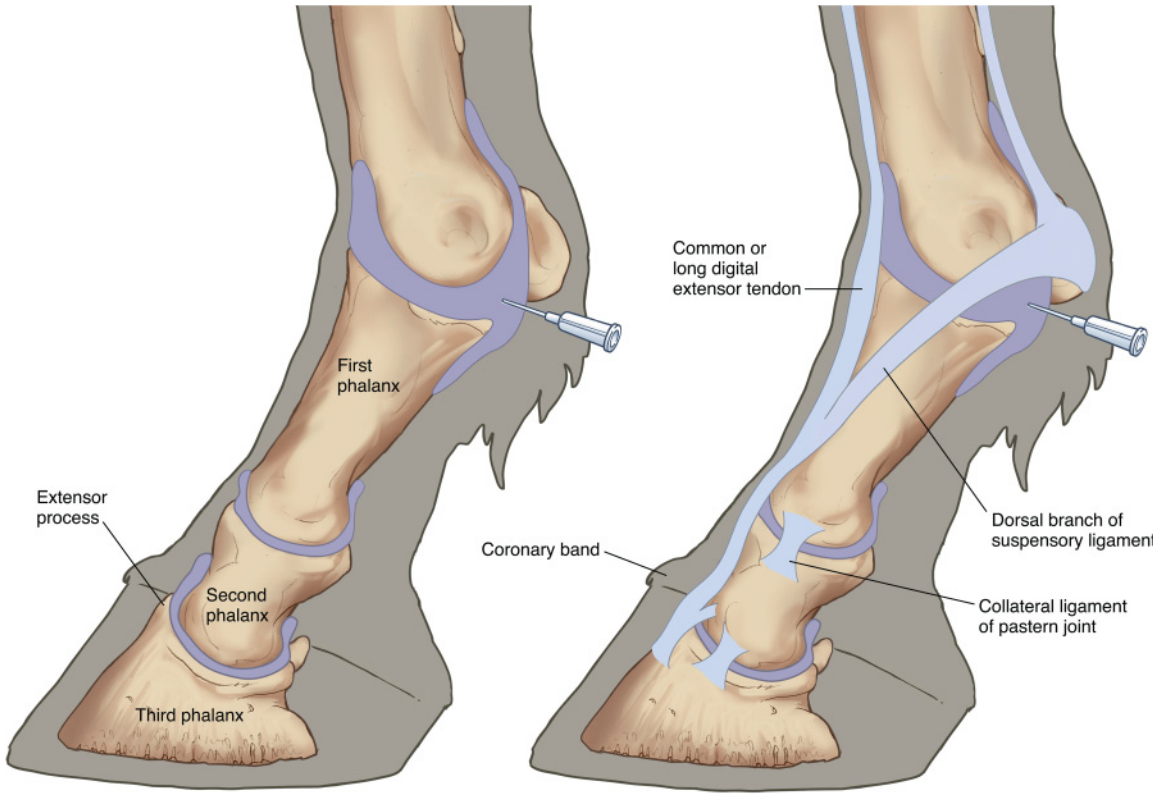


**Figure 3.81.** Distal palmar/plantar approach to the fetlock joint.

aspect of the carpus. The injection is made with a 1-inch, 20- or 22-gauge needle midway between the distal radius and proximal row of carpal bones.

- Palmarolateral approach (Figure 3.84B): The landmarks for the palmarolateral approach to the radiocarpal joint are the palmarolateral aspect of the radius, proximolateral aspect of the accessory carpal bone, and palmarolateral aspect of the ulnar carpal bone. A 1-inch, 20-gauge needle is inserted in this palpable depression at 90° to the long axis of the limb and the needle is directed dorsomedially. Another palmarolateral approach is at the midaccessory carpal bone level in a palpable "V" between the tendons of the ulnaris lateralis and the lateral digital extensor. The needle is inserted perpendicular to the skin in a small depression 1/2 to 1 inch distal to the "V" in the space between the distal lateral aspect of the radius (vestigal ulna) and the proximal lateral aspect of the ulnar carpal bone.

**Pitfalls:**

1. Placing the needle too distally when using the palmar approach
2. Inability to palpate the palmar pouch of the radiocarpal joint
3. Damaging the articular cartilage using the dorsal approach

## Elbow Joint

**Quantity of Local Anesthetic:** 20 to 30 mL
**Needle Size:** 1-1/2 to 3-1/2 inches, 20 gauge
**Injection Techniques:**

- Lateral approach (Figure 3.85): All approaches to the elbow are performed with the horse bearing weight on the limb. The landmark for the lateral approach is the lateral collateral ligament that extends across the joint from the lateral epicondyle of the humerus to the lateral tuberosity of the radius. The elbow joint can be entered either cranial or caudal to the collateral ligament. The site for injection is 2/3

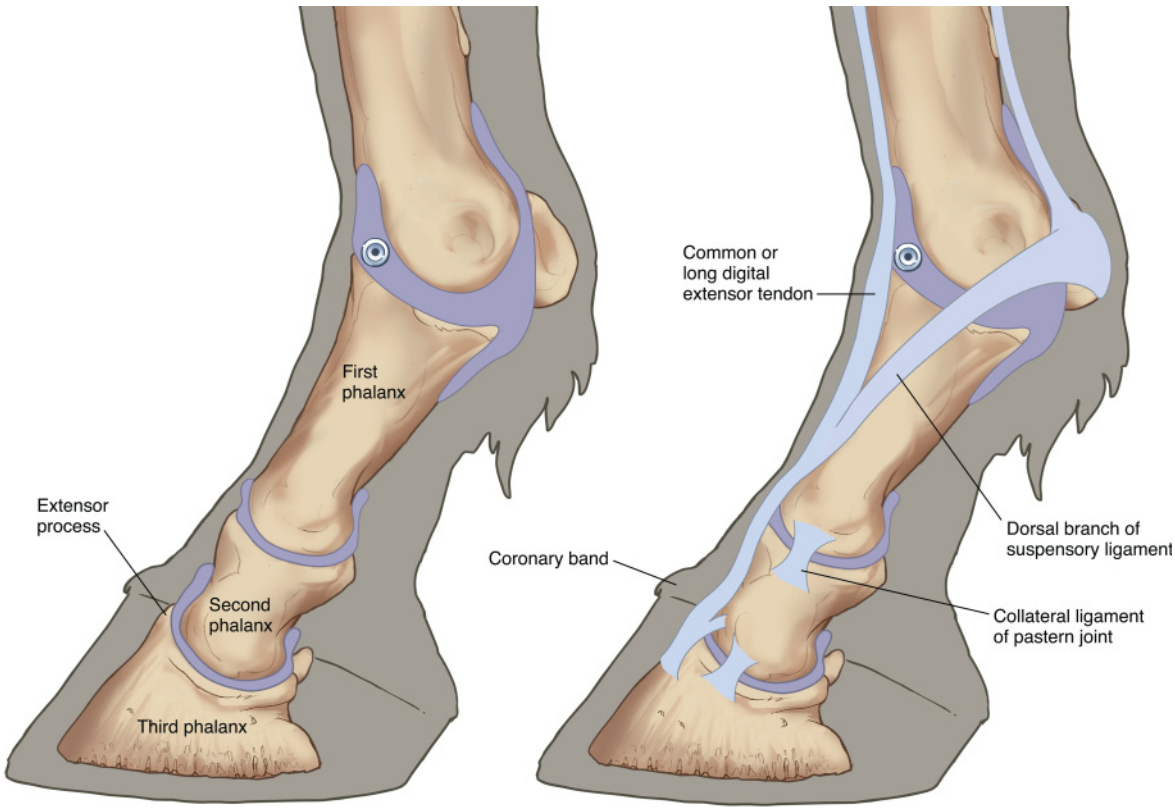


**Figure 3.82.** Dorsal injection site for the fetlock joint in the standing horse.

the distance distally measured from the lateral epicondyle of the humerus to the lateral tuberosity of the radius. A 1-1/2-inch, 20-gauge needle is inserted at a 90° angle to the skin just cranial or caudal to the lateral collateral ligament. If injected caudally, the needle may enter the bursa of the ulnaris lateralis muscle, which is thought to communicate with the elbow joint. However, communication between the bursa and the elbow joint occurred in only 9/24 (37.5%) of the joints examined.

- Caudolateral approach (Figure 3.86): The caudolateral approach is an alternative to placing the needle directly caudal to the collateral ligament using the lateral approach. The injection site is caudal to the palpable humeral epicondyle in the aconeal notch within the humero-ulna joint. This palpable V-shaped depression is usually just below the triceps muscles and 6 to 8 cm cranio-distal from the point of the olecranon process. A 1-1/2- to 3-1/2-inch, 20-gauge needle is inserted at a 45° angle to the skin and directed craniomedially.
- Caudal approach (Figure 3.87): The large caudal joint pouch of the elbow can be entered from a more proximal location. The landmarks are the lateral supracondylar crest of the distal humerus and the most proximal point of the olecranon process. The injection site is 1/2 inch proximal to and 1/3 of the distance measured caudally from the supracondylar eminence to the point of the olecranon. A 3-1/2-inch, 18- to 20-gauge spinal needle is directed distomedially through the triceps musculature at a 45° angle to the long axis of the limb into the olecranon fossa.

**Pitfalls:**

1. Difficulty in palpating the radial tuberosity or the lateral humeral epicondyle
2. Hitting bone when advancing the needle between the radius and humerus, and between the humerus and the ulna

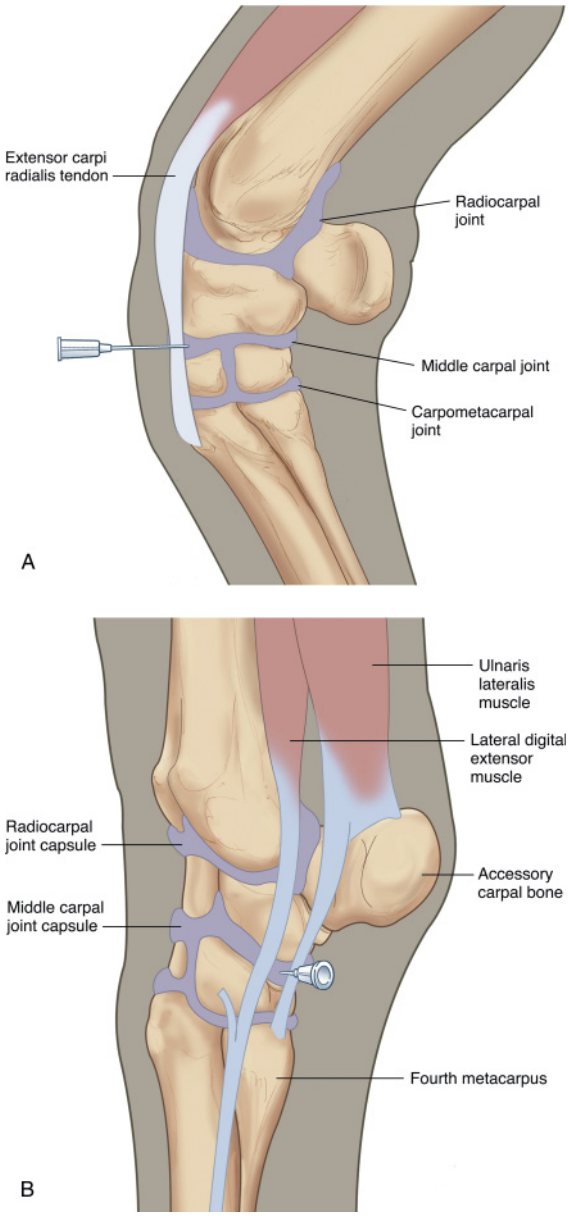


**Figure 3.83.** Dorsal flexed (A) and palmarolateral standing (B) approaches to the middle carpal joint.

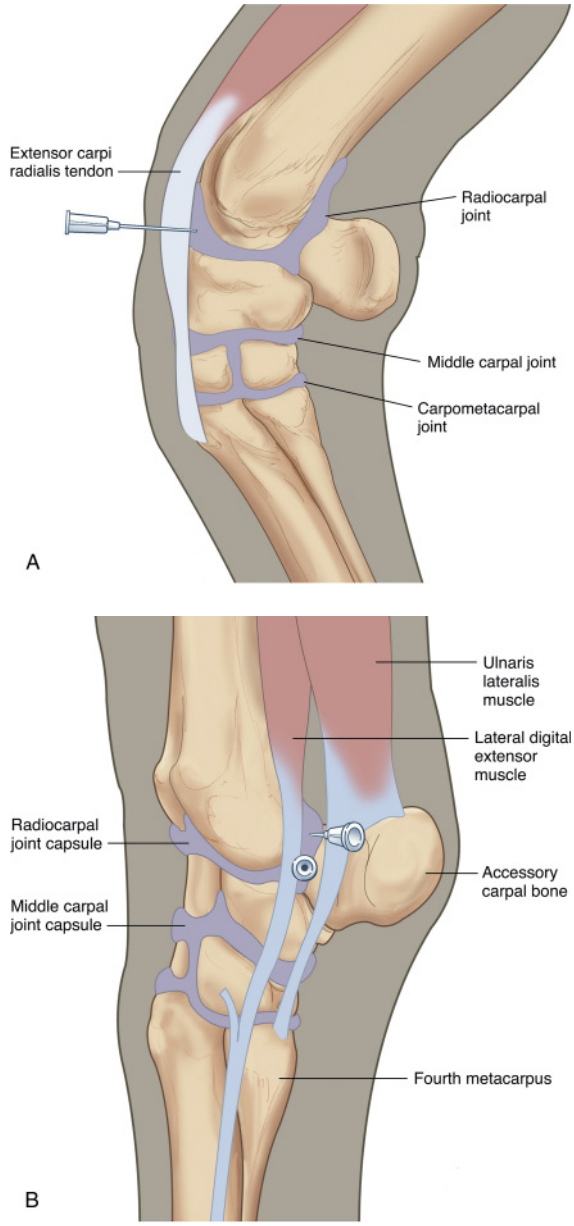


**Figure 3.84.** Dorsal flexed (A) and palmarolateral standing (B) approaches to the radiocarpal joint.

3. Radial nerve paralysis from injecting anesthetic outside the joint when placing the needle cranial to the collateral ligament
4. Inability to obtain synovial fluid

## Shoulder Joint

**Quantity of Local Anesthetic:** 30 to 40 mL
**Needle Size:** 3-1/2 inches, 18 to 20 gauge spinal needle

**Injection Techniques:**

- Craniolateral approach (Figure 3.88): All approaches to the shoulder are performed with the horse standing. The site for the craniolateral approach to the shoulder joint is located in the notch formed between the cranial and caudal prominences of the lateral tuberosity of the humerus. The caudal prominence (point of the shoulder) is easiest to

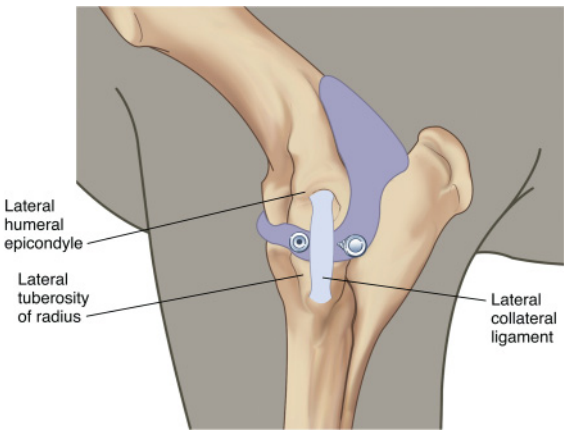


**Figure 3.85.** Lateral approaches cranial or caudal to the collateral ligament of the elbow joint.

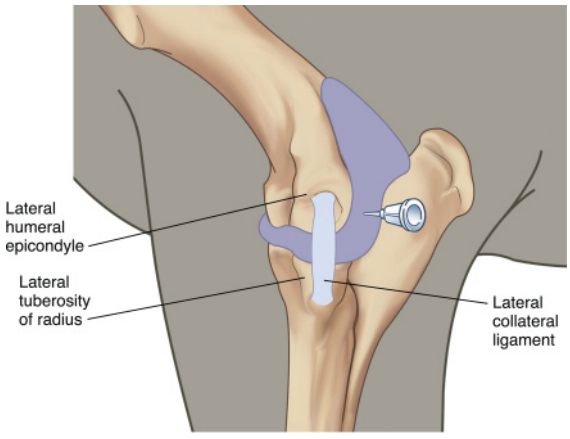


**Figure 3.86.** Caudolateral approach to the humeral-ulnar joint.

palpate; by exerting deep finger pressure the depression for needle insertion can be palpated 3 to 4 cm cranial to the caudal prominence. This notch is not as readily palpable in heavily muscled horses. A 3-1/2-inch, 18- 20-gauge spinal needle is inserted into this notch and directed parallel to the ground in a caudomedial direction toward the opposite elbow. The depth of penetration depends on the size of the horse, but the joint capsule is usually entered at a depth of 2 to 3 inches. Synovial fluid usually can be aspirated and is the only definitive method to document correct needle placement.

Alternatively, the spinal needle may be inserted slightly more proximal on the limb in a distinct depression located cranial to the infraspinatus tendon and slightly proximal

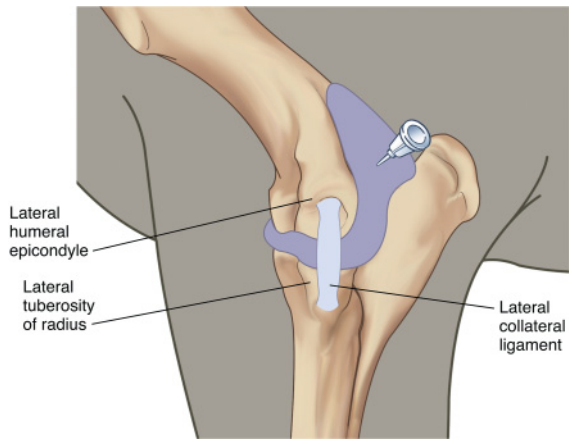


**Figure 3.87.** The approach to the large caudal outpouching of the elbow joint is 0.5 inch (1 cm) proximal to and one-third of the distance measured caudally from the supracondylar eminence to the point of the olecranon. A 3.5-inch (8.9-cm), 18- to 20-gauge spinal needle is directed distomedially through the triceps musculature at a 45° angle to the long axis of the limb into the olecranon fossa.

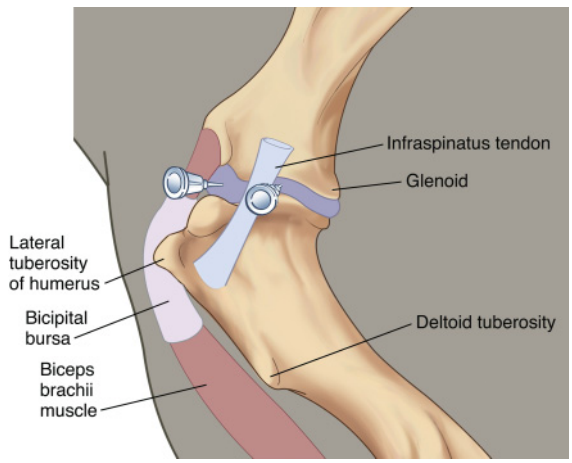


**Figure 3.88.** Craniolateral and lateral approaches to the shoulder joint.

and cranial to the point of the shoulder. The needle is placed parallel to the ground or slightly downward and directed caudomedially at a 45° angle until bone is contacted.

- Lateral approach: The landmarks for the lateral approach to the shoulder are the lateral humeral tuberosity and the infraspinatus tendon. A 3-1/2-inch, 18- to 20-gauge spinal needle is inserted 1 to 2 cm caudal and distal to the infraspinatus tendon in line with the lateral humeral tuberosity. The needle is

directed slightly caudally and upward toward the lateral aspect of the humeral head. In general, this approach is more difficult than the craniolateral approach.

**Pitfalls:**

1. Needle directed too proximal and hits the glenoid of scapula
2. Needle directed too medial to lateral and diverges across the humeral tuberosity
3. Inadvertent anesthesia of the bicipital bursa; communicates with the shoulder joint in some horses
4. Inability to aspirate synovial fluid

## Tarsometatarsal (TMT) Joint

**Quantity of Local Anesthetic:** 4 to 6 mL
**Needle Size:** 1 to 1-1/2 inches, 20-gauge
**Injection Technique:**

- Lateral approach (Figure 3.89): The landmarks for injection are the proximal head of the fourth metatarsal (MTIV) bone and the lateral edge of the SDFT. A 1- to 1-1/2-inch, 20-gauge needle is inserted in the small palpable depression just proximal to the head of MTIV. The needle is directed toward the dorsomedial aspect of the tarsus in a slightly downward direction to a depth of 1/2 to 1 inch. The block is nearly always performed with the horse standing.

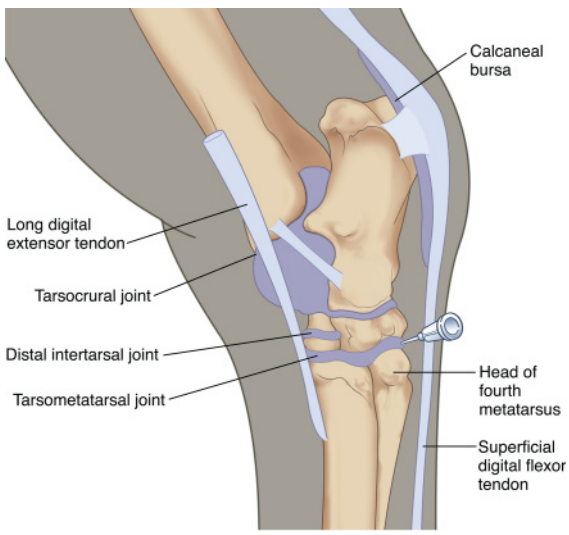


**Figure 3.89.** Lateral approach to the TMT joint.

**Pitfalls:**

1. Placing the needle too distally and hitting the head of the lateral splint bone
2. Directing the needle too medial to lateral or caudal to cranial
3. Anesthetizing the lateral plantar nerve that innervates the proximal suspensory
4. Anesthesia of the proximal suspensory region due to distention of TMT joint pouches.

## Distal Intertarsal (DIT) Joint

**Quantity of Local Anesthetic:** 3 to 5 mL
**Needle Size:** 5/8 to 1 inch, 22 to 25 gauge
**Injection Techniques:**

- Medial approach (Figure 3.90A): The medial approach to the DIT joint is used most often and is performed in the standing horse. The landmarks are midway between the plantar and dorsal aspect of the distal tarsus, just below the palpable distal border of the cunean tendon in a notch between the combined first and second tarsal bones and the third and the central tarsal bones. The needle is directed parallel to the ground and slightly caudally. Another approach is to identify the medial eminence of the talus and medial eminence of the central tarsal bone. The site for injection is halfway between these landmarks and 1/2 inch distal to the eminence of the central tarsal bone.
- Dorsolateral approach (Figure 3.90B): The DIT or centrodistal joint also can be entered using a dorsolateral approach. The injection site is 2 to 3 mm lateral to the long digital extensor tendon and 6 to 8 mm proximal to a line drawn perpendicular to the axis of the third metatarsal bone through the head of the fourth metatarsal bone. This is usually distal to the palpable lateral trochlear ridge of the talus. The needle is directed plantaromedially at an angle of approximately 70° from the sagittal plane until bone is contacted. This approach is safer for the clinician because it is performed on the lateral aspect of the tarsus, but is technically more difficult in the author's hands.

**Pitfalls:**

1. Inability to advance the needle—joint space is difficult to hit with medial approach
2. Excessive pressure when injecting—usually not within joint space
3. Injecting the proximal intertarsal joint by placing needle too high with the medial approach

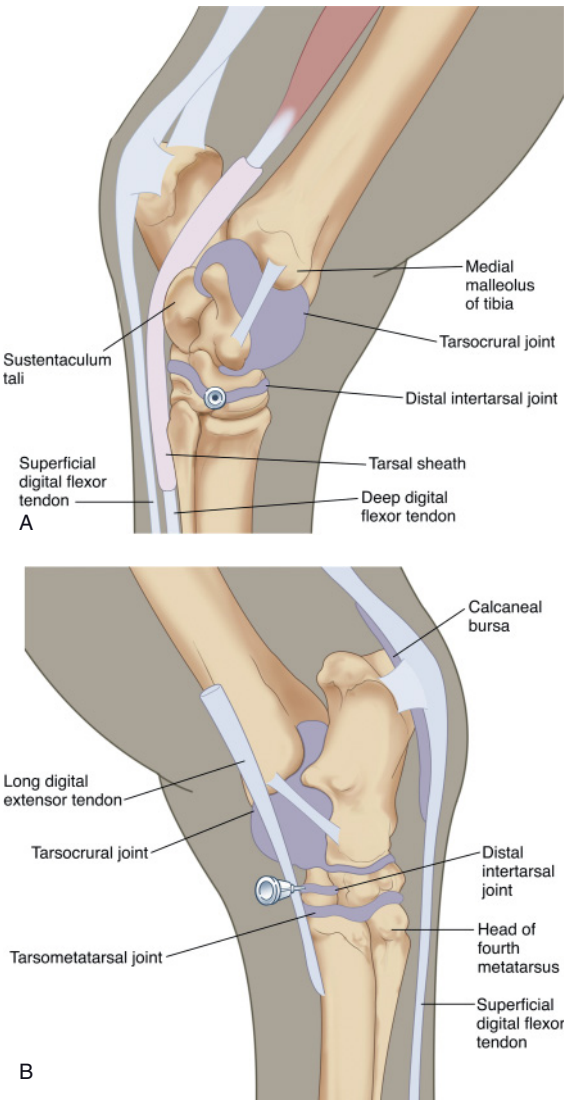


**Figure 3.90.** Medial (A) and dorsolateral (B) approaches to the DIT joint. The dorsolateral injection site is 2 to 3 mm lateral to the long digital extensor tendon and approximately 6 to 8 mm proximal to a line drawn perpendicular to the axis of MTIII through the head of MTIV. The needle is directed plantaromedially at an angle of approximately 70° from the sagittal plane until bone is contacted.

4. Placing needle too far caudally and missing the notch between the tarsal bones
5. Placing needle too proximal with the dorsolateral approach and entering the tarsocrural joint

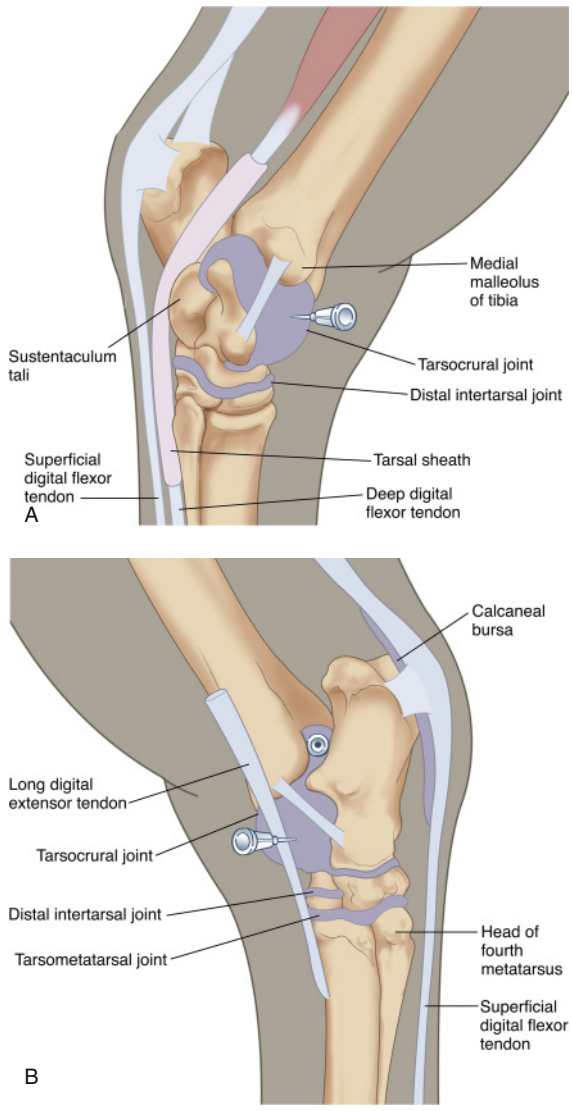


**Figure 3.91.** Dorsomedial (A), dorsolateral (B), and plantarolateral (B) approaches to the tarsocrural joint.

## Tarsocrural (TC) Joint

**Quantity of Local Anesthetic:** 15 to 20 mL
**Needle Size:** 1 to 1-1/2 inches, 20 to 22 gauge
**Injection Techniques:**

- Dorsomedial approach (Figure 3.91A): The joint may be entered dorsally (usually medially) or plantarly (usually laterally) depending on the clinical situation. For the dorsomedial approach, a 1- to 1-1/2-inch, 20-gauge needle is inserted 1 to 1-1/2 inches distal to the medial malleolus of the tibia, medial or lateral to the

cranial branch of the medial saphenous vein. The needle is advanced in a plantarolateral direction at approximately a 45° angle. The dorsomedial approach is usually performed in the weight-bearing limb from the opposite side of the horse, but can be performed from the same side of the horse.

- Plantar approaches (Figure 3.91B): The medial or lateral plantar outpouchings of the TC joint may be used for arthrocentesis, especially if significant synovial effusion is present. The palpable landmarks of the lateral plantar pouch are bordered by the tuber calcis caudally, the caudal aspect of the distal tibia cranially, and the proximal aspect of the lateral trochlear ridge of the talus distally. Confirmation that fluid swellings in this location are part of the TC joint can be determined by applying finger pressure to the swellings and feeling the dorsal pouches of the TC joint distend. A 1-inch, 20-gauge needle is inserted perpendicular to the skin at the site of the effusion with the limb bearing weight.

**Pitfalls:**

1. Inadvertent puncture of the saphenous vein with the dorsomedial approach
2. Difficulty in palpating the plantar pouches if minimal joint effusion is present

### Medial Femorotibial (MFT) Joint

**Quantity of Local Anesthetic:** 20 to 30 mL
**Needle Size:** 1-1/2 inches, 20 gauge
**Injection Technique:**

- Medial approach (Figure 3.92): The site for injection of the MFT joint is located in the space between the medial patellar and medial collateral ligaments just above the palpable proximomedial edge of the tibia in the weight-bearing limb. The needle is inserted just caudal to the medial patellar ligament, 1 cm proximal to the tibia, and directed perpendicular to the long axis of the limb. The needle may need to be repositioned slightly cranially or caudally to help obtain synovial fluid. This approach may be performed from the same side (facing the stifle) or from the opposite side, reaching under the horse's belly.
- Sartorius muscle approach: Another approach to the MFT joint is located 1/2 to 1 inch proximal to the medial tibial plateau in the depression between the medial patella ligament and the tendon of insertion of the sartorius muscle. The needle is directed in a cranial to caudal direction parallel to the ground and parallel to a plane that bisects the limb. The needle enters a medial outpouching of the MFT joint and avoids inadvertent penetration of the medial meniscus and the medial femoral condyle.

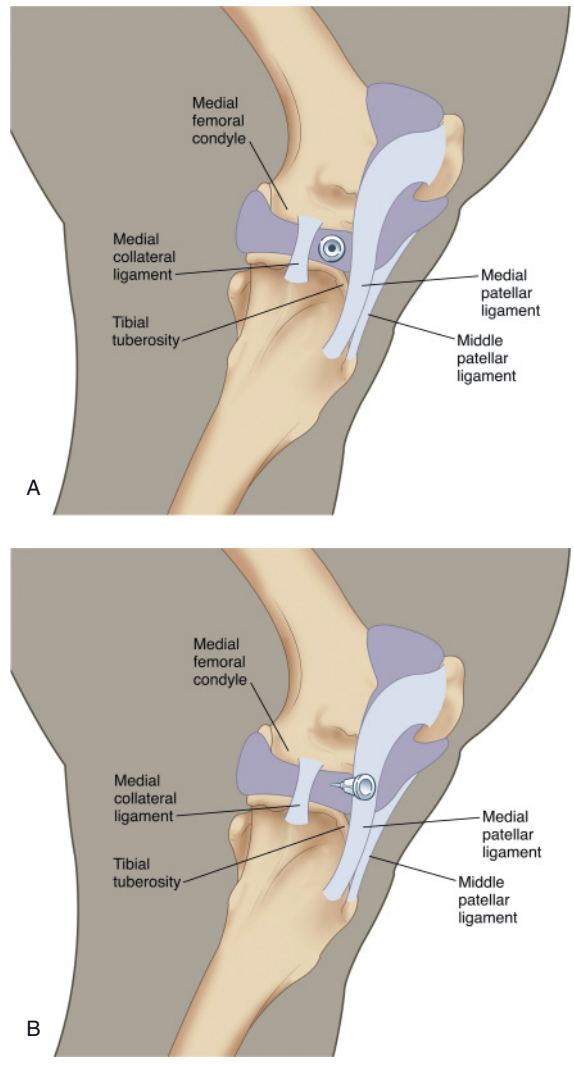


**Figure 3.92.** Medial approaches (A and B) to the MFT joint.

**Pitfalls:**

1. Hitting bone—needle inserted too low (tibia) or too high (medial condyle)
2. Inability to obtain synovial fluid—needle may be entering meniscus
3. Contacting the medial meniscus if the needle is inserted too far caudally or too close to the proximal tibia

4. Difficulty in finding the medial outpouching of the MFT joint using the sartorius muscle approach

## Femoropatellar (FP) Joint

**Quantity of Local Anesthetic:** 30 to 40 mL
**Needle Size:** 1-1/2 to 3-1/2 inches, 20 gauge
**Injection Technique:**

- Cranial approach (Figure 3.93A): In one cranial approach, a 3-1/2-inch, 20-gauge needle is inserted approximately 1 to 1-1/2 inches proximal to the tibial crest between the middle and medial patella ligaments, and is directed proximally under the patella. This approach is best performed with the limb in a partial weight-bearing (slightly flexed) position. Alternatively, the needle can be directed parallel to the ground with the limb fully bearing weight. The FP joint also can be entered just distal to the apex of the patella on either side of the middle patellar ligament with the limb bearing weight. The joint capsule is superficial at this location and a 1-1/2-inch, 20-gauge needle is directed at right angles to the skin.
- Lateral approach (Figure 3.93B): The lateral approach to the FP joint is performed with the horse bearing weight. The lateral cul-de-sac of the joint is located caudal to the lateral patellar ligament and approximately 2 inches proximal to the lateral tibial condyle. A 1-1/2-inch, 20-gauge needle is inserted into the recess perpendicular to the long axis of the femur until the nonarticular portion of the lateral trochlea is contacted. Synovial fluid can be retrieved in most cases and this approach is usually well tolerated by the horse.

**Pitfalls:**

1. Hitting bone—needle inserted too low (tibial crest) or too high (patella) with cranial approaches
2. Inability to obtain synovial fluid—needle may be within fat pad
3. Placing needle too proximally or distally with lateral approach

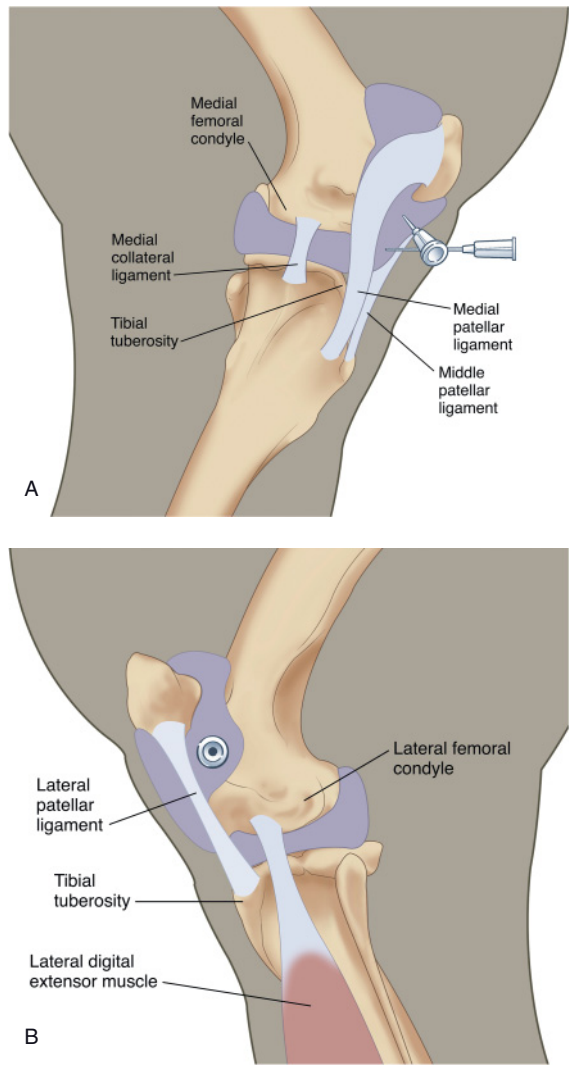


**Figure 3.93.** Cranial (A) and lateral (B) approaches to the femoropatellar joint.

## Lateral Femorotibial (LFT) Joint (Figure 3.94)

**Quantity of Local Anesthetic:** 20 to 30 mL
**Needle Size:** 1-1/2 inches, 20 gauge
**Injection Technique:** The site for injection of the LFT joint is slightly caudal to the palpable edge of the lateral patellar ligament just above the proximolateral edge of the tibia with the limb bearing weight. A 1-1/2-inch, 20-gauge needle is inserted at right angles to the long axis of the femur and directed from lateral to medial to a depth of 1 inch. An alternative approach is to insert the needle just proximal to the tibia in the space between the lateral collateral ligament of the LFT joint and the tendon of origin of the long digital extensor tendon. The palpable head of the fibula helps to identify these structures. The needle is inserted slowly to a depth of approximately 1 inch until the joint capsule is entered.

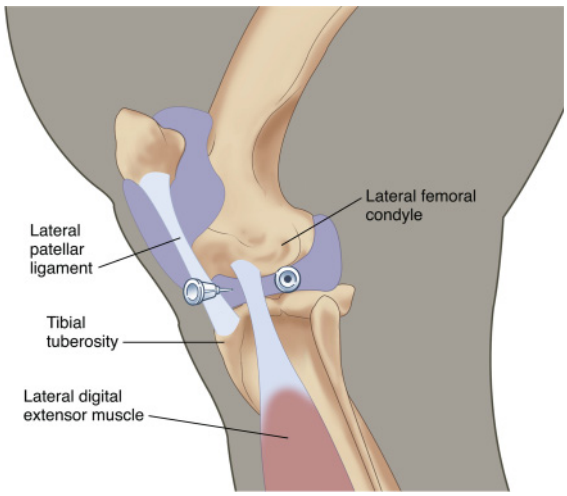


**Figure 3.94.** Injection sites for the lateral femorotibial joint just proximal to the tibia in the space between the lateral collateral ligament of the joint and the tendon of origin of the long digital extensor or just caudal to the lateral patellar ligament.

**Pitfalls:**

1. Hitting bone—needle inserted too low (tibial crest) or too high (lateral condyle)
2. Inability to obtain synovial fluid—needle may be within lateral meniscus

## Coxofemoral Joint

**Quantity of Local Anesthetic:** 30 to 50 mL
**Needle Size:** 6- to 8-inch, 16- to 18-gauge spinal needle
**Injection Techniques:** The lateral approach above the palpable greater trochanter is used most commonly to inject the coxofemoral joint (Figure 3.95). The most important landmarks to palpate are the paired summits of the greater trochanter of the femur. The trochanter is located about 2/3 the distance between the tuber coxae and the tuber ischii. The greater trochanter is approximately 4 inches wide with a notch between the cranial and caudal protuberances that can be difficult to palpate. The site for injection is just above the middle of the proximal summit of the trochanter. A small bleb of local anesthetic is injected subcutaneously over the injection site; a small stab incision may aid needle insertion. A 6- to 8-inch, 16- to 18-gauge spinal needle is directed in a horizontal plane perpendicular to the vertebral column. The needle should be directed slightly downward to stay close to the femoral neck so that it is approximately 1/2 inch lower than the insertion site after it has been advanced 3 to 4 inches. Alternatively, the needle can be positioned more proximally above the greater trochanter and directed downward at a steeper angle. Ultrasound can be helpful in directing the needle into the joint with either approach. The block is nearly always performed with the horse standing and usually restrained within stocks.

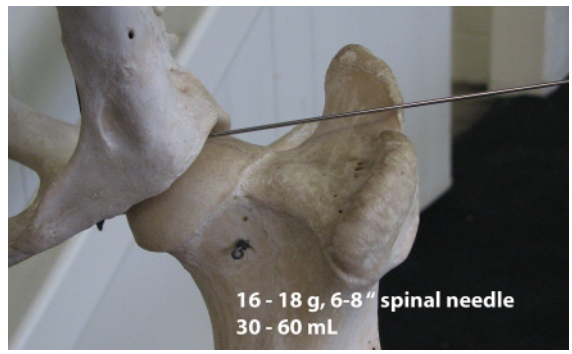


**Figure 3.95.** Lateral view of the injection site for the coxofemoral joint. The needle is inserted in the trochanteric notch and directed along the femoral neck until the joint is entered.

**Pitfalls:**

1. Inability to palpate the greater trochanter
2. Directing the needle too proximally and hitting the acetabulum above the joint
3. Inability to obtain synovial fluid
4. Inadvertent bending of the needle and not knowing where the tip of the needle is
5. Not contacting bone—needle usually directed too far cranially or caudally and not along the femoral neck

## Sacroiliac (SI) Joint

**Quantity of Local Anesthetic:** 7 to 10 mL
**Needle Size:** 8- to 10-inch, 15- to 18-gauge spinal needle
**Injection Techniques:** The horse is usually restrained in stocks, and the injection site anesthetized with local anesthesia. The landmark for injection is the cranial aspect of the tuber sacrale (Figure 3.96). A 10-inch, 15- to 16-gauge spinal needle is bent to an angle of about 40° in the direction of the needle's bevel. The needle is inserted through a stab incision in the skin 1 inch cranial to the contralateral tuber sacrale, and directed at a 60° angle to the vertical plane. The needle is advanced across the midline, aiming

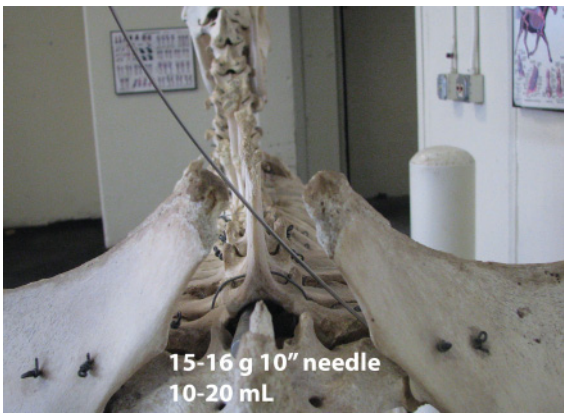


**Figure 3.96.** Needle location and angulation of the spinal needle used to inject the contralateral sacroiliac joint from the cranial aspect of the tuber sacrale. The needle is inserted through a stab incision in the skin 1 inch (2 cm) cranial to the contralateral tuber sacrale, and directed at a 60° angle to the vertical plane.

for a point midway between the ipsilateral tuber coxae and the greater trochanter of the femur until it contacts the medial aspect of the tuber sacrale. The needle hub is lifted and the needle is advanced at a steeper angle along the medial aspect of the ileal wing until it contacts the dorsal surface of the sacrum at a depth of approximately 6 to 8 inches. Approximately 8 to 10 mL of anesthetic can be used for diagnostic purposes, although inadvertent anesthesia of the sciatic nerve is a risk. Ultrasound-guided SI injections also may be performed, and both cranial and caudal approaches have been described.

**Pitfalls:**

1. Not contacting the sacrum—needle directed too far cranially or caudally
2. Bending of the needle during advancement—not knowing where the end of the needle is
3. Inadvertent anesthesia of the sciatic nerve—horse becomes ataxic or recumbent
4. Resentment by the horse—large needle penetrating substantial soft tissue around sacrum

## *Tendon Sheath Anesthesia*

### Digital Flexor Tendon Sheath (DFTS)

**Quantity of Local Anesthetic:** 10 to 15 mL
**Needle Size:** 1 to 1-1/2 inches, 20 to 22 gauge

**Injection Techniques:**

- Proximal approach (Figure 3.97): The site for injection of the proximal pouch of the DFTS is 1 cm proximal to the palmar/plantar annular ligament and 1 cm palmar/plantar to the lateral branch of the suspensory ligament. A 1- to 1-1/2-inch, 20-gauge needle is directed slightly distally until the sheath is penetrated.
- Distal approach (Figure 3.98): The distal outpouching of the DFTS in the pastern region is often palpable as a distinct "bubble" when effusion is present. It is located between the proximal and distal digital annular ligaments and between the diverging branches of the SDFT where the DDFT lies close to the skin. A 1-inch, 20-gauge needle is directed in a lateral to medial direction just beneath the skin so as not to penetrate the DDFT.
- Axial sesamoidean approach (Figure 3.99): The axial sesamoidean approach at the level of the fetlock and the medial or lateral approach between the annular ligament and proximal digital annular ligament can be performed in the distended and non-distended

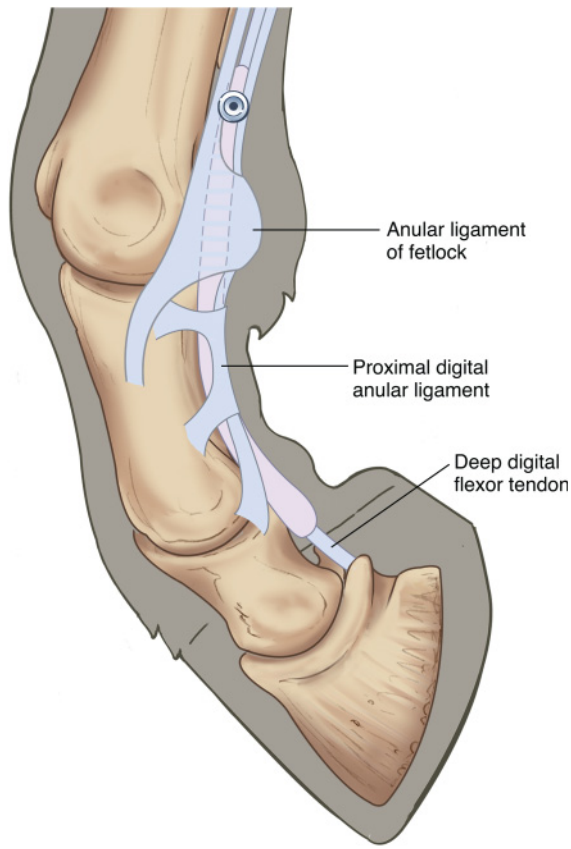


**Figure 3.97.** The proximal approach to the DFTS can be performed with the limb slightly flexed or with the limb weight bearing.

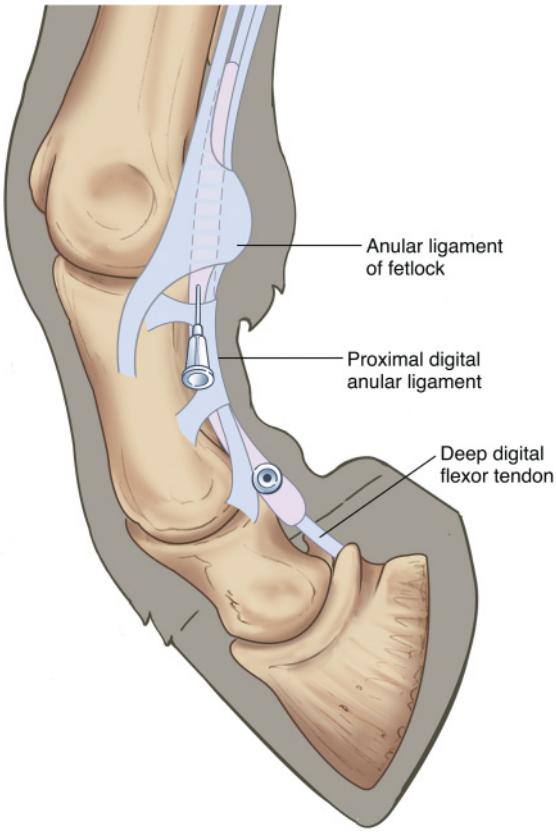


**Figure 3.98.** Distal approaches to the DFTS.

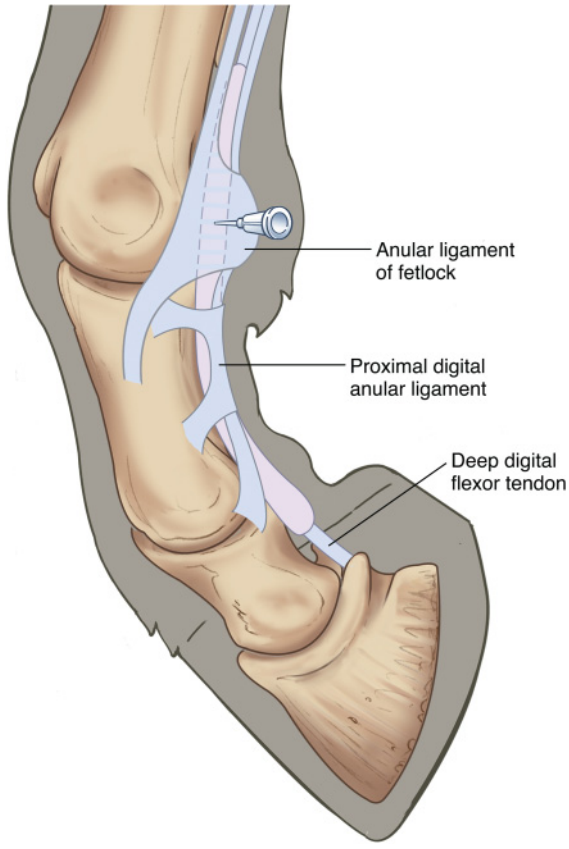


**Figure 3.99.** Injection site for the DFTS on the axial surface of the proximal sesamoid bone through the annular ligament.

DFTS. Both approaches are best performed with the limb held with the fetlock slightly flexed. The axial sesamoidean approach is performed 3 mm axial to the palpable border of the midbody of the lateral proximal sesamoid bone using a 1- to 1-1/2-inch, 20-gauge needle. The needle is directed at a 45° angle to the sagittal plane to a depth of approximately 1.5 to 2 cm. Alternatively, the needle can be inserted into the outpouching of the DFTS abaxial and distal to the sesamoid bones between the annular and proximal digital annular ligaments. The needle is inserted in a distal to proximal direction at approximately a 45° angle to the sagittal plane (Figure 3.98).

**Pitfalls:**

1. Difficulty in palpating the proximal pouch of the DFTS when non-distended
2. Inability to aspirate fluid—needle against tendons
3. Contacting bone with the axial sesamoidean approach—needle inserted too far abaxially

## Carpal Sheath

**Quantity of Local Anesthetic:** 15 to 30 mL
**Needle Size:** 1-1/2 to 3-1/2 inches, 20 to 22 gauge

**Injection Techniques:** The carpal synovial sheath enclosing the digital flexor tendons extends from a level 8 to 10 cm proximal to the radiocarpal joint distally to near the middle of the metacarpus on the medial aspect of the carpus. If distended, the sheath is easiest to access medially anywhere along its course using a 1-1/2-inch, 20-gauge needle. If non-distended, a lateral approach 3.5 cm proximal to the distal radial physis between the tendons of the lateral digital extensor and ulnaris lateralis muscles with the carpus flexed is recommended. A 3-1/2-inch, 20-gauge spinal needle is directed distomedially from this location to contact the caudal aspect of the radius.

**Pitfalls:**

1. Inability to aspirate fluid if no sheath effusion
2. Directing needle too superficially from lateral aspect of limb and not entering the carpal sheath

### Tarsal Sheath

**Quantity of Local Anesthetic:** 15 to 20 mL
**Needle Size:** 1 to 1-1/2 inches, 20 to 22 gauge
**Injection Techniques:** The tarsal sheath is located on the medial aspect of the tarsus and begins approximately 5 to 8 cm proximal to the medial malleolus and extends distally to the proximal 1/3 of the metatarsus. It encloses the DDFT of the hindlimb as it courses over the sustentaculum tali on the medial aspect of the tarsus. When distended, the tarsal sheath can be accessed for synoviocentesis anywhere along its course. The easiest location is usually either above or below the palpable sustentaculum tali on the medial aspect of the tarsus (Figure 3.100). A 1-1/2-inch, 20-gauge needle is directed proximolaterally or distolaterally at about a 45° angle with the long axis of the limb.

**Pitfall**

1. Inability to aspirate fluid if no sheath effusion

### Extensor Carpi Radialis (ECR) Tendon Sheath

**Quantity of Local Anesthetic:** 10 to 20 mL
**Needle Size:** 1 to 1-1/2 inches, 20 to 22 gauge
**Injection Technique:** The ECR tendon sheath extends from the mid-radius to the distal aspect of the carpus on the cranial aspect of the limb. It is very difficult to palpate unless there is synovial effusion present. When distended, it can be aspirated or injected anywhere along its length with a 1-1/2-inch, 20-gauge needle. Flexing the carpus will often make the ECR tendon sheath more distended, enabling easier access.

**Pitfalls:**

1. Inability to aspirate fluid if no sheath effusion
2. Blood contamination of fluid—not uncommon

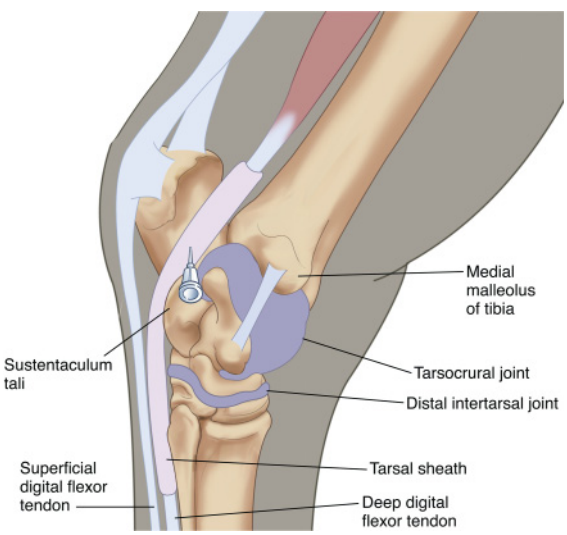


**Figure 3.100.** Injection site for the tarsal sheath.

## *Bursa Anesthesia*

### Navicular Bursa

**Quantity of Local Anesthetic:** 2 to 3 mL
**Needle Size:** 2-1/2 to 3-1/2 inches, 18–20 gauge
**Injection Techniques:** There are various techniques for needle entry into the navicular bursa, but the technique through the heel bulbs is thought to be most accurate (Figure 3.101). With this approach, a 3-1/2-inch, 20-gauge spinal needle is inserted between the heel bulbs just above the coronary band. The needle is advanced along a sagittal plane aiming for a point 1 cm below the coronary band, midway between the

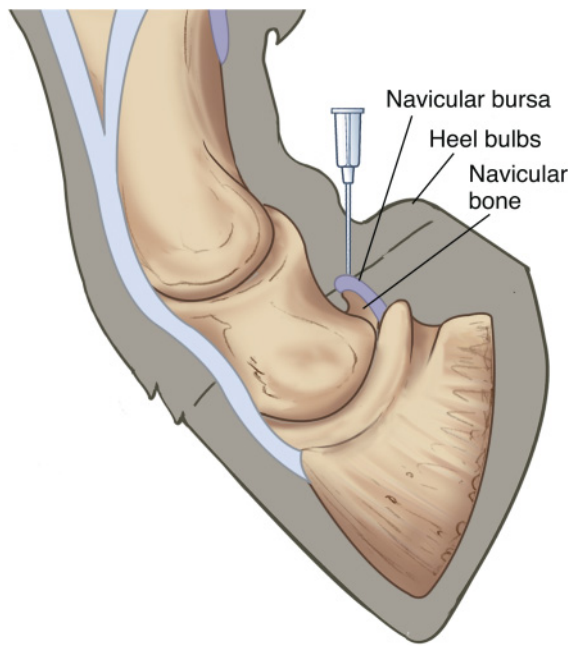


**Figure 3.101.** Lateral view of the foot demonstrating the correct angulation of the spinal needle to enter the navicular bursa using the palmar approach between the heel bulbs.

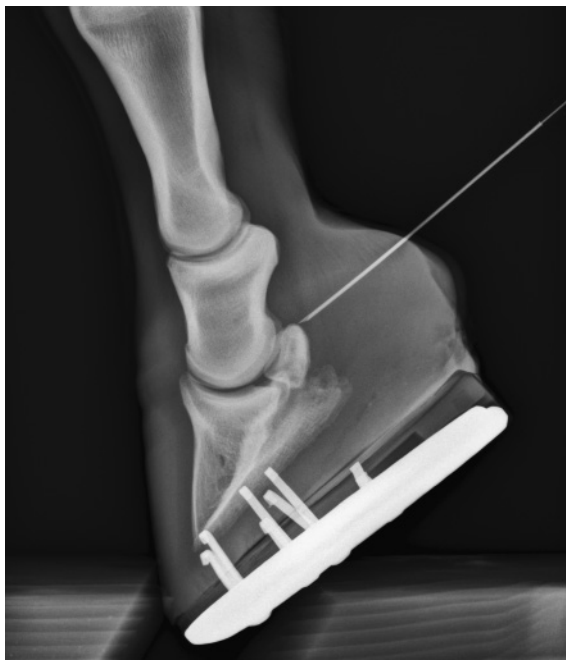

**Figure 3.102.** Lateral radiograph of the foot after needle placement to confirm the correct location of the needle palmar to the navicular bone.

toe and the heel. The needle is advanced until bone is contacted. Usually only 2 to 4 mL of anesthetic or medication can be injected, and flexing the lower limb will decrease the resistance to injection. A special wooden block for foot placement that unweights the heel and flexes the distal limb can facilitate the procedure. Radiographic or fluoroscopic documentation of the needle's location is recommended in most cases because it is easy to pass the needle over to the proximal border of the navicular bone into the DIP joint (Figure 3.102).

**Pitfalls:**

1. Needle directed too proximally and enters the DIP joint
2. Excessive pressure when injecting—unweight the limb or needle may be against the navicular bone
3. Anesthesia of palmar soft tissues of the foot due to extravasation of anesthetic

## Calcaneal Bursa

**Quantity of Local Anesthetic:** 10 to 15 mL
**Needle Size:** 1-1/2 inches, 20 gauge
**Injection Technique:** The calcaneal bursa is located between the SDFT and the caudal aspect

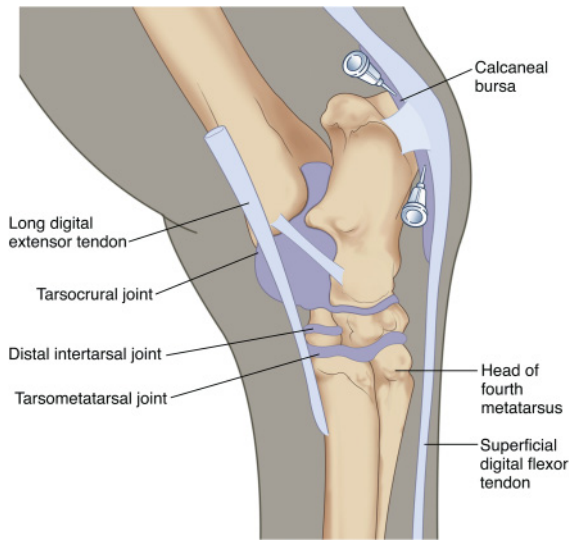


**Figure 3.103.** Injection sites for the calcaneal bursa are located either above or below the retinaculum of the SDFT. These injection sites can be difficult to find without effusion.

of the calcaneus. When distended, the bursa has synovial outpouchings medial and lateral to the tendon both proximal and distal to the SDFT retinaculum. These can often be seen as four distinct pockets of fluid surrounding the point of the hock in horses with bursal distention. Synovial aspiration is best performed using the lateral synovial outpouchings either above or below the SDFT retinaculum with the horse bearing weight (Figure 3.103). A 1-1/2-inch, 20-gauge needle is angled proximally or distally within these outpouchings to avoid the SDFT. The sites for needle placement are the same as those described for insertion of the arthroscope into the calcaneal bursa, 1 cm dorsal to the SDFT and 1 cm distal to the medial or lateral aspect of the SDFT retinaculum.

**Pitfalls:**

1. Needle contacts bone—needle directed too deep
2. Excessive pressure when injecting—needle against or within the SDFT
3. Inability to aspirate synovial fluid

## Bicipital Bursa

**Quantity of Local Anesthetic:** 20 to 30 mL
**Needle Size:** 1-1/2 to 3-1/2 inches, 18 to 20 gauge

**Injection Techniques:**

- Distal approach (Figure 3.104A): The cranial prominence of the lateral tuberosity of the humerus is used as the landmark as was done for the shoulder joint. The site of injection is 2-1/2 inches distal and 3 inches caudal to this prominence. A 3-1/2-inch, 18- to 20-gauge spinal needle is directed proximomedially toward the intertuberal groove until it contacts the humerus. The depth of the needle depends on the size of the horse, but a 3-1/-2-inch spinal needle usually is inserted to the hub in most mature horses. Alternatively, the deltoid tuberosity of the humerus can be palpated and used as a landmark. The needle is inserted 1-1/2 inches proximal to the distal aspect of the deltoid tuberosity and directed proximomedially (toward to opposite ear) to a depth of 2 to 3 inches.

  Proximal approach (Figure 3.104B): The proximal approach is performed in the intertuberal groove, which can be palpated medial to the edge of the cranial prominence of the lateral tuberosity of the humerus. A 1-1/2-inch, 20-gauge needle is inserted into the intertuberal groove in a plane parallel to the bearing surface of the foot at about a 45° angle to the sagittal axis of the horse until the needle strikes cartilage. The primary advantages of the proximal approach compared to the distal approach are a slightly improved accuracy of entering the bursa and not needing a 3-1/2-inch spinal needle.

  **Pitfalls:**

1. Needle directed too superficially and does not enter bursa
2. Inability to aspirate synovial fluid—not uncommon
3. Difficulty palpating the deltoid tuberosity to determine correct needle placement

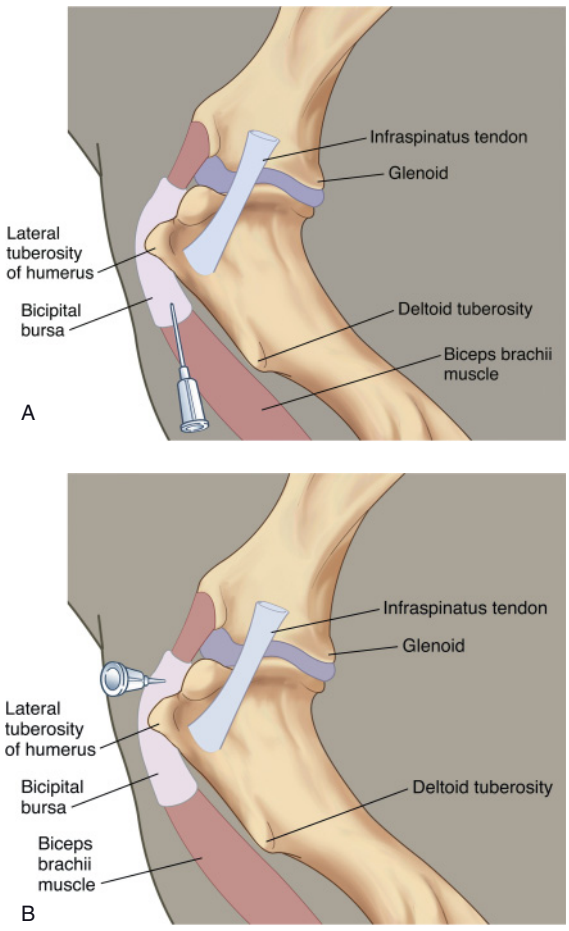


**Figure 3.104.** Distal (A) and proximal (B) approaches to the bicipital bursa from the lateral aspect of the limb in the standing horse.

## Trochanteric Bursa

**Quantity of Local Anesthetic:** 10 to 15 mL
**Needle Size:** 1 to 1-1/2 inches, 18 to 20 gauge
**Injection Techniques:** The trochanteric bursa is located beneath the tendon of insertion of the middle gluteal muscle on the cranial aspect of the greater trochanter of the femur. The site for injection is between the tendon and the lateral surface of the greater trochanter at the most cranial aspect of the palpable greater trochanter. A 1-1/2-inch, 18- to 20-gauge needle is usually all that is needed, although larger Warmblood horses may require a longer needle in some cases. The needle is inserted and directed horizontally at right angles to the sagittal plane until bone is encountered. An alternate method is to direct the needle medially through the middle gluteal muscle directly over the bursa toward the trochanter.

**Pitfalls:**

1. Needle directed too superficially and does not enter bursa
2. Inability to aspirate synovial fluid
3. Difficulty palpating the cranial border of the greater trochanter

## Cunean Bursa

**Quantity of Local Anesthetic:** 2 to 3 mL
**Needle Size:** 1 inch, 22 gauge
**Injection Techniques:** The cunean bursa is located on the medial surface of the distal tarsus

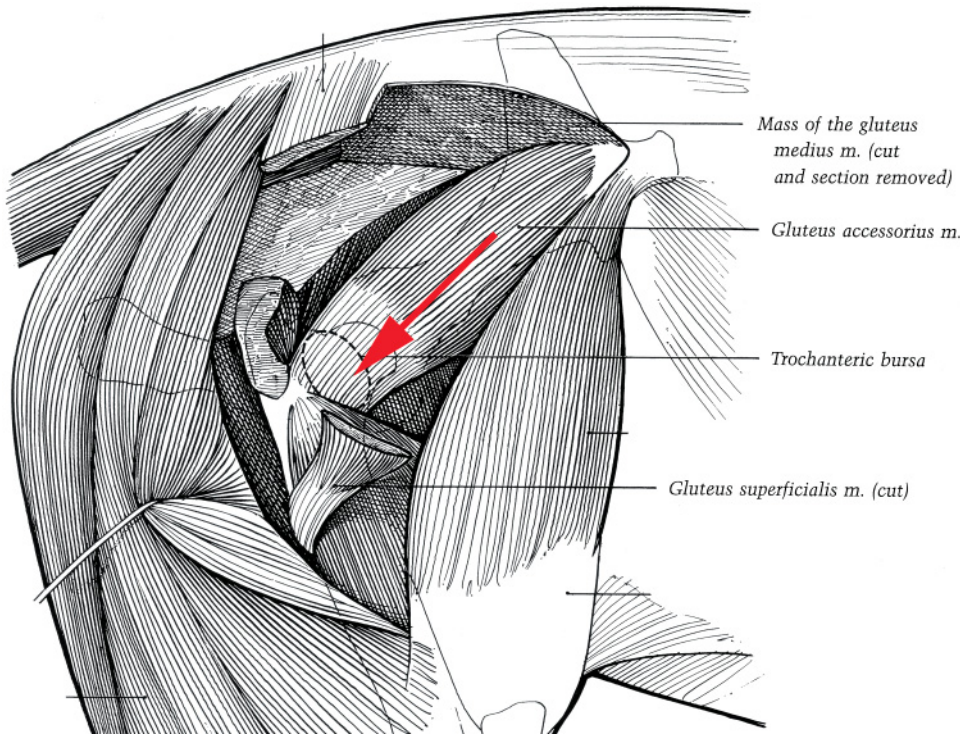


**Figure 3.105.** Injection site to enter the trochanteric bursa under the tendon of the middle gluteal muscle as it attaches to the greater trochanter.

between the medial collateral ligament of the tarsus and the medial branch of the tibialis cranialis (cunean) tendon. The bursa is relatively small and is not routinely anesthetized or treated alone because it often communicates with the DIT joint. A 1-inch, 22-gauge needle is inserted under the distal border of the cunean tendon and directed proximally to enter the bursa. Some clinicians will treat the cunean bursa concurrently when medicating the DIT joint in horses with distal tarsal OA. However, the cunean bursa is thought to communicate with the DIT joint in many horses.

**Pitfalls:**

1. Needle directed too superficially into the subcutaneous space
2. Rarely obtain synovial fluid
3. Inadvertent anesthesia of DIT joint because the cunean bursa and DIT joint can communicate

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**Revised from "Examination for Lameness" in *Adams and Stashak's Lameness in Horses, Sixth Edition,* by Gary M. Baxter and Ted S. Stashak.**

4

CHAPTER

# Imaging

## RADIOGRAPHY

During the past decade, digital radiography has replaced screen-film systems in most university veterinary teaching hospitals. This technology also has become commonplace in the private sector, from large referral hospitals to ambulatory practice. However, the conversion from conventional to digital radiography is currently in transition and the use of film-based radiography remains widely acceptable worldwide. Regardless of the radiographic system used, the X-ray machine and radiation safety practices remain exactly the same for either conventional or digital radiography.

### *Equipment*

Knowledge of radiography equipment, including X-ray machines, detector systems, film processing, image viewing devices, and accessory equipment, is necessary for safely obtaining good quality diagnostic radiographs. Knowing the advantages and disadvantages of the different equipment options that are available in the market for veterinary practice is especially necessary when deciding to upgrade from conventional to digital radiography.

#### X-Ray Machines

The basic control settings of an X-ray machine, the milliamperage (mA), exposure time, and kilovoltage potential (kVp), are located on the control panel and may be changed to vary the exposure. Milliamperage is the tube current and refers to the quantity of electrons flowing/second in the X-ray tube. Ultimately, it determines the quantity of X-rays emitted from the X-ray tube.

Exposure time is an important variable in equine radiology. Because of problems related to patient and detector movement, exposure time should be 0.1 second or less, if possible, for equine limb examinations. The use of electronic timers is recommended for accurate timing when the exposure is less than 0.1 second. For equine radiology, an electronic timer with a two-step exposure button is desirable. The first step warms the X-ray tube filament; the exposure is made in the second step. The two-step exposure button prolongs the X-ray tube life.

Because exposure time (in seconds) multiplied by the milliamperage equals milliamperage-seconds (mAs), radiation exposure is directly related to milliamperage-seconds:

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

0.1 second × 10 mA = 1 mAs
0.1 second × 15 mA = 1.5 mAs
0.2 second × 10 mA = 2 mAs

Kilovoltage determines the energy of X-rays being produced. Kilovoltage potential should range from 70 to 90 for equine limb radiographs on mature horses when a portable X-ray machine is used. A 10% increase or decrease in kilovoltage potential effectively doubles or halves the radiation exposure.

The focal spot-film distance (FFD) is the distance from the X-ray tube focal spot to the detector. The focal spot location usually is marked on the outside tube housing; if not, the approximate center of the tube housing can be used for its location. A constant FFD is necessary to minimize improper exposures, since the intensity of X-rays that expose the film is inversely proportional to the square of the FFD. A small difference in the FFD can, therefore, dramatically change the exposure on the X-ray film. For example, if the FFD were changed from 36 to 40 inches, the exposure from the X-ray machines would have to be increased by 23% to maintain a constant film exposure. In other words, if 10 mAs provided a good exposure at an FFD of 36 inches, 12.3 mAs would be required at 40 inches.

Some method of measuring the FFD before each exposure should be employed. Items such as a lightweight metal bar or a small rope can be used for fast FFD measurement. More elaborate measuring devices with converging light beams at the correctly set FFD also may be used. The FFD for equine limb radiography should be between 36 and 40 inches (85 and 100 cm) and no less than 24 inches (60 cm). When the FFD is less than 24 inches, the object is magnified and spatial resolution is reduced. FFDs greater than 40 inches can be used if exposure times are not excessively long.

Collimation is the process of limiting or restricting the primary X-ray beam to the appropriate size to cover the anatomical region of interest within the X-ray detector being used. Collimating the primary beam is a safety practice that must be monitored for each exposure and must be reset if the cassette size changes. Limiting the size of the primary X-ray beam is a major factor in reducing scatter radiation, which keeps radiation exposure to personnel as low as possible. Fixed cylinders or cones and adjustable light-beam collimators are available. The disadvantage of fixed primary beam restrictors is that they do not conform to different cassette sizes. Adjustable light-beam collimation is recommended for equine radiography.

Adjustable collimators can be affixed to most X-ray machines. They come with an internal light source, preferably 40 W or greater, so the limits of the primary X-ray beam are projected on the X-ray cassette/detector as visible light.

The type of X-ray machines best suited for equine practice depends on whether the practice is out-of-hospital or in-hospital. The features of the X-ray machines must be matched to fit different practice situations. Some compromised or trade-offs have to be made (e.g., less milliamperage or kilovoltage potential for more portability). An ideal X-ray machine for equine radiography has the following features:

- Easily and quietly movable, with a tube head that can extend to the floor or ground surface
- Adjustable milliamperage and kilovoltage potential setting
- An electronic, two-step timer capable of accurate exposure times of 0.1 second or faster
- Some form of tube head support so that handholding the X-ray tube during the exposure is not necessary
- Line voltage compensator and compensation meter
- Primary X-ray beam restrictor (the preferred system is an adjustable light-beam collimator)
- Free of radiation or electrical hazards.

The types of X-ray machines available for equine radiography are portable, mobile, and fixed (ceiling suspended). These machines differ in size and capacity from small, portable 15-mA machines to large, fixed 800-mA machines.

Portable X-ray machines are best suited for out-of-hospital locations. They are lightweight (30 to 55 lb) and can be easily stored for transport. Even though portable machines are lightweight and easily moved, for radiation safety purposes they should be used with a stand or other mechanical support system. Impediments for using X-ray tube stands include cost, incompatibility with the machines, and lack of field versatility. A two-legged stand system is the most adaptable to the variety of conditions encountered in the field. The maximum milliamperage on portable X-ray machines is between 10 and 40 mA, and the kilovoltage potential varies between 50 and 100 kVp. Milliamperage and kilovoltage potential settings on portable X-ray machines are usually interdependent (e.g., 10 mA at 80 kVp and 20 mA at 60 kVp). Multiple kilovoltage potential settings and multiple time set-

tings are desirable control settings on a portable X-ray machine. Time setting increments should be 50% or less below 0.5 second.

High-frequency transformers that reduce the exposure time are available for portable X-ray machines. They are equivalent to a three-phase, 12-pulse X-ray generator and provide a 40% to 50% increase in exposure compared with a full-wave rectified machine. The practical application is to reduce the exposure time and still obtain an adequate exposure for a diagnostic radiograph. A line voltage compensator also is desirable for portable X-ray machines. It is especially important in the field, where the line voltage may fluctuate with the simultaneous use of the electrical equipment, and it can compensate for a line voltage drop when a long electrical extension cord is used.

Mobile X-ray machines are best suited for in-hospital radiography. They can be moved easily and quietly, although they are not as easily moved as portable machines. Mobile X-ray machines generally have a milliamperage range between 100 and 200 mA. The X-ray tube should be moveable in the vertical plane to extend to the floor. Sometimes, mechanical modifications must be made to provide the necessary degree of X-ray tube travel. Timers on mobile X-ray machines usually have exposure times as fast as 1/60th or 1/120th of a second.

Fixed X-ray machines usually are suspended from the ceiling. They are limited to use within a single room. These machines can be coupled with large transformer systems that are capable of providing high milliamperage (800 to 1,000 mA). Ceiling-suspended tubes are easily movable and can be locked into position by magnetic locks while the X-ray exposure is made. The tube should be capable of extending to the floor. X-ray machines with high milliamperage capability have fast exposure times, eliminating most motion artifacts when radiographing equine limbs.

### Detectors, Film Processing, and Viewing Devices

The differences between digital and conventional radiography equipment are the detector used for image capture, post-capture image processing, and the image viewing devices. Conventional radiography uses screen-film technology in which the film, after being exposed to radiation, is processed in a dark room. The radiographs are then evaluated on a viewbox.

Digital radiography can be subdivided into computed radiography (CR) or direct digital radiography (DDR). CR uses a detector panel that stores the image and then uses a separate image readout unit. In DDR, the detector panel converts the X-rays into electrical charges by means of a direct read out process.

DDR systems can be further divided into direct or indirect conversion technologies, depending on the type of X-ray conversion used. Both CR and DDR systems convert the electronic signal to digital data and send it to an acquisition device. At this point, the digital data is considered "raw" and it can be further processed. The term "image processing" is ample and incorporates multiple steps in the acquisition of a digital image before the study is completed. During image processing, the raw image can be manipulated to obtain the best image possible for interpretation at the viewing station. One of the key factors that can be manipulated is the lookup table (LUT), which allows the operator to change the grayscale of the image by altering the actual pixel values. Once the study is completed, the images are sent to the viewing station where digital imaging and communication in medicine (DICOM®) viewing software is used for image display on a computer monitor.

## *Digital Radiography Systems*

### Computed Radiography

CR systems are similar to screen-film systems, in which a cassette stores the latent image until processing. The difference is the way the latent image is stored. As explained above, screen-film systems store the latent image in the radiographic film after the light from the intensifying screen strikes the silver crystals on the film. CR detectors eliminate the intensifying screens and replace the radiographic film with an imaging plate that is also protected in a cassette. This imaging plate has photostimulable phosphors (PSP) made of a mix of europium activated bromide, chlorine, or iodide (e.g., $BaFBr:Eu^{2}+$).

After the imaging plate is exposed to X-ray photons, the PSP changes to a higher energy state and stores the latent image. The PSP can store the latent image while remaining in that higher energy state for several hours, depending on the phosphor crystals used.

#### CR Reading Process

The data stored in the imaging plate PSP must be released to create a visible image. This process occurs when the cassette is placed in the reader unit (Figure 4.1). Once the cassette is inserted in


**Figure 4.1.** (A and B) Single- and multiple-plate computer radiography reading units. Used with permission from Agfa HealthCare Corporation, Copyright 2009. All rights reserved.

the reader unit, the imaging plate is automatically removed from the cassette and scanned by a laser beam. The laser light stimulates the energy trapped in the PSP and visible light is released from the plate. The released light strikes a photomultiplier tube and is converted into an electronic signal. The magnitude of the electronic signal represents the degree of X-ray attenuation of the structure that was imaged and is also assigned a corresponding shade of gray after conversion from analog to digital format. The data is then stored temporarily in a local hard disk. Subsequently, the imaging plate is exposed to bright white light to erase any residual trapped energy before it is returned to the cassette for reuse. The whole readout process for a 14-×-17-inch image plate takes approximately 30 to 40 seconds.

## Direct Digital Radiography

As mentioned above, direct digital radiography systems can be further divided into direct and indirect conversion. The main difference is that indirect technology converts the X-rays to light, which is then converted into an electrical charge. Direct technology converts X-rays immediately to an electrical charge with no light conversion during the process. Both systems have a direct read-out, meaning that the detector sends the signal straight to the computer instead of using a reading unit like the CR systems. The time lapse between exposure to image display takes less than 10 seconds, which increases the patient through-put and becomes an important factor to consider, especially in busy hospitals.

### Direct Conversion

A direct flat panel detector (Figure 4.2) is formed by different layers consisting of photoconductors as well as thin film transistors that contain rows and columns of individual detector elements with storage capacitors and read-out electronics. The photoconductors can be made of amorphous selenium (most commonly used), lead iodide, lead oxide, thallium bromide, and gadolinium compounds. The photoconductors are the first layer in contact with the X-ray photons that exit the patient and are responsible for converting them into electrical charges. The electrical charges are stored in capacitors and read row by row, and the information is sent to the computer after analog to digital conversion (Figure 4.3).


**Figure 4.2.** Direct flat panel detector (9 × 11 inch imaging area). Courtesy of Alejandro Valdés-Martínez.

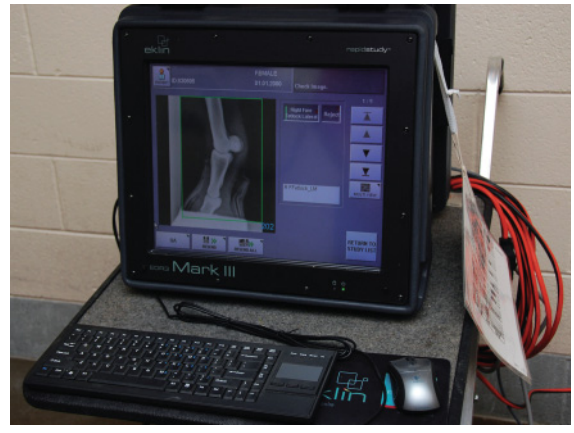



**Figure 4.3.** Mark III portable digital radiography system. Courtesy of Alejandro Valdés-Martínez.

### Indirect Conversion

Indirect conversion can be done with two technologies: indirect flat panel detector or charged coupled device (CCD). An indirect flat panel detector consists of a scintillator layer (usually made of cesium iodide crystals), an amorphous silicon photodiode circuit layer, and a thin film transistor array. Light is produced by

the scintillator after being irradiated by the X-rays exiting the patient. The light is absorbed and converted to electrical charges by the photodiodes. The read-out of the electrical charges is accomplished with the thin film transistor array in a similar way to that of the direct digital radiography systems.

A charged coupled device also is a light-sensitive unit for recording images. The X-rays from the patient are converted to light after striking the scintillator in a similar way to that of an intensifying screen in conventional screen-film systems. The light then is usually minified with a lens to fit in the small CCD, where it is recorded and converted into electrical charges. In the process of light minification, some of the light photons are lost and do not reach the CCD unit. This results in increased image noise compared with the flat panel detectors, in which minification is not applied.

## Digital Image Display

A computer used in a digital image viewing station consists of two basic parts: one or more monitors (hardware) and a DICOM® viewer (software) (Figure 4.4). A monitor used for digital radiography should have excellent resolution, high brightness (luminance), and wide dynamic range. The resolution of a monitor is dictated by the pixel and matrix size. The monitor's resolution improves as the pixel size decreases and the matrix size increases. Due to the high spatial resolution (ability to distinguish or separate two objects that are close to each other) required for evaluating digital radiographs, medical grade monitors with at least two to three megapixels are recommended. Monitor brightness or luminance is calculated in foot-lamberts (ft-L) or nit (candela/$m^2$) and should be at least 50 ft-L. The dynamic range of a monitor is the luminance ratio between the darkest (black) shade and the brightest (white) shade that the monitor can display. The dynamic range of a monitor usually is correlated to the luminance, thus the higher the monitor brightness, the higher the dynamic range.

A wide variety of Windows- or Macintosh-based DICOM® software programs are available and include free Internet downloadable versions to more sophisticated software that requires purchase or leasing contracts. The various DICOM® viewing software provides the user with the ability to further manipulate and improve the image displayed on the monitor. Features such as modifying contrast (window and level), image sharpening, edge enhancement, zoom, and measurement tools are included in most DICOM® viewers, but differ among vendors, depending on the quality of the software.

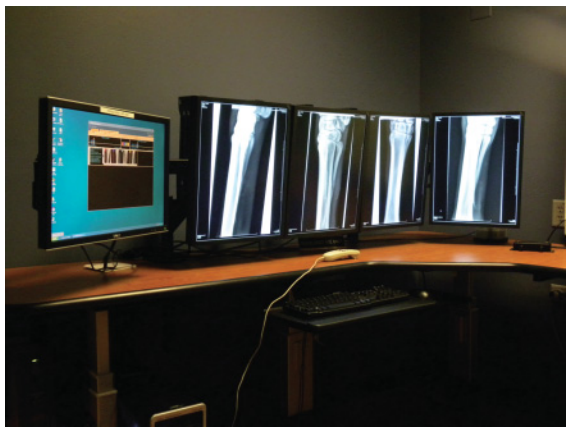


**Figure 4.4.** Digital radiography viewing station. A color monitor (far left) is used to access the PACS. Four black and white, 3MP medical grade monitors are used for display and evaluation of the radiographs. Courtesy of Alejandro Valdés-Martínez.

## Digital Image Storage, HIS/RIS, PACS, and DICOM®

The advantages of replacing films with digital radiographic images include a smaller physical storage area (magnetic tape, CD, DVD, hard drives, USB flash drives, etc.), which consequently eases the organization and provides quick retrieval of previous exams. The digital image quality does not degrade with time, as is the case with conventional radiographs. In addition, the ability to refer a study for a second opinion is literally only an e-mail away. The practice of teleradiology is becoming more popular among veterinary radiologists, both in private practice and academia, as a service to referring veterinarians.

Most university-based veterinary teaching hospitals and some large private referral hospitals have a hospital or radiology information system (HIS or RIS), a picture archive and communication system (PACS), and DICOM® viewing software. The combination of these three technologies tremendously improves the daily work flow of busy operations. Every step in the process of obtaining a diagnostic imaging study, from the time the patient enters the hospital to the time it exits, is directed with a HIS or RIS, PACS, and a DICOM® image viewer.

The use of this technology not only improves the efficiency of a radiology unit by increasing patient through-put, providing immediate access to digital images, and retrieving imaging reports electronically; it also practically eliminates errors such as wrong patient information and losing/mixing studies.

### *Radiation Safety*

Radiation safety becomes even more important as the use of radiology increases in equine practice. Safe radiation procedures often are not followed because of insufficient knowledge of biologic radiation effects, lack of awareness of radiation safety principles, inadequate radiation safety equipment, and/or neglect of radiation safety practices because these practices require too much time and effort. None of these reasons justifies the unsafe use of radiation.

Veterinarians in equine practice may receive low doses of radiation over long periods of time. Because the clinician's extremities (hands, eyes, and feet) are the most common body parts exposed to radiation, they are subject to chronic radiation injury. Chronic radiation injury may manifest as skin ridge flattening on fingertips, ridging of fingernails, or, in severe cases, skin carcinomas. Such changes may result from not routinely following safe radiation procedures.

The general principles of radiation safety are:

1. Keeping personnel as far away from the radiation source (X-ray tube) as possible
2. Using protective barriers
3. Reducing X-ray exposure factors
4. Using a radiation monitoring system

Keeping personnel as far away from the X-ray source as possible can be accomplished by having nonessential personnel leave the immediate area, using cassette holders and positioning blocks, providing tranquilization or general anesthesia when needed, using an X-ray tube stand or support, and having a 1- to 2-meter-long cord connected to the exposure button. No part of the body should be exposed to the primary X-ray beam.

Protective barriers should always be used. Walls and lead screens are good protective barriers to use when practical. Adequate wall thickness in a new or remodeled facility should be determined by consulting with a health physicist. The personnel subject to the greatest exposure when performing equine examinations are those holding the horse's limb, the detector holder, and those holding the halter. Therefore, personnel who must be near the animal when the X-ray examination is performed should wear lead aprons and gloves. Aprons and gloves should be radiographed periodically to check for cracks and holes in the lead-impregnated lining.

Fast film-screen combinations and a decreased FFD can be used to reduce X-ray exposure factors. As mentioned, for good-quality films, the FFD should not be less than 24 inches and preferably not more than 36 inches.

A radiation-monitoring system should be used by all radiology personnel. These systems not only provide safety guidance but also protect against possible legal implications. Film-badge monitoring systems and service can be purchased from commercial sources.

The equipment necessary for safely operating an X-ray machine includes detector holders, lead aprons and gloves, aluminum filters, and an adjustable light-beam collimator. Detector holders eliminate the need to handhold detectors, increasing the distance between hands and the X-ray beam (Figure 4.5). The detector holder should be durable and lightweight.

Lead aprons and gloves should be worn by everyone assisting with the X-ray examination. Protective gear should have at least 0.5-mm lead equivalent. Lead aprons and gloves provide adequate protection from secondary and scattered radiation but not from primary radiation. The life of lead aprons and gloves can be prolonged by hanging them up when they are not in use, which prevents cracks or holes forming from improper care.

Primary beam filtration should be at least 2.5-mm aluminum. The filtration should be added at the X-ray tube port. Filtration makes the X-ray beam more energetic (hardens) and reduces the

**Figure 4.5.** Direct flat panel detector with handle attached. Courtesy of Alejandro Valdés-Martínez.

amount of less energetic (soft) radiation, which decreases the amount of scattered radiation to surrounding personnel.

An X-ray-beam-limiting device or collimator is one of the important, yet overlooked, pieces of radiation safety equipment. The size of the primary X-ray beam is a major factor in determining radiation dose to the hands. Several beam-limiting devices are available. Fixed-size cones and cylinders and adjustable light-beam collimators limit the primary X-ray beam. Adjustable light-beam collimators have the advantage of limiting the primary X-ray beam to the exact cassette size, regardless of the FFD. The light also assists alignment of the primary X-ray beam with the cassette. Every X-ray machine should have a primary beam-limiting device. An adjustable light-beam collimator is highly recommended.

Recent studies in human radiography have shown that some digital radiography systems may be capable of reducing the exposure settings with minimally compromising the diagnostic value of the radiograph. However, the reduction of the exposure is limited to some extent and depends on the area of examination and the clinical indication. Radiation exposure reduction to both the patient and the personnel can be simply achieved by minimizing the number of nondiagnostic radiographs due to technique factors. This is most feasible in digital radiography as a result of the wider dynamic range of these systems.

In summary, the following safety measures should be observed:

- Never handhold the X-ray detector during an exposure. Detector holders or general anesthesia should be used.
- All personnel not needed for assistance with the X-ray examination should leave the immediate area.
- All individuals assisting with the X-ray examination should wear lead gloves and aprons.
- Use a primary X-ray-beam-restricting device, preferably an adjustable light-beam collimator.
- Use primary beam filtration equivalent to 2.5-mm aluminum.
- If possible, rotate personnel responsible for holding the detector; avoid routinely using the same person for this job.
- Do not allow anyone under 18 years of age or anyone who is pregnant to assist with an X-ray examination.
- Use consistent X-ray exposures and darkroom techniques. Repeat exposures require unnecessary radiation exposure to personnel.
- Use a radiation-monitoring system.

The veterinarian in charge is responsible for the radiation safety practices used by his/her employees. Providing necessary radiation safety equipment and following these rules should keep exposure levels below the limits recommended by the National Council on Radiation Protection.

### *Principles of Radiographic Interpretation*

There are three basic steps to radiographic interpretation:

1. Evaluating the film and quality of the examination
2. Reading the radiograph
3. Formulating a radiographic impression, diagnosis, and/or prognosis.

Film quality should be evaluated by checking film exposure, labeling, collimation, and positioning. This is an important step because poor-quality radiographs result in missed or improperly diagnosed conditions. A properly exposed radiograph should have enough film contrast latitude to allow observation of bone and soft tissue outlines, and the film detail should be sufficient to demonstrate bone trabeculae. The exposure becomes a less critical factor when using digital radiography systems due to the greater contrast latitude. The person evaluating the study should be familiar with artifacts that may originate from the patient or be particular to the system used.

Positioning should be evaluated by inspecting joint space and bone alignment. Poorly positioned studies may result from the horse standing with the limb not perpendicular to the ground, the cassette not parallel with the limb, or the X-ray tube not perpendicular to the X-ray cassette or the part being examined.

A thorough radiographic examination should be done on each part for which pathology is expected from the physical examination. The routine examination may consist of two to eight views, depending on the part examined. Sometimes additional views are needed to better define and demonstrate suspected lesions.

The second step in radiographic interpretation is reading the radiograph. If the clinician is in a hurry to make a diagnosis, this step may be overlooked or cut short, resulting in interpretational errors. A systematic, thorough inspection of the entire film should be done so that nothing is missed. Identifying radiographic abnormalities requires knowledge of both radiographic anatomy and radiographic signs of disease. Without knowledge of either, a correct radiographic interpretation usually is not made.

The third step is formulating a radiographic impression, diagnosis, or differential diagnosis. Knowledge of disease pathophysiology and its relationship to radiographic signs is necessary for this step. Finally, the radiographic diagnosis should be integrated with other diagnostic information, such as history, physical examinations, and perineural/intrasynovial anesthesia results, to arrive at a final diagnosis.

## Radiology of Soft Tissue Structures

Soft tissue changes may be primary pathologic changes, secondary to more serious bone changes, or an incidental finding of no clinical significance. A bright light is helpful for evaluating soft tissue structures when using conventional (screen-film) radiographic systems. Fascial planes, tendons, ligaments, and some portion of joint capsules may be seen because of adipose tissue (fat) within and around these structures. Fat is less opaque and appears slightly darker than muscle, skin, tendons, or ligaments on a radiograph (Figure 4.6A). Soft tissue structures should be evaluated for thickening, mineralization, and free gas (radiolucencies).

### Soft Tissue Thickening

Soft tissue thickening in the equine extremity is usually caused by swelling secondary to inflammation from infection or trauma. However, nonclinically significant soft tissue thickening can also be identified radiographically, such as in cases of elbow or carpal hygroma where the thickening originates from chronic trauma but is simply a cosmetic blemish. The soft tissue thickening may be localized or diffuse. Localized thickening may be identified radiographically within or around joints, tendons, or muscles (Figure 4.6B). Radiographic signs of soft tissue thickening include an increased soft tissue prominence, displacement of fat bodies (adipose tissue) around the joint capsule or tendon sheaths, and mottling or obliteration of adipose tissue in fascial planes around muscles, joint capsules, or tendons.

### Mineralization

Soft tissue mineralization in equine limbs may be dystrophic or metastatic. Dystrophic mineralization is most frequent and is present in damaged tissues after physical, chemical, or thermal trauma (Figure 4.7). Hematomas, necrotic areas, and post-inflammatory foci and cartilaginous

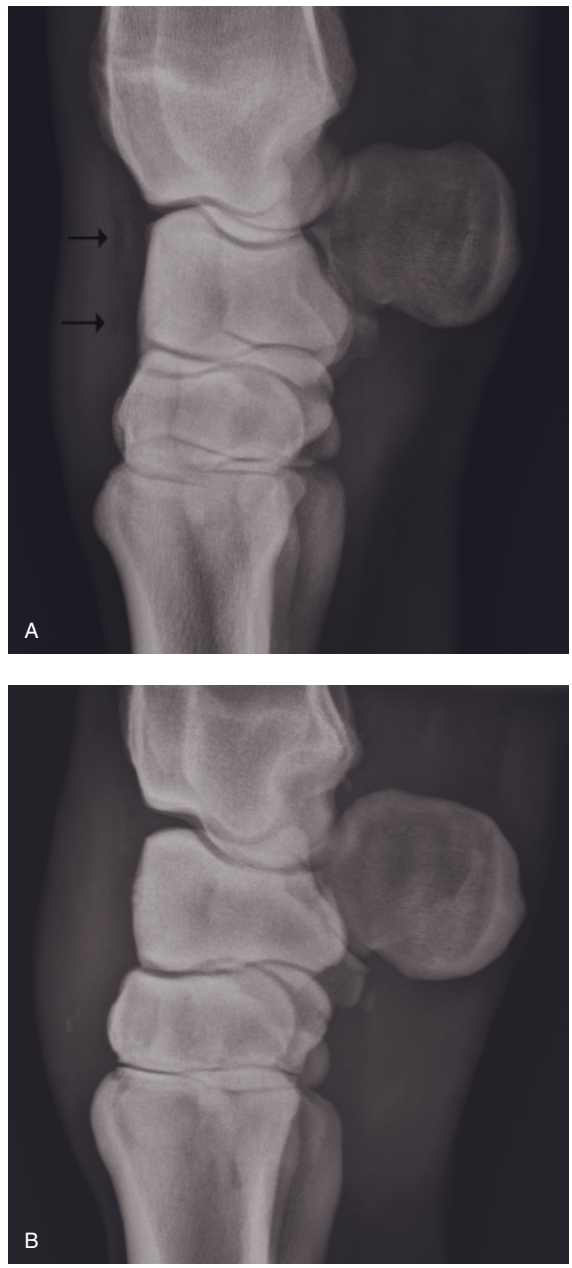



**Figure 4.6.** (A) Lateromedial (LM) projection of a normal carpus showing the fat pads (adipose tissue bodies) as lucent structures within the dorsal soft tissues (arrows). (B) Lateromedial (LM) projection of carpal effusion. Note the soft tissue thickening on the dorsal aspect of the carpus obliterating the normal fat pads seen in (A). Courtesy of Alejandro Valdés-Martínez.

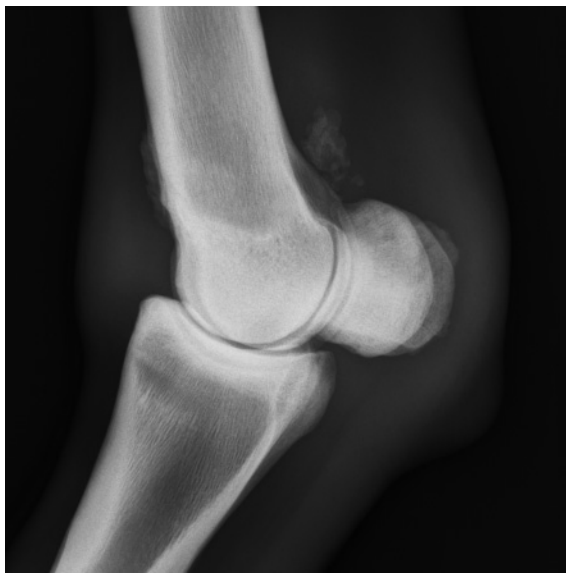


**Figure 4.7.** Lateromedial (LM) projection of the metacarpophalangeal joint. Amorphous soft tissue mineralization is present just palmar to the distal MCIII and proximal to the sesamoid bones, consistent with dystrophic mineralization of the palmar joint capsule. Courtesy of New Bolton Center, School of Veterinary Medicine, University of Pennsylvania.

areas are frequent sites of dystrophic mineralization. Calcinosis circumscripta is a form of dystrophic mineralization and is most frequently seen periarticular in the horse. Metastatic mineralization primarily occurs in normal soft tissue from a disturbance in calcium and phosphorus metabolism but is seldom observed in the horse.

Radiographic signs of soft tissue mineralization include an amorphous radiopacity within soft tissue structures, absence of trabecular or cortical bone within the radiopacity, indistinct borders with dystrophic mineralization, and well-defined and distinct borders. A round "cauliflower-shaped" appearance is usually present with calcinosis circumpscripta.

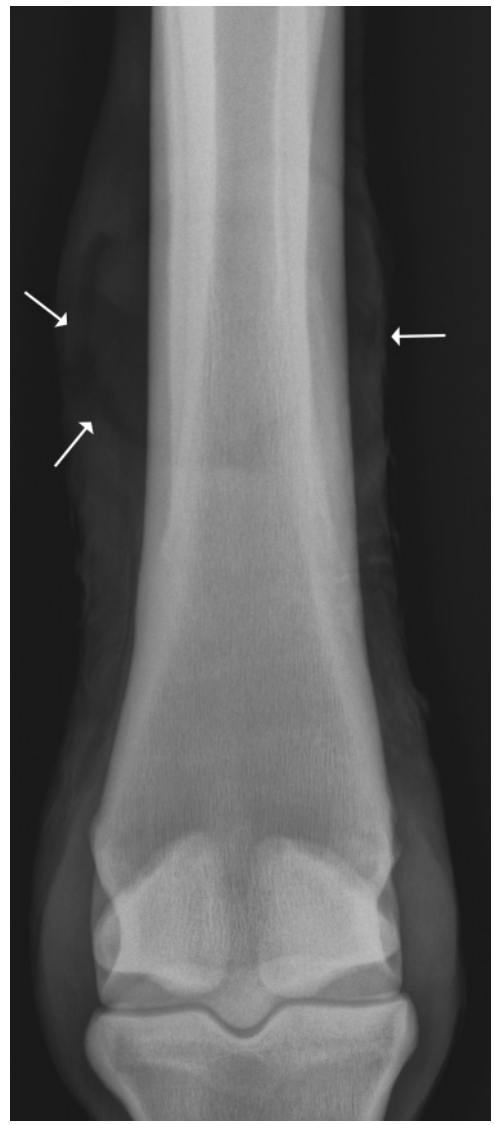


**Figure 4.8.** Dorsopalmar (DP) projection of the metacarpus. Surrounding the mid third metacarpal bone, the skin margins are irregular and there is gas within the soft tissue secondary to a skin laceration (arrows). Courtesy of Alejandro Valdés-Martínez.

## Gas

Gas may be present in the soft tissue structures of equine limbs as a result of traumatic lacerations, puncture wounds, needle centesis, or gas-producing bacterial organisms (Figure 4.8). Radiographic signs of soft tissue emphysema include radiolucent regions within soft tissue structures (the radiolucencies should be differentiated from fat) and a focal accumulation of gas with an air-fluid level that occurs with abscesses. The radiographic evidence of gas within the soft tissues in addition to an irregular skin surface should prompt the diagnosis of skin laceration.

Gas lucencies can be identified radiographically in subcutaneous tissue, muscle fascial planes, intramuscular tissue, and within the joint. Localization of soft tissue gas is important

diagnostically and prognostically. Gas within muscle tissue occurs with a gas phlegmon, intra-articular gas may be associated with the "vacuum phenomenon" (non-clinically significant) when a joint is flexed, or an air-fluid level within soft tissue may be diagnostic of an abscess.

#### Entheses

An enthesis is a point in the bone at which a soft tissue structure attaches. The soft tissues involved can include tendons, ligaments, or joint capsules. A pathologic change at these sites is known as enthesopathy and can be secondary to many disorders, but most commonly including trauma and degenerative or inflammatory conditions, and may be intra- or extra-articular (Figure 4.9). In cases of an acute traumatic event, an avulsion fracture at the enthesis may occur.

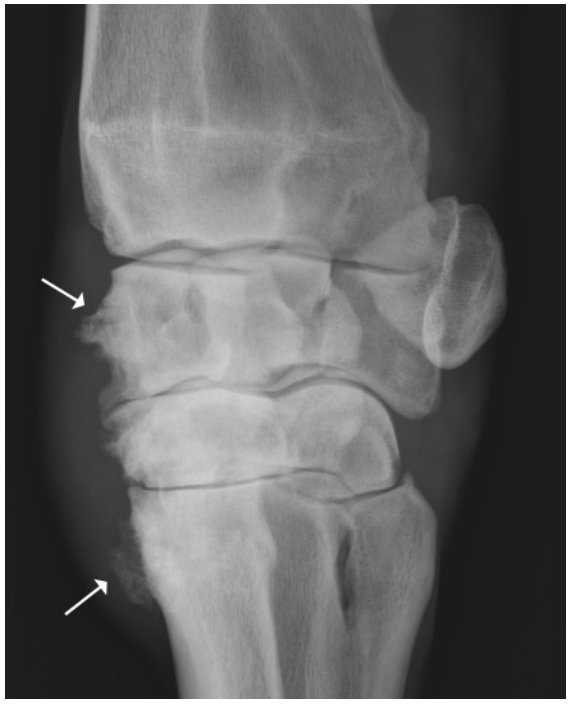


**Figure 4.9.** Dorsolateral-palmaromedial oblique (D45L-PaMO) projection of the carpus with periarticular osteophytosis and lysis at the middle carpal and carpometacarpal joints secondary to osteoarthritis. Note the rounded, bone proliferation on the dorsal surface of the intermediate carpal and proximal aspect of the third metacarpal bones consistent with enthesiopathy of the insertion of the joint capsule and extensor carpi radialis, respectively (arrows). Courtesy of Alejandro Valdés-Martínez.

Radiographic changes associated with enthesopathy include bone erosion, bone proliferation or hyperostosis (thickening of cortical bone), sclerosis, fragmentation, and adjacent soft tissue mineralization.

### *Radiology of Bone*

Knowledge of normal radiographic anatomy and basic bone response patterns is essential for evaluating bone structures radiographically in equine limbs. The clinician should be familiar with the normal shape of bones and the location and appearance of protuberances and fossae in mature animals; this information is available from standard references.

#### Fundamental Patterns of Bone Response

The response of bone to different pathologic processes is limited and consists in one or a combination of the following processes: new bone production/formation, bone resorption/destruction, or shape alteration. Bone remodeling and bone modeling are two terms commonly used interchangeably when referring to musculoskeletal radiology. Physiologically, osteoclastic and osteoblastic activities occur in both processes. During the bone remodeling process, the osteoblasts and osteoclasts are coupled and act together; therefore, bone resorption and formation occur at the same time and at the same site on a bone surface. On the other hand, during the bone modeling process, the bone alterations result from independent actions from osteoclasts and osteoblasts. This means that the bone resorption and formation may occur on different sites. In addition, modeling may cause large changes in bone structure, whereas remodeling will replace bone, maintaining the current amount of bone structure. Therefore, the correct term for describing visible bone structural changes is modeling rather than remodeling.

Radiographically, new bone production commonly is manifested as a periosteal or endosteal reaction (adjacent to the cortex), new bone at periarticular margins or entheses, or as increased bone opacity (sclerosis). Areas of bone destruction are seen as bone lysis (aggressive bone lesions or osteoarthritis) or decreased bone opacity (osteopenia).

Pathologic shape alteration is commonly radiographically seen in, but not particular to, skeletally immature horses, usually secondary to physeal problems or abnormal weight-bearing. To formulate a correct diagnosis, the clinician

should note fundamental bone response patterns and distribution within bones and any associated soft tissue changes on equine limb radiographs. Also, the clinician should be able to differentiate whether these changes are a response to pathologic processes or secondary to normal bone modeling as adaptation to a particular athletic activity.

### Periosteal Reactions

The periosteum is stimulated when elevated by hemorrhage, pus, edema, or infiltrating neoplastic cells. In the horse, direct trauma; extension of soft tissue infections; and avulsion of ligaments, tendons, and/or joint capsules are most frequently associated with periosteal new bone production.

Periosteal bone production may be acute or chronic. Acute periosteal bone production has an irregular, indistinct border and may be continuous or interrupted, laminated or spiculated (Figure 4.9). Acute periosteal reaction is usually active. Chronic periosteal bone production has a smooth, well-defined border, is solid, and often blends with the adjacent cortex. This type of periosteal reaction is usually inactive and often indicates a healed process, such as a healed fracture or previous active periosteal bone production that has changed to a chronic, probably inactive stage.

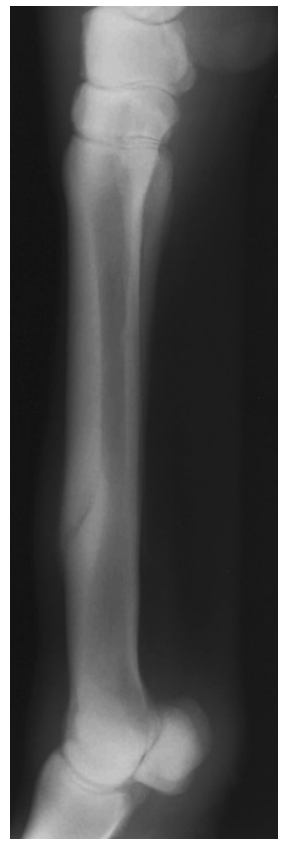


**Figure 4.10.** Lateromedial (LM) projection of the metacarpus. A radiolucent line is seen through the dorsal cortex of the mid MCIII, consistent with a stress fracture. There is also associated cortical and soft tissue thickening. Courtesy of Alejandro Valdés-Martínez.

### Cortical Changes

Cortical changes that can be identified radiographically consist of defects, erosions, lysis, and changes in thickness. Cortical defects seen most frequently in equine extremities are caused by fractures. Fractures must be differentiated from nutrient foramina, physeal lines, and edge enhancement shadows caused by superimposed bones (Figure 4.10). Long bone cortical stress fractures may not be evident in all cases as a distinct fracture line; a periosteal and/or endosteal reaction may be the only visible radiographic change (Figure 4.11).

Cortical lysis usually is caused by infection and typically has a permeative and/or moth-eaten pattern. A sequestrum also may be associated with a focal area of cortical lysis (Figure 4.12). In such cases, a dense sequestered piece of bone can be identified surrounded by a lytic zone (cloaca), which, in turn, is surrounded by bone sclerosis, producing an involucrum.

Cortical erosion changes can extend from either the endosteal or the periosteal surface. In the horse, they are most frequently encountered adjacent to the periosteal surface. Erosive changes with an irregular border usually result from infiltration into the bone and are most often caused by infectious processes. Cortical erosive areas with a smooth border are the result of pressure erosion, such as that seen with proliferative synovitis in the MCP/MTP joints.

Cortical erosions seen on the flexor surface of the navicular bone are secondary to a degenerative disorder initiated and promoted by excessive and sustained forces of compression against the flexor surface (Figure 4.13). Similar microscopic changes consisting of focal cartilage degeneration and lysis, thickened subchondral bone, and fibrous ankylosis to the opposing surface at the sites of subchondral bone destruction also are

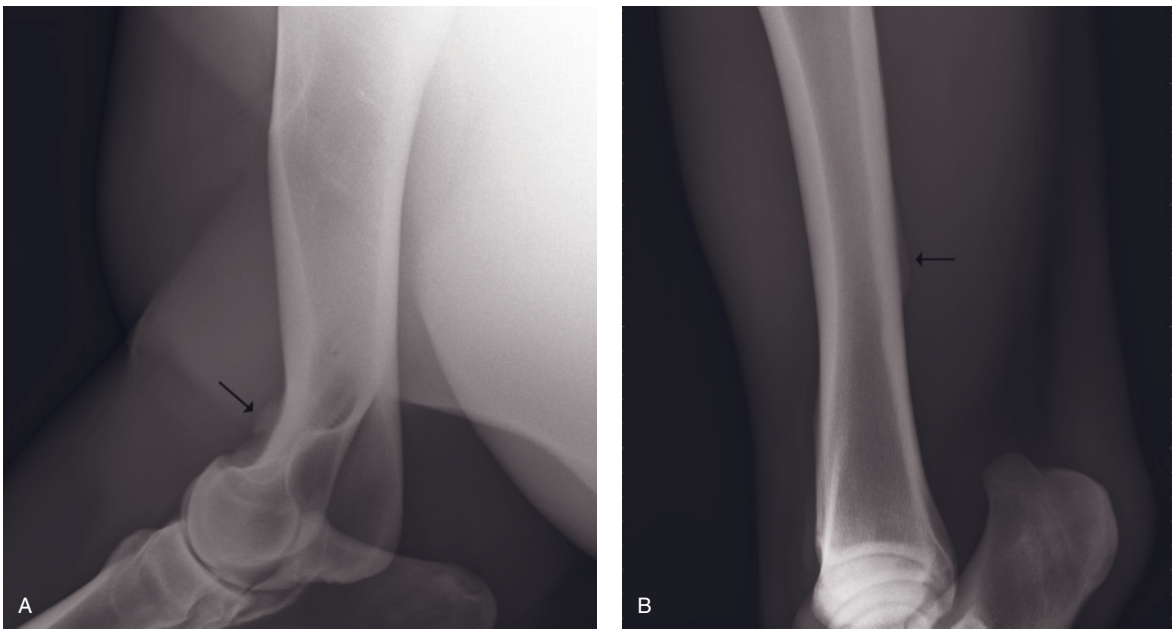



**Figure 4.11.** Examples of humeral (A) and tibial (B) stress fractures with only radiographic evidence of a periosteal reaction (arrows). Courtesy of New Bolton Center, School of Veterinary Medicine, University of Pennsylvania.

seen in the periarticular margins and articular surfaces of high-load, low-motion joints; these include the PIP joint and the DIT and TMT joints (Figure 4.14).

Generalized bone opacity may be decreased or increased. Decreased bone density is seen secondary to disuse of the limb or distal to a fracture. The osteopenia that develops in these limbs can be recognized radiographically as a coarse primary trabecular pattern with or without thin cortices. Increased bone opacity is identified radiographically with loss of the trabecular pattern secondary to bone deposition within the medullary cavity. Sclerosis is common on the third carpal bone and the proximal MCIII or MTIII at the origin of the suspensory ligament (Figure 4.15).

### Radiographic Signs (Bone Response Patterns) with Osteomyelitis

Osteomyelitis in equine limbs may be hematogenous in origin or result from penetrating wounds or open fractures. The region affected depends on the source and route of infection. Both acute and chronic osteomyelitis can be identified radiographically (Figure 4.16). It generally takes seven to 10 days after clinical signs of acute osteomyelitis are observed before the earliest detectable radiographic bone changes occur because at least 50% of mineral content must be depleted from the bone to be radiographically visible.

Because osteomyelitis can affect any bone in an equine limb and must be differentiated from other focal bone lesions, it will be used to illustrate the use of radiographic signs or bone response patterns to arrive at a radiographic diagnosis. Identifying radiographic signs requires close inspection of the radiograph and is an important step in accurately establishing a specific or differential diagnosis.

The following are radiographic signs manifested by acute osteomyelitis:

- Soft tissue thickening adjacent to the bone. This thickening is manifested by increased opacity, mottling, and obliteration of adipose tissue in fascial planes.
- Periosteal new bone production. This new bone has an irregular, indistinct border and parallels the bone cortices. Subtle areas of subperiosteal bone lysis may be seen in association with the acute periosteal bone reaction. These changes are not usually seen until seven to 10 days after clinical signs of the disease have been observed. As the disease progresses, the periosteal bone production parallels the

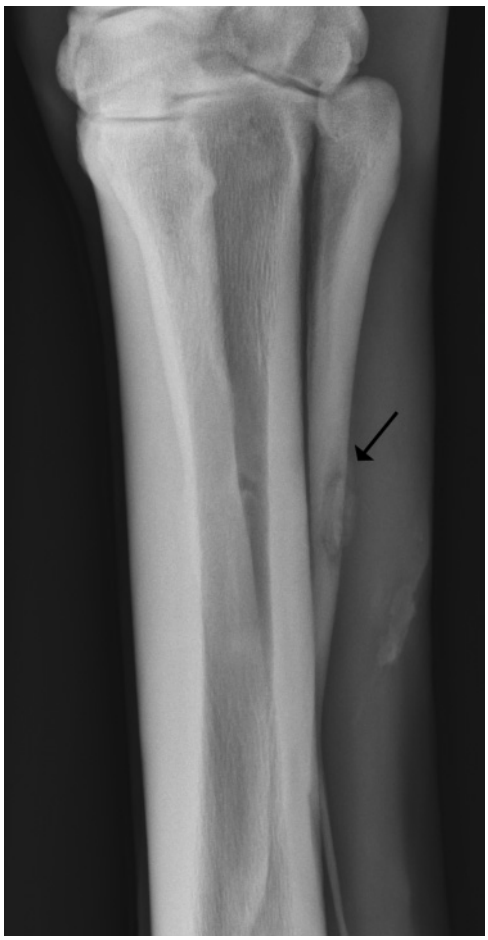


**Figure 4.12.** Dorsolateral-palmaromedial oblique (DL-PaMO) projection of the metacarpus, showing a well defined osseous body (sequestrum) surrounded by a radiolucent rim (cloaca) on the mid diaphysis of MCIV. There is mild surrounding sclerosis and a small periosteal reaction just proximal to the cloaca representing the involucrum (arrow). Courtesy of Alejandro Valdés-Martínez.

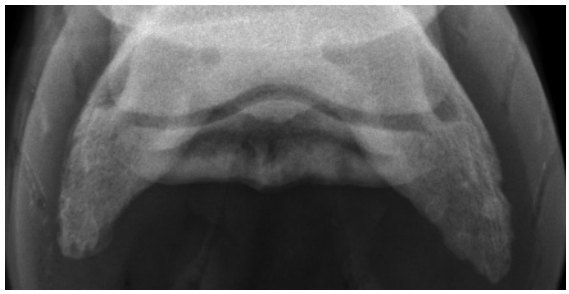


**Figure 4.13.** Palmaroproximal-palmarodistal oblique (Pa45Pr-PaDiO) projection of the navicular bone. On the flexor aspect of the navicular bone, note the poor corticomedullary definition, irregular medullary sclerosis, and cortical erosions consistent with degenerative changes.

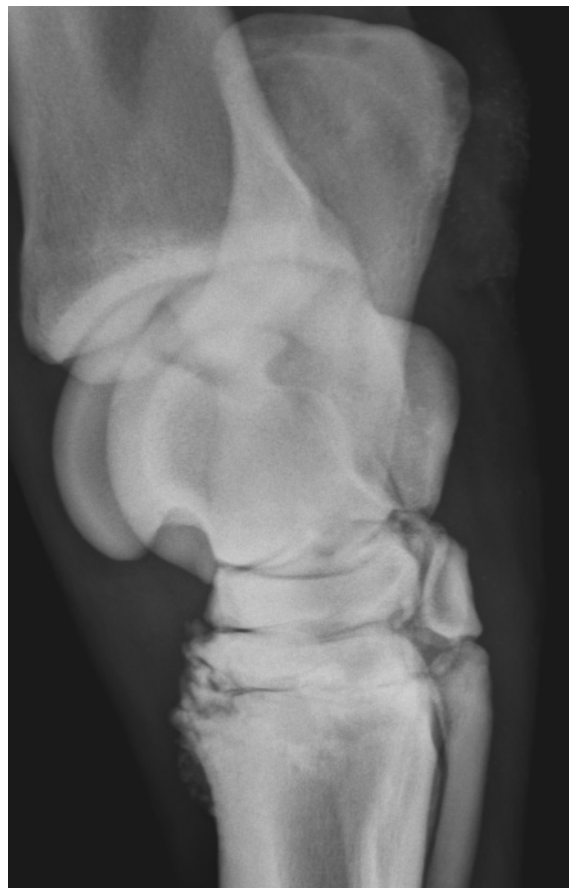


**Figure 4.14.** Dorsomedial-plantarolateral oblique (D55M-PlLO) projection of the tarsus. Sclerosis of the dorsal aspect of the central and third tarsal bones and irregular periarticular osteophytosis and subchondral bone erosions are present at the dorsolateral aspect of the DIT and TMT joints, consistent with severe osteoarthritis. Irregular periosteal reaction also is seen on the proximal dorsolateral aspect of MTIII secondary to enthesiopathy at the insertion of the peroneus tertius and tibialis cranialis muscles. Courtesy of Alejandro Valdés-Martínez.

cortex and spreads proximal and distal from the original infection site.

- Permeative lysis. This is observed as small, round lucent areas, 2 to 3 mm in size, within cortical bone and may extend into the medullary cavity. A moth-eaten pattern of lysis with larger lucent areas also can be seen in acute and active aggressive lesions. These changes are usually seen in association with acute periosteal bone production.
- Areas of bone lysis within the physis, metaphysis, or epiphysis secondary to septic osteomyelitis in young animals. This lysis is very

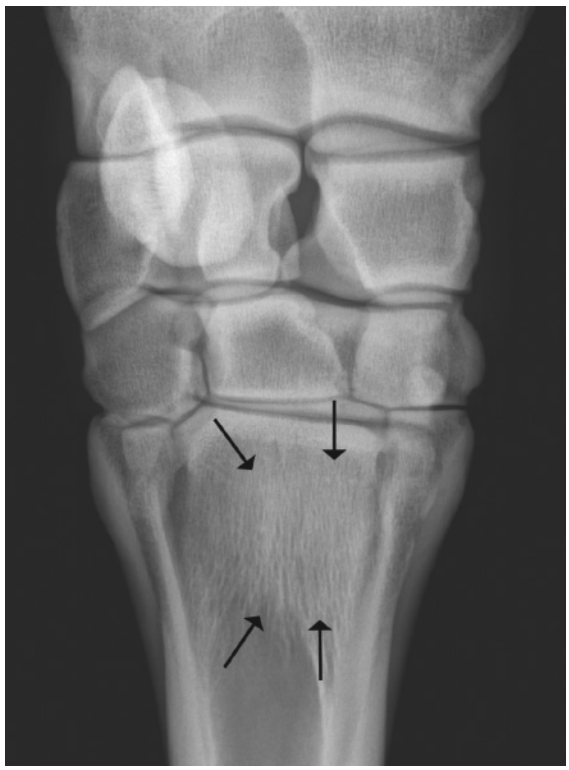


**Figure 4.15.** Dorsopalmar (DP) projection of the carpus, demonstrating sclerosis of the proximal aspect of MCIII with partial loss of the trabecular pattern, suggesting desmitis at the origin of the suspensory ligament (arrows). Courtesy of Alejandro Valdés-Martínez.

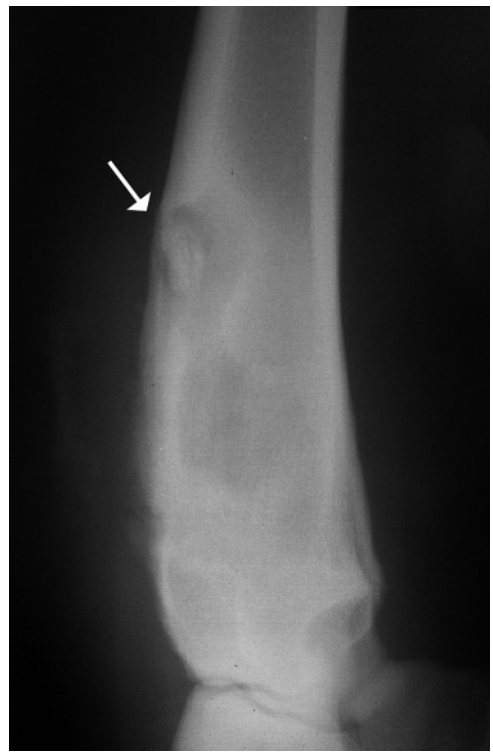


**Figure 4.16.** Lateromedial (LM) projection of the distal radius with radiographic changes consistent with osteomyelitis. Note the irregular and discontinued periosteal reaction with lysis of the craniodistal cortex of the distal radius and adjacent ill defined increased medullary opacity and soft tissue thickening. On the most proximal aspect of the lesion, the cranial cortex is wide and presents a circular mineral opacity with a surrounding radiolucent rim and sclerosis suggesting the presence of a sequestrum (arrow).

aggressive and mostly destructive, giving little opportunity to the body to produce bone in the attempt to wall off the infection.

Chronic osteomyelitis may have the following radiographic changes:

- Large cortical defects, some of which may be as large as 1 cm in size and can also involve the medullary cavity. Although rare, bone abscesses may appear as a geographic pattern of lysis represented by a single, well-defined medullary lytic area with surrounding sclerosis.
- Localized increased bone densities (sclerosis), which are produced within the host bone( e.g., thick cortices) and in which a sequestrum sometimes may be identified within the sclerotic and lytic bone patterns
- Periosteal bone production, which is usually abundant with a well-defined, irregular or smooth border.

## *Radiology of Synovial Joints*

Radiographic evaluation of joints in the equine limb is an important part of the diagnostic workup for lameness and encompasses evaluation of several joint structures or areas, including soft tissue structures (both intracapsular and extracapsular); joint margins; subchondral bone; the "joint space"; ligament, tendon, and joint capsule attachment areas; and joint alignment.

### Radiographic Changes Associated with Joint Disease

The radiographic examination is helpful for evaluating the type and extent of joint disease. The radiographic manifestations of joint disease occur in the soft tissue and bone structures and

may develop before or after clinical signs of the disease develop. The bone changes follow clinical signs in septic arthritis and may precede or follow clinical manifestations with osteoarthritis (OA).

Soft tissue changes that may be observed radiographically are periarticular soft tissue thickening, joint capsule distension, and mineralization. The location of fat bodies (adipose tissue masses) and adipose tissue in fascial planes can be used to evaluate periarticular swelling and joint capsule distension. Periarticular mineralization may be associated with numerous causes, but in the horse is predominantly dystrophic or secondary to blunt soft tissue trauma.

Marginal joint changes consist of periarticular osteophyte formation and bone lysis. Marginal periarticular osteophytes usually are associated with OA (Figure 4.17), whereas marginal bone lysis is most often seen with septic arthritis (Figure 4.18). In early stages, marginal changes may be subtle, but with advanced or more severe disease, the changes are easily identified.

Subchondral bone changes consist of sclerosis, lysis, and fragmentation. Subchondral bone sclerosis may be present with OA, although it seems to be recognized radiographically in only the more pronounced or longstanding cases. Subchondral bone lysis can have a local or general distribution within the joint and may be seen in association with subchondral bone fragments. Subchondral bone lysis may be present with septic arthritis, OA, osteochondrosis, or "traumatic arthritis." Localized or general subchondral lytic patterns with irregular, indistinct margins are associated with septic arthritis. Localized, well-defined lytic lesions are seen with osteochondrosis, which may develop into subchondral cyst-like lesions. Focal subchondral lytic areas associated with bone fragments are seen with osteochronditis dissecans and traumatic arthritis lesions from chronic mircofractures in the subchondral bone. Traumatic arthritis

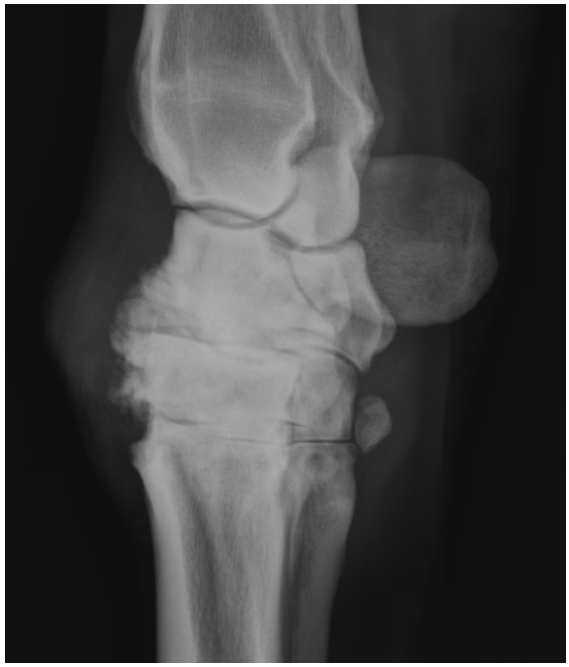


**Figure 4.17.** Dorsomedial-palmarolateral oblique (D30M-PaLO) projection of the carpus. Severe periarticular osteophytosis is present at the middle carpal and the carpo metacarpal joints with sclerosis of the cuboidal bones and focal soft tissue thickening consistent with osteoarthritis. Courtesy of Alejandro Valdés-Martínez.

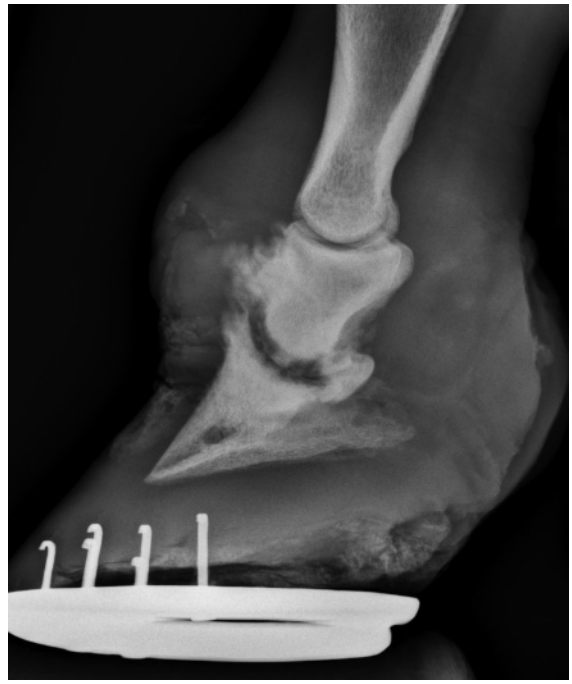


**Figure 4.18.** Lateromedial (LM) projection of the distal limb showing radiographic changes consistent with septic arthritis. Note the severe irregular subchondral bone lysis on the adjacent articular surfaces of the DIP joint with marked articular cartilage loss, the irregular periosteal proliferation on the dorsal and palmar surfaces of P2 and dorsal P3, and the severe soft tissue thickening with irregular skin surface centered over the dorsal aspect of the DIP joint. In addition, there is a coarse trabecular pattern on P1 consistent with disuse osteopenia.

lesions are usually seen on the dorsal surfaces of joints and are caused by hyperextension trauma.

The joint space width may be increased or decreased. An increased width may be associated with joint effusion, although in weight-bearing studies, this is seldom apparent. An increased joint space associated with subchondral bone lysis can be seen with extensive septic arthritis. A decreased joint space, either general or localized within the joint, is associated with cartilaginous lesions and degeneration and occurs predominately with OA (Figure 4.17).

Periarticular enthesiophytes usually are associated with joint capsule, ligament, or tendon damage or avulsion from their bony attachments (Figure 4.9). The enthesiophytes (periosteal new bone production) are irregular in the acute stages, which distinguishes them from marginal periarticular osteophytes, and occur at tendinous and ligamentous attachment areas.

Alignment abnormalities may consist of subluxation or luxation of a joint or may simply result in an abnormal degree of flexion or extension of a joint in a resting position. Abnormal joint alignment may be associated with ligament laxity and/or injury (Figure 4.19), tendon injury or contracture, abnormal bone growth (i.e., angular limb deformities in foals) and healed malaligned fractures. Chronic alignment abnormalities predispose the joint to degenerative disease from abnormal weigh-bearing and stress distribution through the joint.

### Radiographic Changes with Specific Joint Conditions

Degenerative joint disease, osteoarthrosis, or OA is a secondary condition in the horse (Figures 4.9, 4.14, 4.17). The severity of radiographic changes usually correlates with the severity and/or duration of the disease process. The following are radiographic changes seen in the horse, listed in order from most common to least common:

- Marginal periarticular osteophytes
- Narrowed joint space, which may involve all or only part of the joint; distinct, smooth borders remain on the subchondral bone adjacent to the articular cartilage
- Well-defined subchondral bone lucencies, as seen in chronic OA of the tarsus and carpus
- Subchondral bone sclerosis
- Subchondral bone cystic degeneration, which occur infrequently as sequelae to OA.

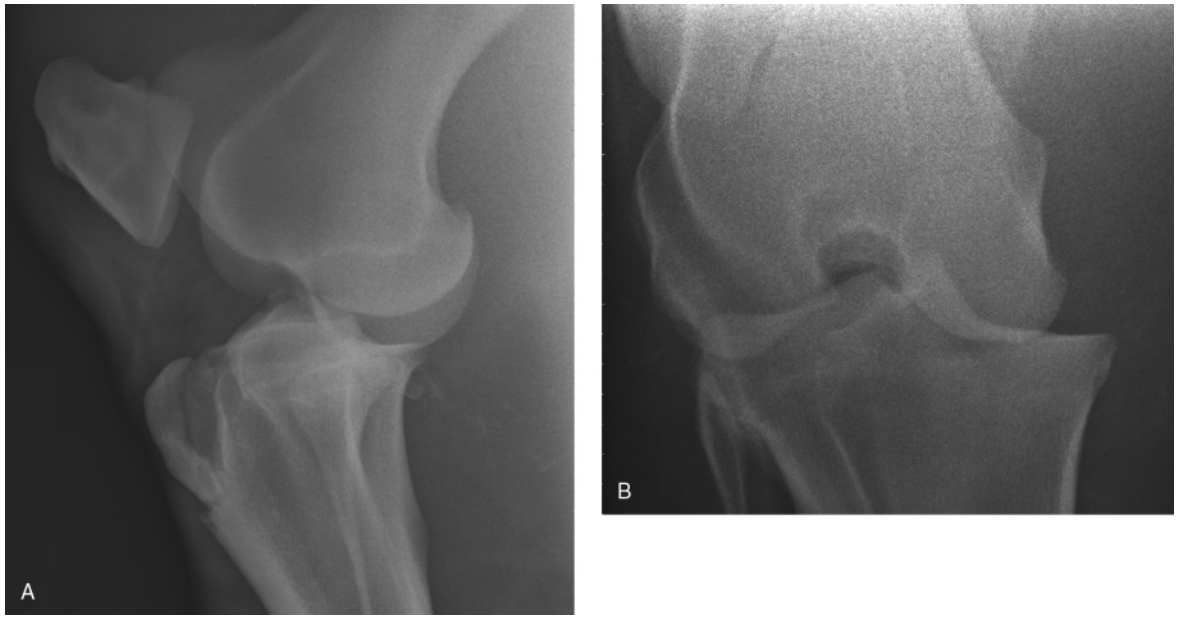



**Figure 4.19.** (A) Lateromedial (LM) and (B) Caudo-cranial (Ca-Cr) projections of the stifle. Image (A) shows cranial displacement of the tibia with partial loss of the cranial fat pad consistent with cranial subluxation and stifle joint effusion likely secondary to cranial cruciate ligament injury. Image (B) shows medial displacement of the tibia consistent with lateral to medial instability secondary to collateral ligament injury. Courtesy of Alejandro Valdés-Martínez.

Septic arthritis may be hematogenous in origin or result from extension of an adjacent osteomyelitis, cellulitis, or a penetrating injury (Figures 4.18, 4.20). The following are radiographic signs of septic arthritis:

- Periarticular soft tissue thickening and joint capsule distension
- Marginal bone lysis, which may occur early in the disease
- Subchondral bone destruction, which may be an extension from or occur without the marginal lysis
- Periosteal reactions, which may be adjacent to the joint but are generally distributed around the joint; when septic arthritis has occurred from extension of an adjacent osteomyelitis or cellulitis, the periosteal reaction may precede the intra-articular changes

Osteochondrosis is associated with regions of high predilection in specific joints, which should be observed when diagnosing the condition. It is caused by defective osteochondral development, which usually involves subchondral bone (Figures 4.21 to 4.23). The following are radiographic changes present with osteochondrosis.

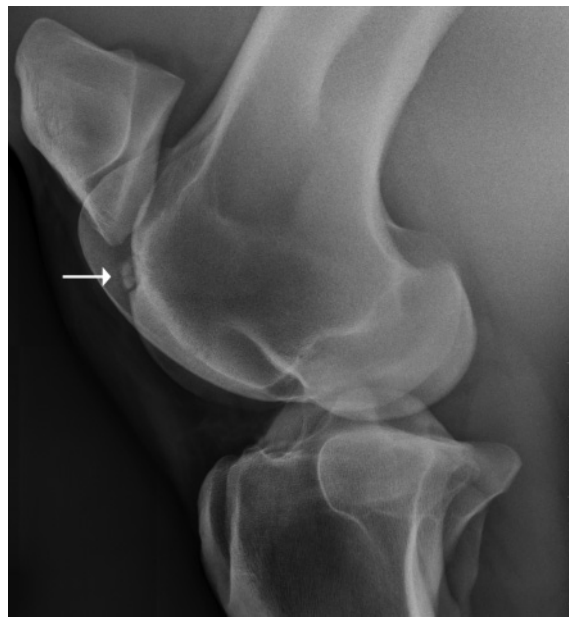


**Figure 4.21.** Lateromedial (LM) projection of the stifle. Note the well defined, oval, osseous body associated with a concave subchondral bone defect on the lateral trochlea ridge (arrow) consistent with osteochondritis dissecans. Courtesy of Alejandro Valdés-Martínez.

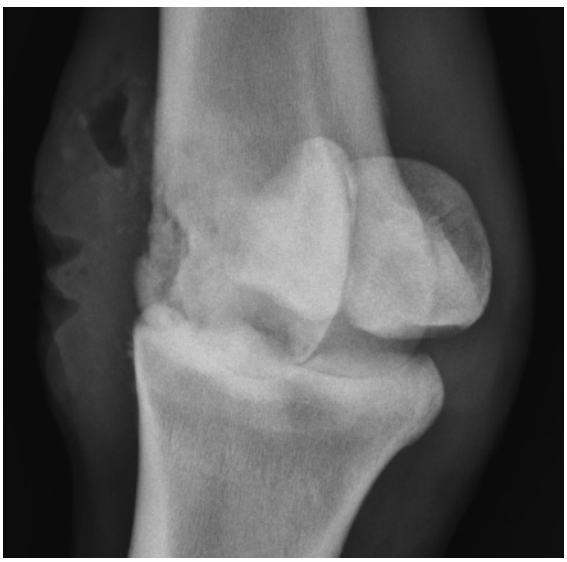


**Figure 4.20.** Dorsomedial-palmarolateral oblique (DMPLO) projection of the MCP joint. An area of permeative lysis is present on the dorsal aspect of the lateral condyle of MCIII. Marked narrowing of the joint space, mild irregular periosteal reaction on the dorsolateral aspect of proximal P1, and associated soft tissue thickening with subcutaneous gas are also present. These findings are consistent with septic arthritis.

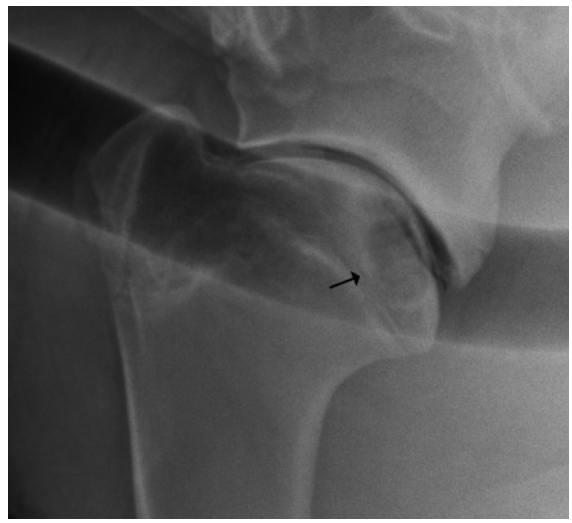


**Figure 4.22.** Mediolateral (ML) projection of the shoulder. A large radiolucent subchondral bone defect (cyst) surrounded by a rim of sclerosis is seen on the caudal aspect of the humeral head (arrow) consistent with osteochondrosis. Courtesy of Alejandro Valdés-Martínez.

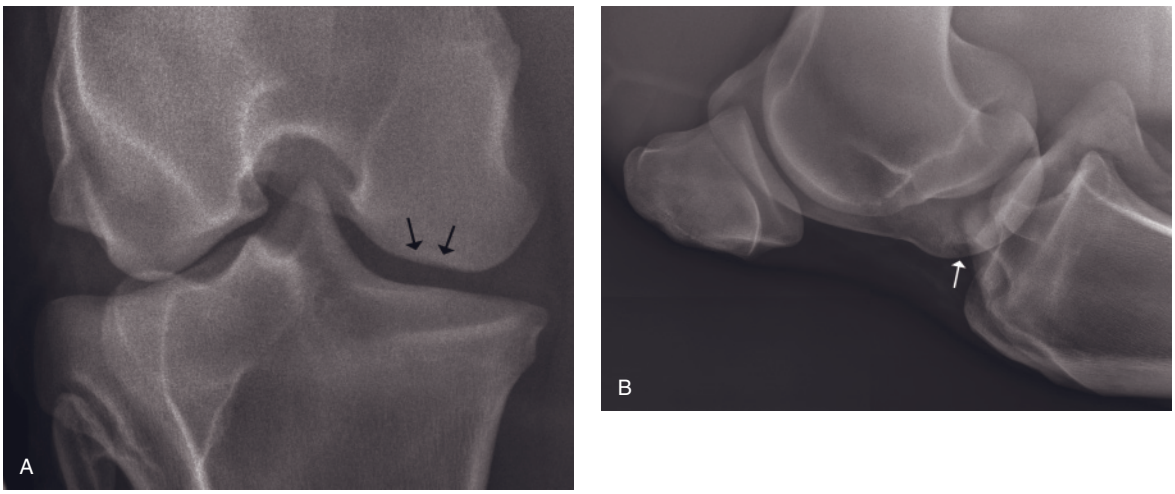



**Figure 4.23.** (A) Caudo-cranial (Ca-Cr) and (B) flexed lateromedial (flexed LM) projections of the same stifle. Image (A) shows a mild flattening and sclerotic region on the articular surface of the medial femoral condyle (arrows). Image (B) shows the cranial aspect of the medial femoral condyle free of superimposition, which allows the identification of a well defined, subchondral bone defect (cyst) surrounded by sclerosis (arrow) consistent with an osteochondrosis lesion. Courtesy of Alejandro Valdés-Martínez.

- Flattening of the subchondral bone surface
- Localized subchondral bone defect (lysis)
- Osteochondral bone fragments, which are seen radiographically as osseous bodies representing osteochondritis dissecans
- Secondary OA changes that may be present
- Subchondral cyst-like lesions that may develop secondary to osteochondrosis

Traumatic joint disease also manifests with subchondral bone lysis. These lesions must be differentiated from true osteochondrosis lesions. Areas of predilection for traumatic joint disease are the MCP joints and carpus. Traumatic joint disease develops from increased weight-bearing and stress on a joint surface, resulting in bone sclerosis and eventual microfractures, which leads to subchondral bone lysis. It also may develop after hyperextension injury in joints such as the carpus, producing microfractures, subchondral bone lysis, and small subchondral bone fragments. The following are radiographic signs of traumatic joint disease:

- Focal subchondral bone lysis with indistinct borders
- Subchondral bone sclerosis
- Bone fragments, which may be adjacent to the subchondral bone lysis
- Incomplete articular fractures (stress fractures)
- Changes in the contour of the bones, such as flattening of the medial femoral condyle or condyles of MCIII/MTIII

### *Limitations of Radiography*

With the lack of detailed soft tissue visualization, radiography has a limited role in the evaluation of areas surrounding the bone. In the majority of cases, soft tissue thickening can be identified in plain radiographs and in combination with the history, clinical signs, and location; the practitioner will be able to differentiate if the soft tissue thickening is secondary to active inflammation or a chronic condition. However, in order to have the most accurate diagnosis and subsequently be able to provide the best care to the patient, visualization of architectural changes within the soft tissues is essential. Ideally, in these cases, other imaging modalities (i.e., ultrasound or magnetic resonance) should be considered to complement the radiographic findings.

Skeletal abnormalities are identified radiographically based on changes in bone density/opacity (reduced or increased) and shape. Because skeletal lesions may only be detected with radiographs after approximately 50% change in bone mineralization is present, some conditions such as acute osteomyelitis, early synovitis, early cartilage loss/erosive arthritis, and minimally or

nondisplaced fractures may not be recognized on the initial radiographic exam. It may take up to two weeks before the bone changes are detected with plain radiographs.

In cases of comminuted fractures, radiography may be unable to define the lesion clearly. In these cases, computed tomography with multiplanar and three-dimensional (3D) reconstructions should be considered for surgical planning.

In summary, the practitioner should be aware of the above mentioned limitations of radiography and that the combined results of different imaging modalities may be necessary to obtain a diagnosis. More important, it should always be remembered that radiographic changes do not necessarily represent lameness.

## ULTRASOUND

### *Introduction*

Diagnostic ultrasound was introduced to equine veterinarians in the early 1980s. At that time veterinary ultrasound systems were predominantly designed and used for reproductive examinations with large, 5-MHz rectal probes. While these probes were less than ideal for examining the superficial structures of the musculoskeletal system, a few innovative equine veterinarians embraced this new technology and began to evaluate the flexor tendons of the metacarpus. Shortly thereafter, higher frequency mechanical sector scanners with multiple frequency transducers (3.5 to 7.5 MHz) became available and allowed detailed examination and classification of soft tissue injuries of the palmar/plantar metacarpus/metatarsus and pastern. This was the first time soft tissue structures of the distal limb could be seen as a tomographic or body slice image. It was now possible to evaluate the flexor tendons and suspensory ligament for morphological change.

Over the last couple of decades, ultrasound technology has dramatically improved and linear array ultrasound systems have been developed that are better suited for musculoskeletal examinations. Many of these high-end systems have 14- to 18-MHz linear tendon probes and 8- to 10-MHz microconvex probes with variable focusing capabilities and multiple frequencies. In addition, mainframe ultrasound platforms have been reduced to the size of notebook-sized computers and the miniaturization of electronics has reduced the quality differences between portable and stationary technologies. Ultrasonography is now considered the imaging modality of choice to evaluate soft tissue injuries in the horse.

### *Science of Ultrasound*

Ultrasonography is a two-dimensional (2D) real-time imaging technique that uses the transfer and propagation of sound waves into soft tissue. Ultrasound is defined as sound above the audible range. Ultrasound waves behave as classic sound waves that operate at frequencies spanning 1 to 20 MHz. These sound waves are mechanical waves that require some sort of medium to allow the waves to form and travel. The propagating medium determines how fast the sound wave travels, how easily the waves can be formed, and how well the traveling waves can remain together.

Ultrasound machines produce a sound wave of longitudinal orientation in which the elements of the medium are compressed and rarefied. The distance between the start of one cycle of compression and rarefaction and the next is considered the wavelength, and most wavelengths are 1 mm or less. The propagation speed of the ultrasound wave is determined by the density and stiffness of a given tissue, with bone propagating at higher speeds, fluid-filled structures propagating at medium speeds, and air propagating at the lowest speeds. The average propagation velocity of the sound wave in soft tissues is around 1,540 meters/second. Ultrasound waves lose energy to the medium in the form of heat through a process termed absorption. Absorption increases directly with distance and frequency. A transducer produces short bursts of specific frequency sound waves, which are transmitted into the patient and reflected back at different tissues and tissue interfaces. The transducer then detects the reflected sound waves and these waves are converted to electrical energy. A computer plots the time the sound waves traveled along with the amplitude of the reflected sound waves.

Echoes are produced at tissue interfaces of different acoustic impedance. Acoustic impedance is a measure of how easily waves can be formed and depends on sound velocity and tissue density. The greater the differences in acoustic impedance of the reflecting interfaces, the greater the intensity of the returning echo. Ultrasound waves are constantly encountering changes in soft tissue that can affect propagation of the sound wave, which cause scatter and a weakening of the return echoes. The brightness of the dot on the monitor screen correlates to the amplitude of the returning echo. The appearance of an image relates to the tissue's echo intensity or echogenicity. The echogenicity of a structure or the degree to which the structure reflects

sound waves determines the brightness of objects on ultrasound. All of this information is displayed as a cross-sectional image developed by an entirely different set of physical parameters of structures (objects) than those measured by other imaging modalities.

Most current musculoskeletal ultrasound systems use variable focus linear and convex array transducers. Flat-face linear and microconvex probes are the most popular probes for musculoskeletal imaging. Linear probes give superior images at tissue depths of 2 cm or less due to less distortion and artifact creation in the near field.

Stand-off pads are available for linear probes to improve contact with the skin, which increases the footprint, and move the superficial structures into the near field focal zone and away from the near field artifact. A stand-off also increases the footprint or image field of the scan head.

Linear probes also provide excellent evaluation of longitudinal fiber alignment. Convex array transducers are used when the skin is contoured and it is difficult to seat the flat-face transducer. The divergent beam allows the examiner to image from a smaller skin contact point. These convex probes can be more difficult to use because it is easier to inadvertently change the beam angle, especially when doing longitudinal assessments of fiber alignment. In addition to the superior imaging, these transducers have lower purchase prices and lower cost of maintenance when compared to sector technology. Many of these probes have multiple frequencies available, which allows the examiner to easily change the frequency without needing to change the probe. Structures within 5 to 7 cm of the skin should be evaluated with transducers of a minimum of 7.5 to 10 MHz or higher. Structures within 7 to 14 cm should be evaluated with 5-MHz transducers. Anything deeper than 14 cm requires lower frequencies such as 2.5 to 3.5 MHz.

### *Diagnostic Ultrasound to Evaluate Tendons and Ligaments*

Ultrasonography has significantly advanced the diagnosis and management of a variety of musculoskeletal injuries in performance horses. A working knowledge of the normal anatomy is critical for tendon and ligament ultrasonographic examinations as well as those involving joints, sheaths, and bursae.

Ultrasonography is routinely used to define morphological change in the superficial digital flexor tendon (SDFT), deep digital flexor tendon (DDFT), suspensory ligament (SL), accessory ligament of the DDFT (ICL), and distal sesamoidean ligaments (DSL) of the pastern region. The subcutaneous tissue, peritendinous tissue, vessels, and the contour of the cortical bone in the region should be assessed as well. Most importantly, diagnostic ultrasound is the most useful and practical tool to monitor the repair of these structures and guide the rehabilitation of tendinous and ligamentous structures.

Many other soft tissue structures also can be evaluated, including muscle, musculotendinous junctions, tendon sheaths, and bursas associated with the tendons and ligaments. Joint injury is very effectively examined with radiography and ultrasonography. These imaging tools are considered complementary and provide more information about a joint than either tool used alone. Joint examination should include evaluation of the periarticular structures such as the collateral ligaments and extensor/flexor tendons, joint capsule, and joint fluid accumulations. The real-time imaging capability of ultrasonography allows the use of interventional techniques (such as needle insertion for injection or aspiration/biopsy), which can provide additional clinical information.

Ultrasonography also is used to evaluate a variety of other problems such as fractures of long bones, osteitis/osteomyelitis, foreign body penetration, and implant infection. It also is used intra-operatively to assist with some surgical procedures.

#### Patient Preparation and Scan Protocol

Confirmation that lameness is associated with a specific structure or area is critical. Localization of lameness should include a clinical examination and the use of diagnostic nerve blocks when necessary. In most instances, injection of diagnostic anesthesia into an area will not interfere with the ultrasonographic examination. Occasionally gas bubbles in the injectate may inhibit sound transmission and necessitate performing the exam on a subsequent day. Tranquilization may be necessary and can assist the examination.

Patient preparation is very important and should include clipping with a #40 blade. Both limbs should be clipped and prepped because strain-induced tendon and ligament injury can occur bilaterally with one limb being more severely affected than the other. Shaving is frequently required to give a higher resolution

image. A scrub with a detergent generally is necessary to remove dirt and debris. Many clinicians do a 5-minute sterile prep with antiseptic solution/detergent followed by an alcohol rinse.

The limb should be liberally coated with ultrasound gel that is left undisturbed for 5 minutes. Excessive gel can cause a lateral image artifact which may compromise image quality. When clipping and shaving is not possible, the limb should be thoroughly washed with warm water and detergent. Application of alcohol to the haircoat may enhance the sound transmission. To acquire the best image, a scan head with a frequency of at least 7.5 MHz but frequencies of 10 to 18 MHz are preferable. Low frame rates should be used to give higher line density and improved resolution. Most superficial structures can be visualized at a scan depth of 2 to 4 cm, often with the use of a stand-off pad.

The ultrasonographic examination should be performed in a systematic manner with each structure evaluated from proximal to distal to ensure a complete and thorough tendon/ligament evaluation. The examiner should develop a systematic approach to screening the limb, such as the technique described by Genovese and Rantanen. This approach provides a survey of all structures including veins, arteries, subcutaneous tissue, paratendinous tissue, and bone contour at specific levels in the metacarpus/metatarsus. A standardized scanning protocol has numerous advantages, but most importantly it provides a means for clinicians to effectively screen the limb as well as provide a means for veterinarians to accurately communicate their findings.

Imaging protocols are based on the premise that the metacarpus is approximately 24 cm in length or roughly 3 hand-widths of a person's hand (8 cm/hand breadth). The metatarsus is longer that the metacarpus and measures approximately 32 cm or roughly 4 handwidths in length. Theses zones are numbered 1 through 3 in the forelimb and 1 through 4 in the hindlimb. Each zone is further subdivided into two equal zones, A and B (each 4 cm), such that the forelimb has zones 1A, 1B, 2A, 2B, 3A, 3B. The area associated with the proximal sesamoids of the fetlock is considered zone 3C (or 4C in the hindlimb). Some authors use a simple numerical scheme with the forelimb having levels 1 to 7 and the hindlimb having levels 1 to 9. These levels are the same zones mentioned above, but without the letter designations (Figures 4.24 to 4.32). To more completely assess the architecture of the SL branches it is necessary to incline the transducer more medial to lateral or lateral to medial as the examiner progresses distally until they attach to the proximal aspect of their respective proximal sesamoid bone (Figure 4.33).

The imaging protocol for the pastern is based on zones related to the proximal and middle pastern bones. The proximal pastern, or P1, has three 2-cm zones associated with it; they are named P1A, P1B, and P1C. The middle pastern bone, or P2, is shorter and has just two 2-cm zones associated with it, named P2A and P2B (Figures 4.34 to 4.41). The overall length of the pastern and height of the collateral cartilages of the distal phalanx can significantly affect the ability to image the DDFT in zone P2A. To evaluate the origin of the oblique sesamoidean ligaments on the distal aspect of the proximal sesamoid bone it is necessary to incline the transducer more dorsally such that a medial-to-lateral or lateral-to-medial orientation is obtained. The pastern is more difficult to examine than the metacarpus because positioning the probe between the distal aspect of the sesamoid bones (and the ergot) and the proximal aspect of the collateral cartilages can be difficult. Placing the foot on a block of wood with the limb more caudal than the opposite limb increases the extension of the fetlock joint and positions the axis of the pastern in a more upright position (Figure 4.42). In general, the DSLs and branches of the SDFT are smaller and inclined at different angles as they course to their insertions, which requires that different scan planes be used to evaluate each of these structures.

Each structure should be examined on cross section and on longitudinal orientation with appropriate placement of the focal zone(s) on the structure of interest at each location. In the metacarpus, each level should have two transverse scans performed. The first scan should have a stand-off placed on the transducer with the focal zone(s) and transducer angle initially directed at the SDFT and DDFT. The second scan should be acquired with or without a stand-off with the focal zone(s) and transducer angle directed at the ICL and the SL (Figure 4.43). In some instances it may be helpful to use a microconvex transducer for this scan because the divergent beam can improve assessment of the medial and lateral borders of the SL. The transverse scan plane should be positioned such that the structures on the left side of the horse are placed to the left side of the screen.

Some clinicians like to place the medial side of the limb on the left side of the screen and the

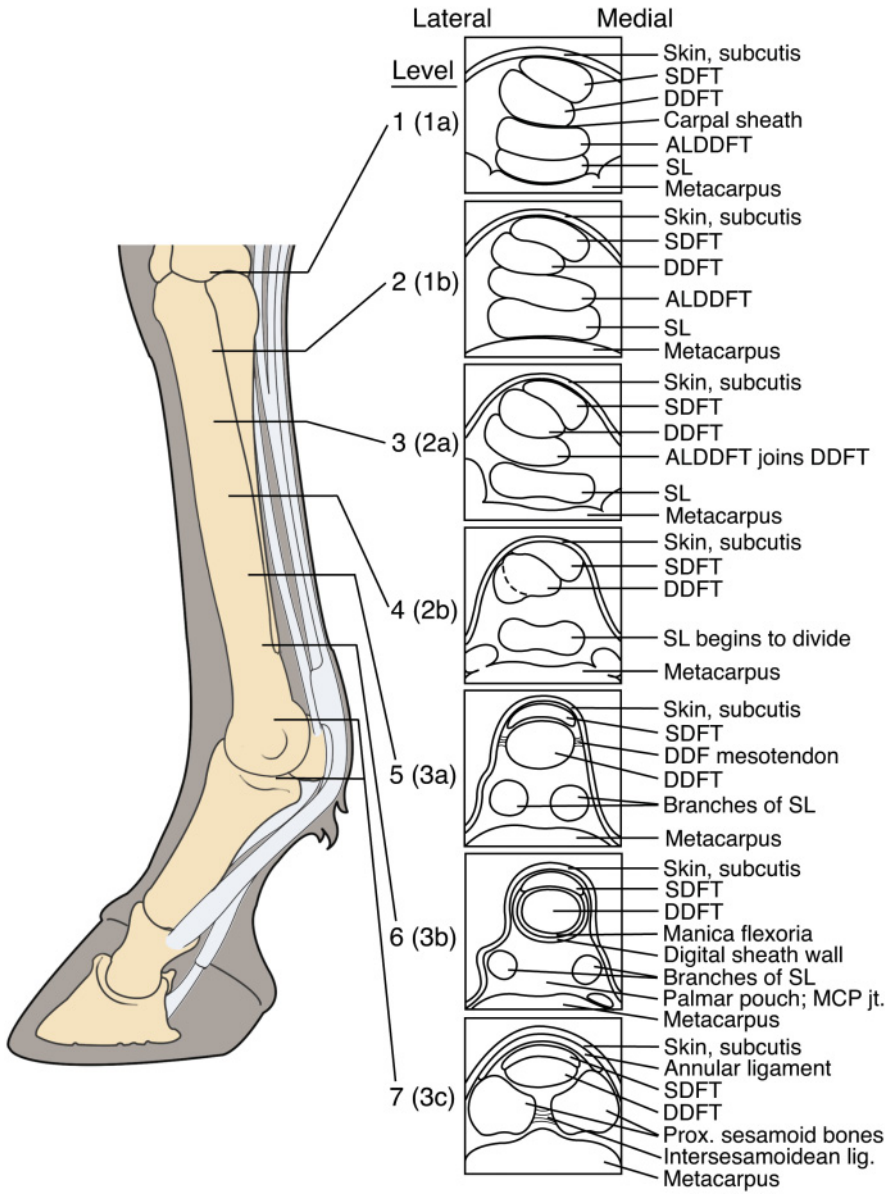



**Figure 4.24.** Ultrasonographic imaging of the metacarpus is based on the length being approximately 24 cm or roughly 3 hand-widths (8 cm/hand-breadth). These zones are numbered 1 through 3 in the forelimb. The zones are further subdivided into two equal zones, A and B (each approximately 4 cm), such that the forelimb has zones 1A, 1B, 2A, 2B, 3A, 3B. The area associated with the proximal sesamoid bones (PSB) of the fetlock is considered zone 3C. Some authors use a simple numerical scheme with the forelimb having levels 1 through 7. The structures identified from the transducer to the palmar metacarpus are the SDFT, DDFT, ICL, and the SL. Courtesy of Rich Redding.

lateral side of the limb on the right side of the screen. In addition, the sagittal scan plane should position structures of the proximal aspect of the limb to the left of the screen and structures to the distal aspect of the limb to the right side of the screen. Some clinicians place these structures opposite with the proximal aspect of the limb to the right of the screen. Whatever protocol is used, it should be done consistently with appropriate labeling on the recorded image.

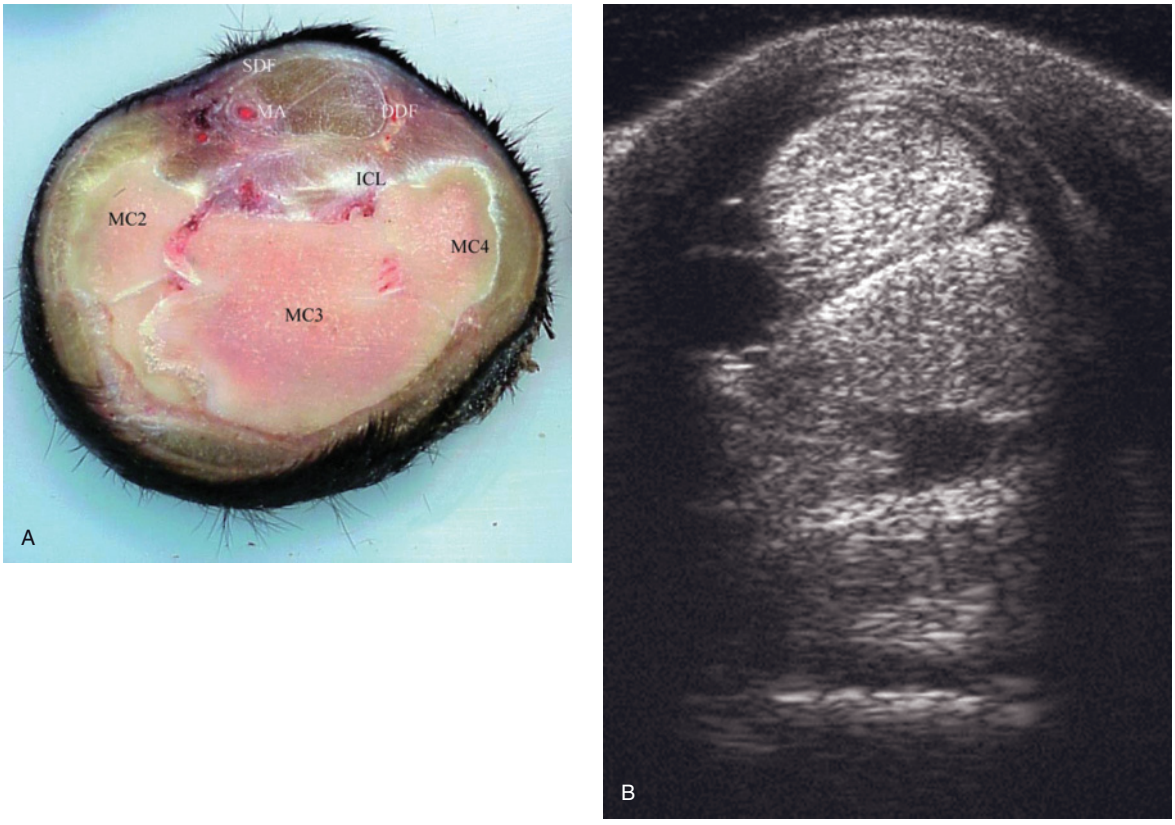



**Figure 4.25.** (A) In Z1A the SDFT is round but with a flat border dorsally where it is adjacent to the DDFT. The DDFT is rounded in shape. The ICL is located dorsal to the DDFT with the carpal sheath visualized as an anechoic triangular structure between these two structures. The SL is located dorsal to the ICL but may only have a few fibers apparent in proximal Z1A. The SL becomes more apparent progressing distally in this zone. (B) Ultrasound image taken at the proximal aspect of Z1A to correspond to the gross section slice plane that is close to the carpometacarpal joint. At this level the ICL is the only structure apparent dorsal to the DDFT. The median artery and large metacarpal vein are prominent on the medial aspect of the DDFT. Courtesy of Rich Redding.

Careful attention should be paid to all soft tissue structures because multiple structures are often involved. The examiner also should be aware that an injury can extend proximally into the carpal sheath or distally into the digital sheath/pastern area. If an abnormality exists, the lesion should be mapped and measured in centimeters from a reference point such as the accessory carpal bone in the forelimb and the point of the hock or tarsometatarsal joint (head of the lateral splint) in the hindlimb. The extent and severity of the lesion should be documented and mapped by measuring the proximal most extent of the lesion from the appropriate anatomical reference point to the distal most extent of the lesion. The lesion should be evaluated on both cross section and longitudinal scan planes. The lesion's maximum size should be determined and mapped. Echogenicity and fiber alignment should be subjectively evaluated throughout the abnormal tissue. Each image should be labeled with the date, owner's name, patient's name, limb being examined, and location of the lesion(s).

A description of the scan plane should be included in every image, and every image recorded as part of the medical record. Documentation of the lesion location, lesion echogenicity, and extent or degree of change with appropriate labeling should be included on the image before being stored and maintained as part of the permanent medical record. Images can be stored as thermal prints (which degrade over time), as video recordings, or as digital images (DICOM® files) for comparison at later

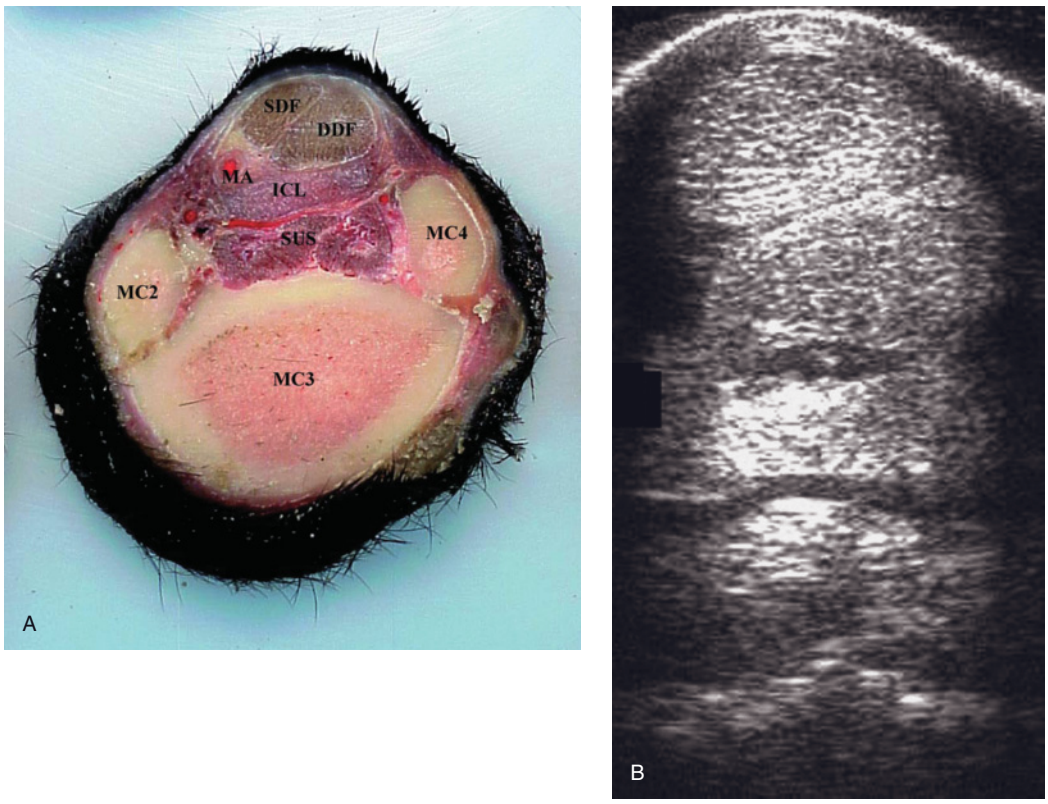
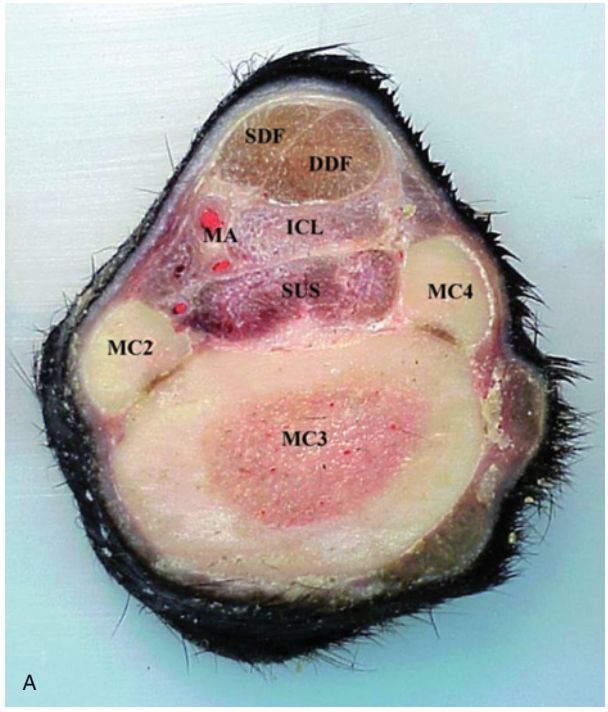



**Figure 4.26.** (A) In Z1B the palmar metacarpal structures appear much as they did in zone 1A. The SDFT appears to flatten palmar to dorsal and the DDFT becomes more rounded. The ICL becomes more inclined toward the DDFT. Fibers of the SL become more pronounced and this structure widens medial to lateral. (B) However, ultrasound imaging of this area has inherent problems with edge artifacts and acoustic enhancement artifacts, which can make evaluation of the SL difficult. Courtesy of Rich Redding.

examinations. Most current machines provide software that allows the image to be traced, which provides a cross-sectional area of the tendon/ligament and lesion. As mentioned earlier, this is critical for the rehabilitation process so that healing can be accurately assessed.

Tendons and ligaments appear similarly as moderately echogenic structures with relatively well-defined margins due to their parallel fascicular arrangement. It is this arrangement of fibers, aligned to resist tensile forces, that creates the intense specular reflections (echoes) seen when the sound beam is perpendicular to the direction of the fascicles. The examiner must pay particular attention to the course of the tendon or ligament being imaged, and maintain the transducer at 90° to the structure. The structures of the metacarpus/metatarsus and the pastern change orientation as they incline distally toward their insertion sites. To perform a complete examination each structure should be evaluated independently with careful attention being paid to correct beam angle and focal zone placement.

The horse should be weight-bearing on the limb and the tendons and ligaments loaded during the ultrasound examination. Imaging while the horse is not bearing weight or only partially bearing weight can create changes in shape and size of these structures, referred to as relaxation artifacts. Relaxation artifacts appear as hypoechoic areas within the normally bright specular reflections seen during routine examination of tendons and ligaments, which can compromise the accuracy of the study. Relaxation artifacts also can occur in some abnormal conditions. For example, complete disruption of the suspensory apparatus relieves the tension in the straight sesamoidean ligament (SSL), resulting in relaxation artifacts in the SSL. This also is apparent when evaluating lacerations or rupture of a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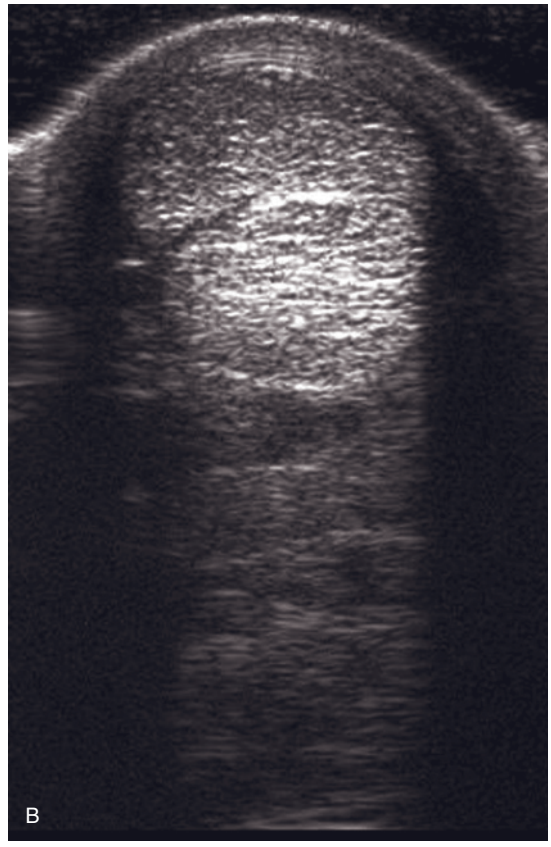
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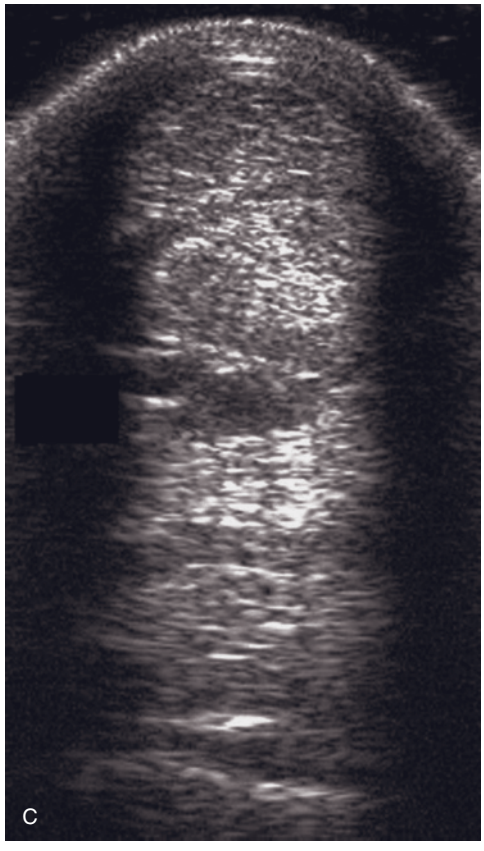
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**Figure 4.27.** In Z2A the ICL narrows palmar to dorsal and the SL increases in area, becoming more discreet and separated from the palmar cortex of MC3. (A) The large metacarpal vessels can be seen medially and laterally. (B and C) These vessels also create edge and acoustic enhancement artifacts, compromising interpretation of the SL architecture. These ultrasound images demonstrate these artifacts and also highlight the importance of focal zone placement. The focal zone in image (B) is placed on the SDFT/DDFT junction with the deeper structures out of focus and poorly imaged, while the focal zone in (C) is placed at the level of the SL. Courtesy of Rich Redding.

tendon/ligament, which relaxes the tensile forces in the affected structures and can create relaxation artifacts most often proximal but also distal to the site of injury.

### *Ultrasonographic Assessment of Tendon/Ligament Pathology*

Tendon and ligament injury is recognized ultrasonographically by changes in size, shape, architecture, position (with respect to surrounding anatomy), and fiber alignment. Cross-sectional area (CSA) measurements are considered a very sensitive indicator of inflammation and the best way to assess increases in size from the transverse images (Figure 4.44). Subtle enlargements of a structure may require comparison to the opposite limb. Any enlargement suggests structural thickening and the rest of the examination should attempt to determine if the change is a result of acute, subacute, or chronic injury.

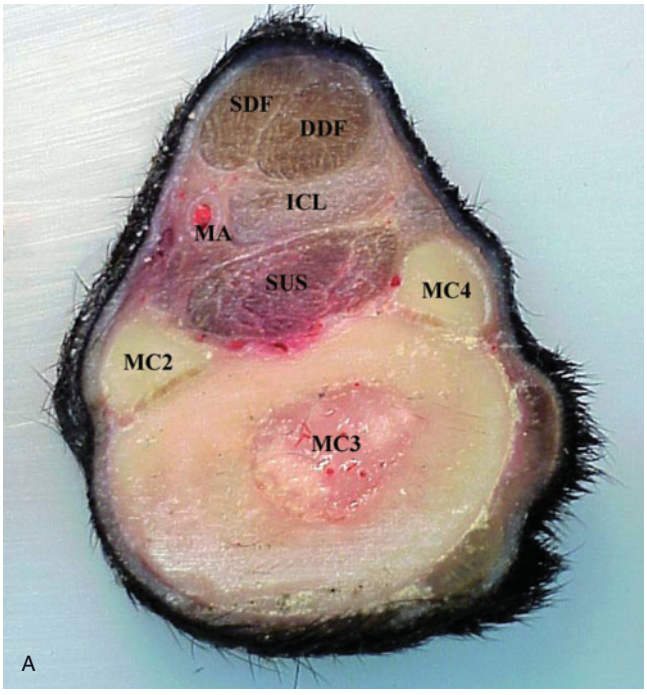
Most current ultrasound machines have the capability to trace the CSA of the frozen image on the screen. Some machines have software that allows stored images to be processed after recovery and storage. Post processing of the image to determine a CSA can be accomplished with digitizing software (DicomWorks), provided the images are stored as DICOM® files; however, this can be quite time consuming. The CSA of the lesion can be compared to the overall tendon/ligament CSA to calculate the proportion of tendon involved. An accurate CSA must be obtained because this measurement is to be used as a baseline in the rehabilitation of tendon and ligaments. CSA may increase for two to three weeks after an injury due to persistent inflammation. A repeat scan at two weeks will provide a more accurate maximum lesion and tendon CSA for comparison during rehabilitation. If normal values for CSA for a structure are not available they can be obtained from the opposite limb at the same level. As the tendon/ligament remodels, the CSA will progressively diminish during the rehabilitation process. Increases in CSA during the rehabilitation period must be carefully assessed and often indicate that a reduction in the level of activity is warranted.

The tendons and ligaments of the distal limb (and to a certain extent the proximal limb as well) have been found to have consistent but unique shapes at each level of the examination. It is normal for these structures to change shape as they course distally in the limb. Therefore, if the examiner perceives a structure to have an abnormal shape it should be compared to the same structure in the opposite limb at the exact same level. It is also helpful in those horses with a change in shape to assess their position with respect to the surrounding anatomy.

Architecture (or texture) is a subjective assessment of the ultrasound image that attempts to describe morphological change or damage. Terms used to describe the architecture of an image relate to the tissue's ultrasonographic intensity. Architectural change is described as a change in echogenicity or the whiteness/brightness of a structure. Echogenicity is a function of each structure's particular density based on several factors, including cellular composition, fiber alignment, and blood supply.

Alterations in echogenicity are subjective interpretations and have been described with the terms isoechoic, anechoic, hypoechoic, and hyperechoic. Isoechoic implies a normal echogenicity, whereas hypoechoic and hyperechoic imply less than and more than isoechoic, respectively. Anechoic implies the structure (or lesion) is mostly black. Fluid is often considered anechoic. In general, the denser the structure, the more echoes it returns and the whiter the structure appears. Alterations in echogenicity reflect changes in cellular and extracellular composition of the tissue. Changes in echogenicity can range from barely perceptible with a mild loss of fiber pattern to complete disruption of fiber pattern from fiber rupture with focal anechoic hemorrhage and/or serous fluid accumulation.

Fibers have a parallel alignment in most normal tendon and ligamentous structures. This parallel fiber bundle alignment is best assessed on longitudinal images. Injury to and inflammation of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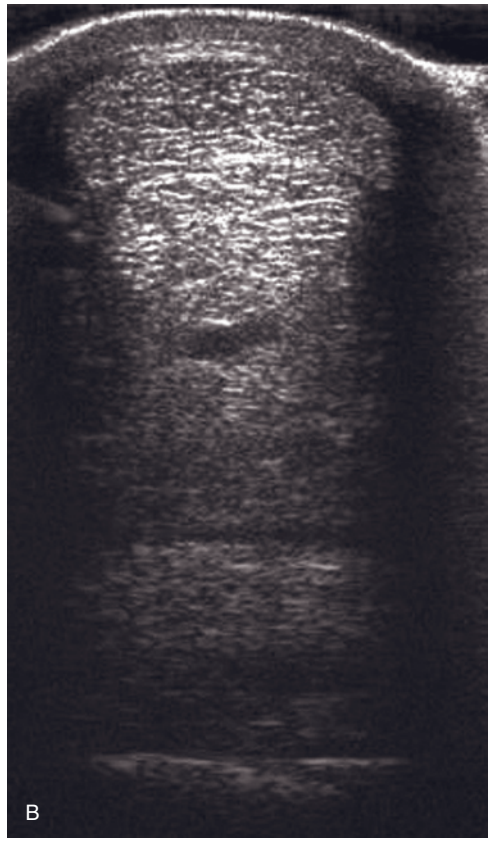
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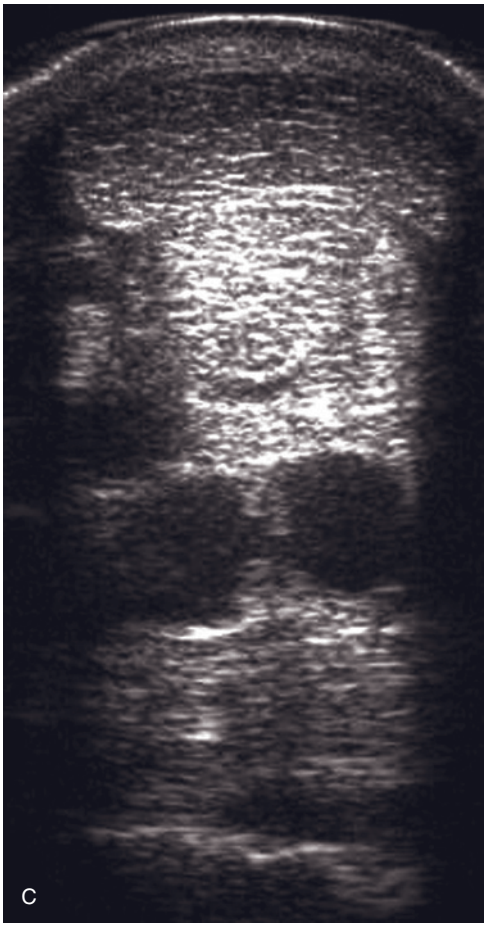
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**Figure 4.28.** (A) In Z2B the SDFT becomes flatter and the ICL remains distinct from, but closely associated with, the DDFT. The metacarpal vessels converge to midline between the ICL and the SL. (B) This ultrasound image with the focal zone placed superficially demonstrates the fibers of the SDFT/DDFT interface, while (C) shows the focal zone at the level of the SL and the convergence of the large metacarpal vessels palmar to the SL. Courtesy of Rich Redding.

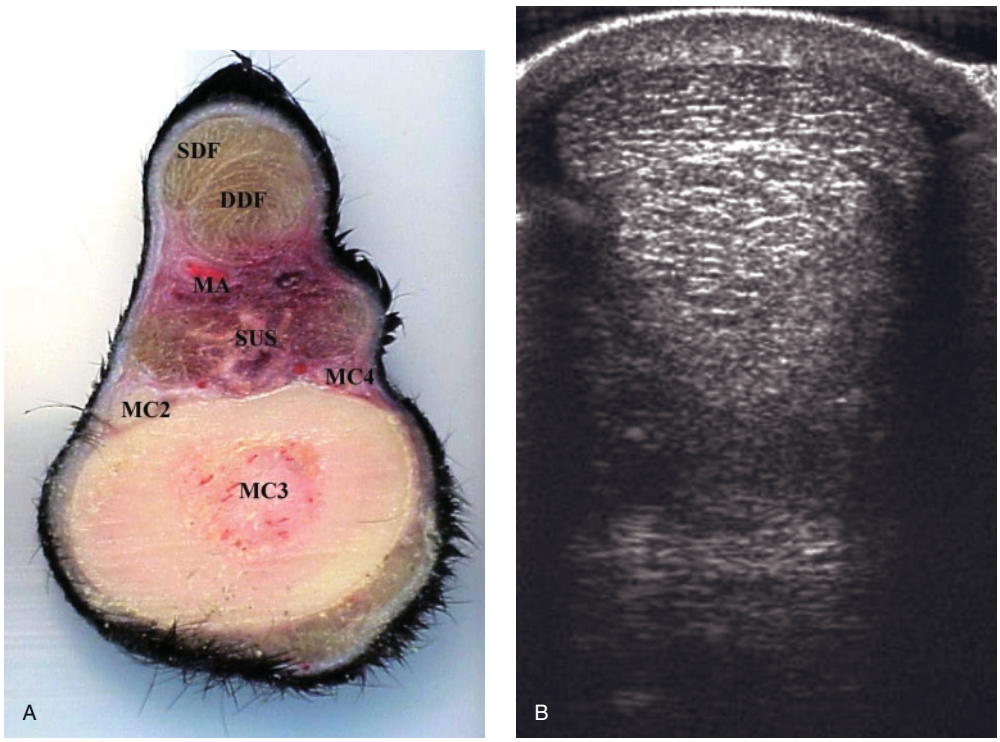



**Figure 4.29.** (A) In the proximal aspect of zone 3A the SDFT thins and begins to expand medial to lateral and appears crescent shaped. The DDFT and ICL are beginning to blend together and can appear as one structure. In the proximal aspect of this zone the SL has not divided and continues to appear as one structure. (B) There may appear to be a hypoechoic area within the central aspect of the SL just prior to the formation of the branches but this should not be confused with a lesion. Courtesy of Rich Redding.

tendons and ligaments can disrupt fiber bundle alignment.

Subtle changes in fiber alignment are best seen on the longitudinal plane images. More severe fiber bundle alignment changes can begin to be appreciated on transverse images. Damage seen on cross section should be confirmed on longitudinal orientation with the longitudinal plane obtained through the affected tissue. Fiber disruption seen as echolucent areas surrounding the fibers is compatible with hemorrhage and edema seen with acute injuries. Nonparallel or random fiber alignment without echolucent fluid content is compatible with chronic injury.

Tendon injury can be focal or generalized such that the distribution of fiber damage can be quite variable. Tendon fiber damage is seen as a continuum of changes ranging from fiber slippage to fiber rupture. Early mild fiber slippage may not be appreciated ultrasonographically; however, mild increases in CSA may indirectly indicate tendinitis. Loss of echogenicity usually indicates fiber disruption but may also reflect edema of the tendon or paratendinous tissue with fluid accumulation.

Acute tendinitis can have a variable appearance based on the severity of conditions. Typically these changes are manifested as decreased echogenicity with increased tendon volume, frequently represented as a rounding of the structure in cross section. Fibroblasts migrate into the damaged area and begin to deposit collagen and

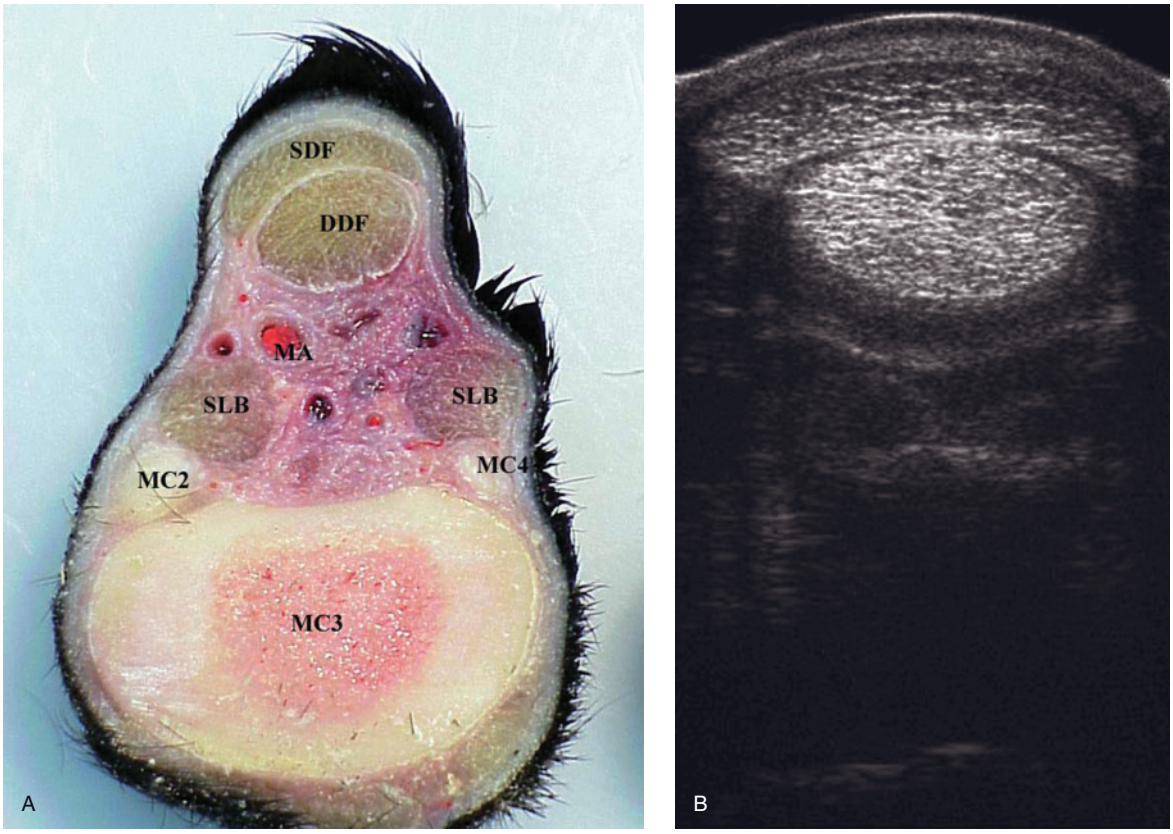



**Figure 4.30.** (A) The distal aspect of Z3A demonstrates the SDFT as elongated in a medial to lateral direction and the division (splitting) of the SL into branches (SLBs). (B) This ultrasound image with the focal zone at the junction of the SDFT and DDFT demonstrates the widening of the SDFT and blending of the fibers of the DDFT and the ICL. Courtesy of Rich Redding.

form granulation tissue. This collagen is laid down randomly and cross links are produced between the fibers. This random disorganized tissue appears hypoechoic on ultrasound and can persist for some time post injury. Rehabilitation with increasing levels of exercise precipitates remodeling of the collagen and a return of the echogenicity and alignment to normal (Figure 4.45).

## *Other Indications for Ultrasonography of the Musculoskeletal System*

### Ultrasonography of Synovial Sheaths

Where there is increased motion or a severe change in direction of a tendon, such as at a joint, the tendon is often surrounded by a synovial sheath *(vagina synovialis tendinis)*. The sheath is lined with synovial cells that produce a synovial-like fluid that facilitates movement of the tendon by minimizing friction and aiding the gliding action of the tendon. The most commonly affected tendon sheaths, in order of occurrence, are: the digital flexor tendon sheath (DFTS), tarsal sheath, carpal sheath, and sheaths of the digital extensor tendons (i.e., extensor carpi radialis) as they cross the dorsal aspect of the carpus and tarsus.

Ultrasound is helpful in differentiating among idiopathic, acute, chronic, and septic tenosynovitis. Most cases of idiopathic tenosynovitis are considered cosmetic blemishes, but ultrasonography is necessary to document normal architecture of the sheath and associated structures. Acute tenosynovitis is usually traumatic in origin. It can be associated with injuries to the tendons within the sheath that can be documented with ultrasound (Figure 4.46). Chronic tenosynovitis often results in persistent effusion

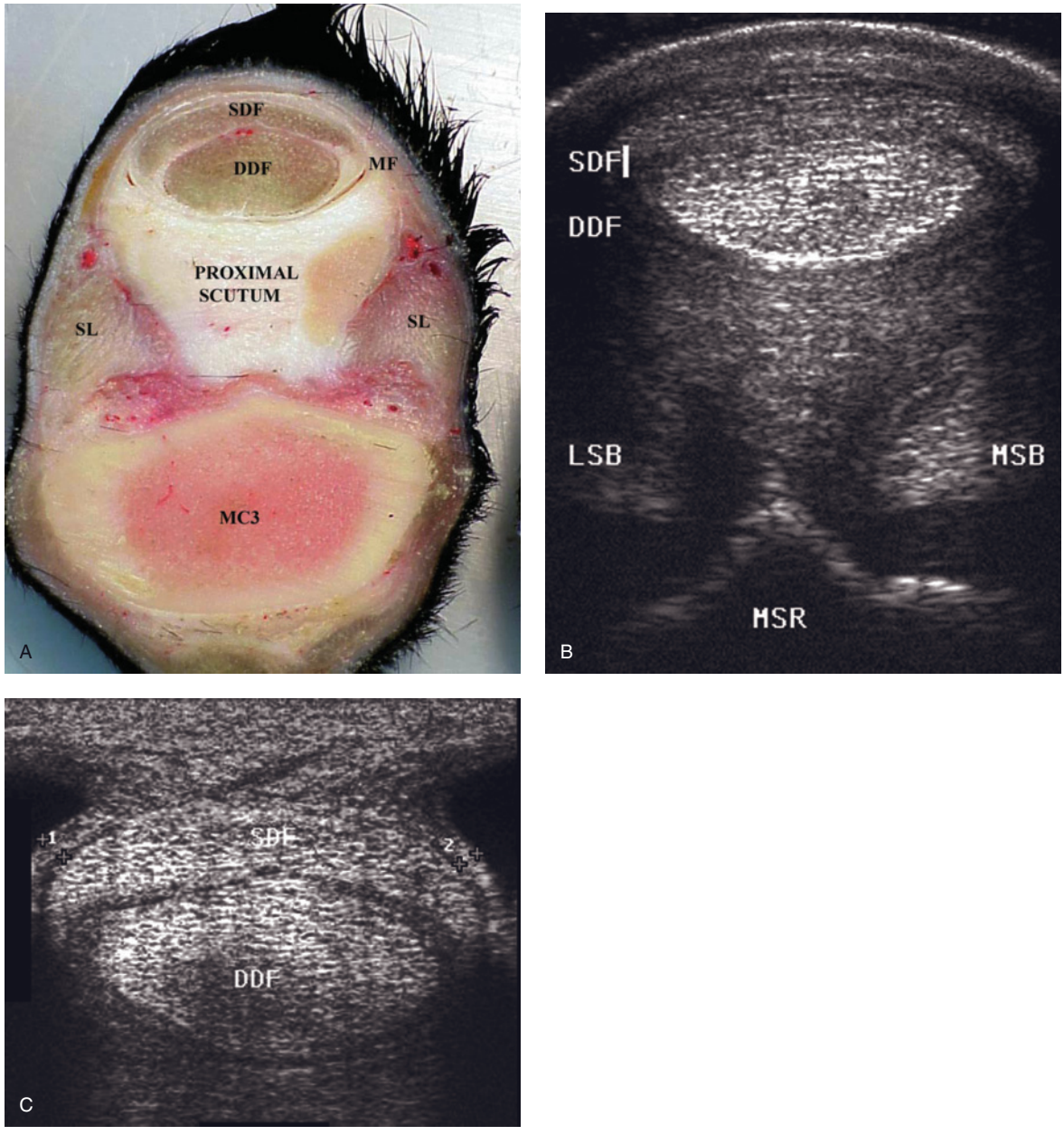



**Figure 4.31.** (A and B) Zone 3B is somewhat longer than the other zones and several important landmarks can be seen in this area. The SDFT and DDFT widen in a medial to lateral direction. These structures can be difficult to image together so each must be examined independently. At the junction of Z3A and 3B after the SL divides into medial and lateral branches, the proximal scutum and palmar aspect of the fetlock joint just proximal to the PSBs can be seen. The proximal aspect of the midsagittal ridge of the palmar metacarpus can be seen. (C) This ultrasound image demonstrates that a moderate amount of effusion within the DFTS allows the natural axial connection of the DFTS to the palmar border of the SDFT proximal to the MCP joint to be visualized. This should not be considered abnormal. Courtesy of Rich Redding.

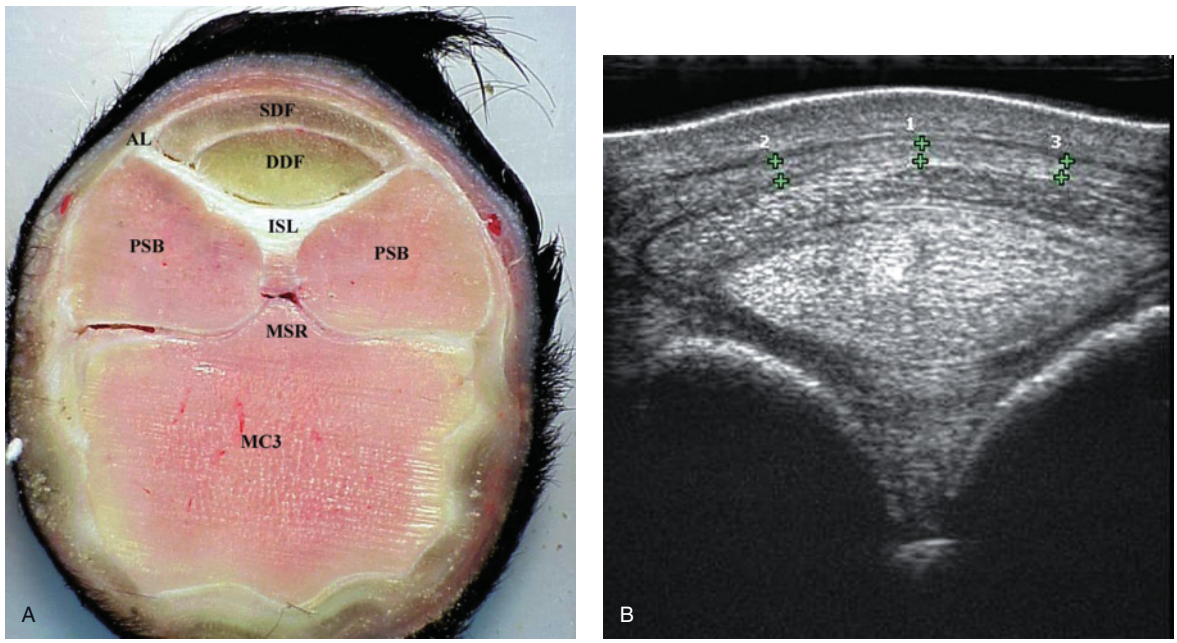



**Figure 4.32.** (A) Zone 3C begins at the level of the PSBs and extends through the fetlock canal. The primary anular ligament (PAL) extends from the palmarolateral to palmaromedial border of the PSBs (abaxial border of each PSB). (B) This ultrasound image demonstrates the PAL wrapping around the SDFT and DDFT. It may be necessary to rotate the transducer in the medial to lateral plane to evaluate the fibers of the PAL. A more abaxial orientation of the transducer also may be necessary to image the attachment to the PSBs. Courtesy of Rich Redding.

and thickening of the fibrous portion of the sheath. Ultrasonographic examination frequently reveals a diffuse proliferative response of the sheath that complicates accurate identification of underlying structural damage. Proliferative nodular masses also may develop within synovial sheaths. Septic tenosynovitis occurs most commonly in the DFTS from punctures, lacerations, and iatrogenic infections. Ultrasonographic examination is not necessary to make a diagnosis of septic tenosynovitis, but can be helpful in finding a pocket of fluid for collection and to document the presence of fibrin within the sheath.

## Evaluation of Bone

Bone appears as a bright hyperechoic line with a strong acoustic shadow. This is due to the high acoustic impedance when compared to the soft tissues. The bone surface appears to be of uniform thickness. Ultrasonography has proven especially useful in the diagnosis of fractures in areas that are not readily accessible to radiographic examination such as the pelvis, femur, scapula, humerus, and spine. Ultrasonography often is used in conjunction with nuclear scintigraphy to focus on areas of bone injury not apparent on radiographs. Fractures can be seen as an anechoic to hypoechoic line that is visible in the cortical bone. With many fractures there is displacement (distraction) of the bone edges that is evident as a "step" in the cortical margin.

Osteitis and osteomyelitis can be evaluated with diagnostic ultrasound; they appear as a fluid interface at the bone surface. Acute trauma may have hemorrhage at the bone surface which can appear similar to osteitis and may indicate more detailed radiographs to rule out a fracture. A repeat scan should be performed in four to five days to document resolution or resorption of the hemorrhage of the fluid interface.

Progression to osteitis is demonstrated by the persistence of fluid, which can vary from hypoechoic to anechoic and may contain hyperechoic echoes consistent with gas in the fluid. Hypoechoic tracts which begin at or just under the skin surface can occasionally be seen tracking to the bone surface. The bone surface may begin to demonstrate a raised area of periosteal new bone production consistent with involucrum formation at the margins of the sequestrum

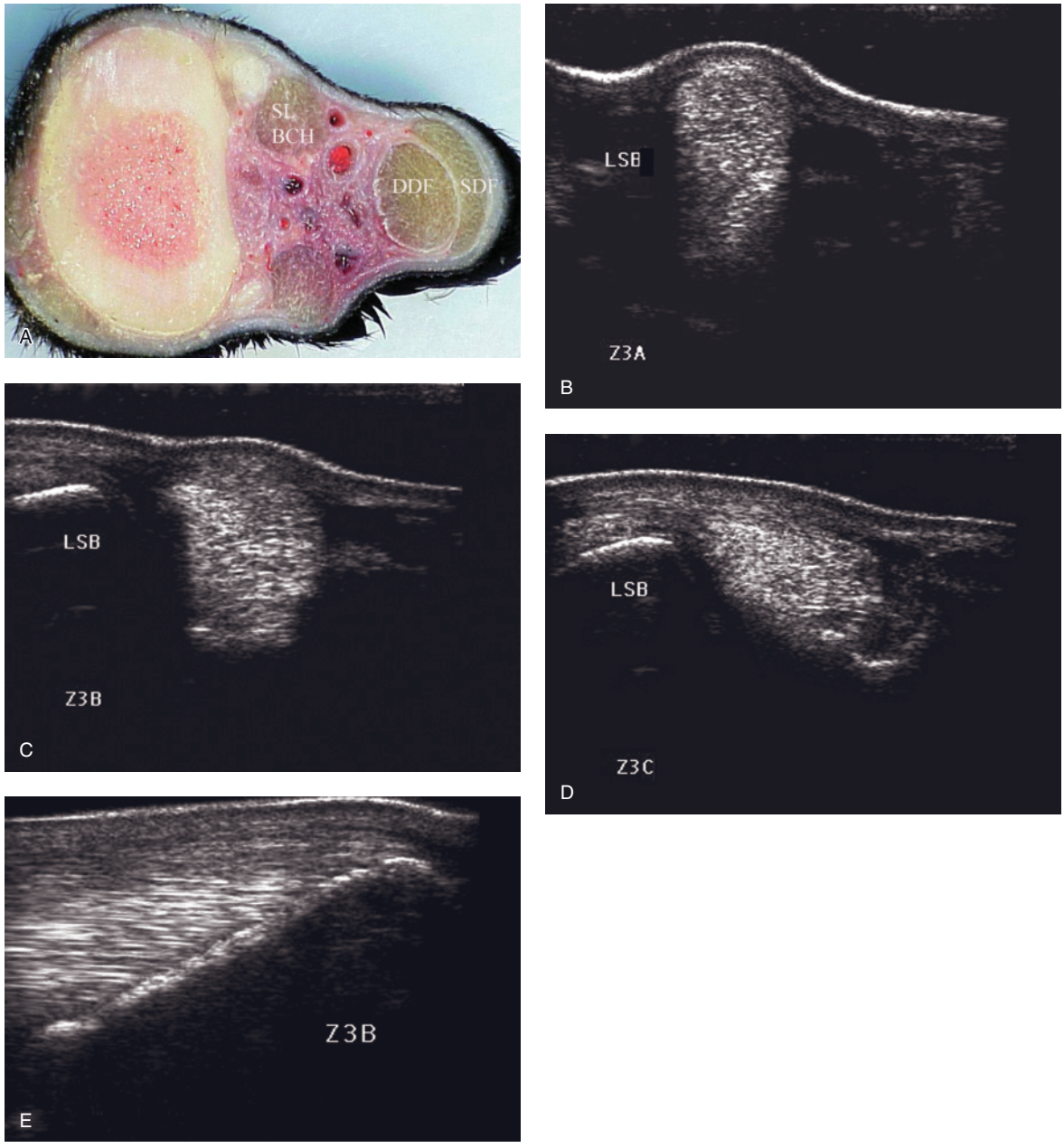



**Figure 4.33.** (A)The branches of the suspensory ligament (SLBs) are best imaged from a medial to lateral and/or lateral to medial orientation. These structures should be evaluated at the level where they begin to divide in distal 3A until their attachments to the PSBs. (B) Initially the SLBs have an oval shape. (C) Further distally the branch becomes somewhat D-shaped. (D) The branch appears to rotate in a dorsal to palmar direction, becoming oval shaped just prior to their attachment onto the PSBs. (E) The SLBs also should be evaluated in the longitudinal plane from their beginning to their attachments to the PSBs. Courtesy of Rich Redding.

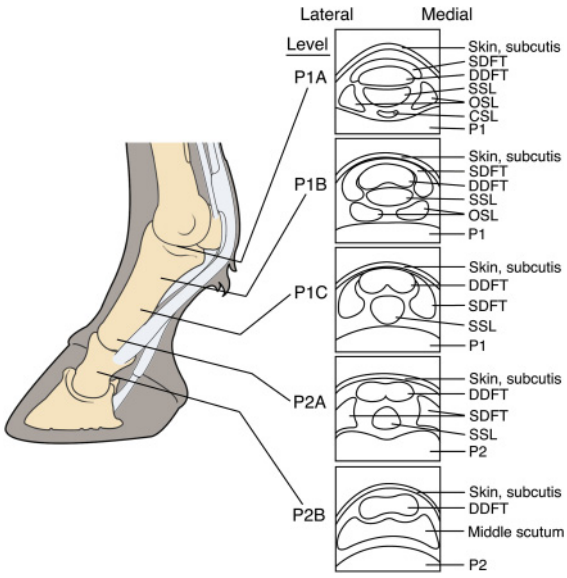



**Figure 4.34.** The SDFT and DDFT continue into the pastern. The DSLs (SSL, paired OSLs, and paired CSLs) originate on the PSBs and course distally to insert on the distal aspects of P1 and P2. The SSL originates from the axial region of the PSBs and crosses the pastern joint to insert on the palmaroproximal aspect of P2. The OSLs originate from the abaxial region of the PSBs and insert on the roughened triangular area of P1. The cruciate sesamoidean ligaments cross from the PSB to the contralateral aspect of palmaroproximal P1. The SDFT encircles the DDFT as they course out of the fetlock canal. The fibers of the SDFT will incline abaxially, moving distally in the pastern to form teardrop shaped branches that insert on the abaxial area of palmaroproximal P2. The DDFT continues distally and becomes bi-lobed in the midpastern area. The DDFT widens in a medial to lateral direction as it courses into the foot to insert on the distal phalanx (P3). Courtesy of Rich Redding.

or bone fragment(s). These periosteal changes can be seen earlier with ultrasound than radiographs. Sequestra appear as hyperechoic structures that cast acoustic shadows (Figure 4.47). Sequestra typically remain adjacent to parent bone and are surrounded by hypoechoic to anechoic fluid. Occasionally the sequestra can be seen displaced from the involucrum and lying in the tract leading to the skin surface. Areas with a predisposition to form sequestra include the metacarpal/metatarsal bone, spine of the scapula, and medial aspect of the radius, but any area with cortical bone that is close to the skin surface may develop a sequestrum.

Ultrasonography is helpful to assess fractures that have been repaired with internal fixation. In the early postoperative period the repair will appear much like acute trauma cases, with hemorrhage and edema surrounding the implants. However, five to seven days postoperatively this fluid interface should begin to become more organized unless there is increased motion or infection of the repair. If this fluid interface persists and the animal manifests systemic signs such as pain, heat, and swelling at the incision site, fever and/or lameness, then infection of the implants, should be suspected.

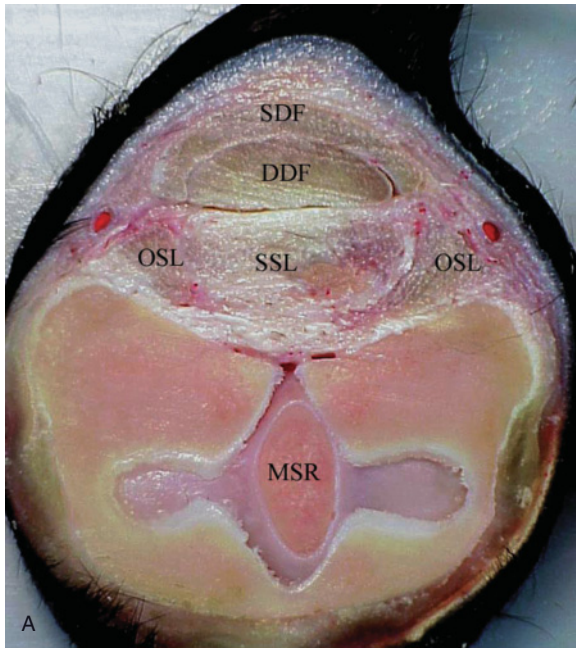
## Evaluation of Punctures and Lacerations

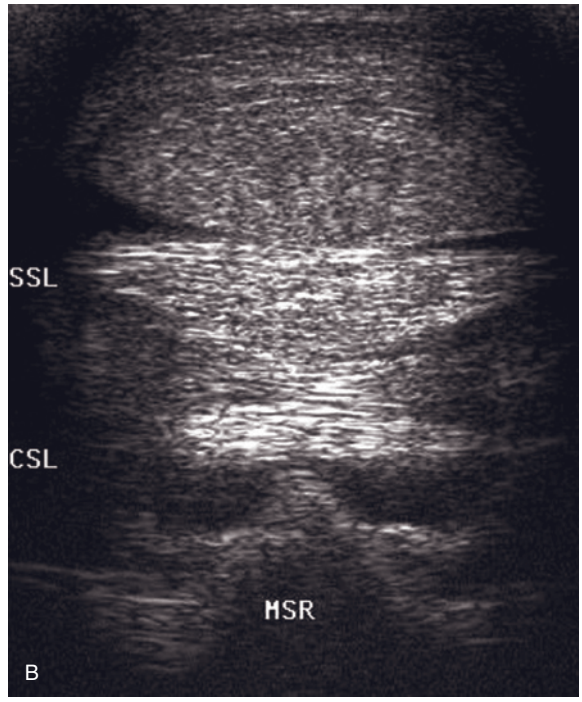
Ultrasonography has proven particularly helpful to define the extent of damage incurred during wounding, either from a puncture or a laceration. Lacerations over the extensor or flexor tendons in the distal extremities require careful examination of the tendons to document involvement and then determine the extent of damage incurred at wounding (Figure 4.48). Also, because these tendinous structures are frequently associated with sheaths and bursa, it is important to determine whether these synovial structures are involved. The wound should have a sterile prep applied to its margins and the wound bed flushed with a balanced electrolyte solution to clean dirt and debris from the wound. Sterile lubricant can be applied to the wound bed and a sterile glove or sheath placed over the probe. The probe can then be placed into the wound to examine the structures deep within the wound bed. Air introduced into the wound may block sound transmission and compromise the study, which can be performed on another day after keeping the wound under a bandage.

Documentation of tendon or sheath involvement significantly changes the management of these types of wounds. Diagnostic ultrasound can be useful to identify synovial distention and assess the character of the synovial fluid. An increase in cellularity and fibrin content in the synovial fluid increases its echogenicity. The presence of gas shadows suggests either an open joint space or the presence of gas-producing organisms in the joint fluid.

## Evaluation of Foreign Bodies

A number of different materials, when introduced into the soft tissues, can create a significant foreign body reaction in the horse. The



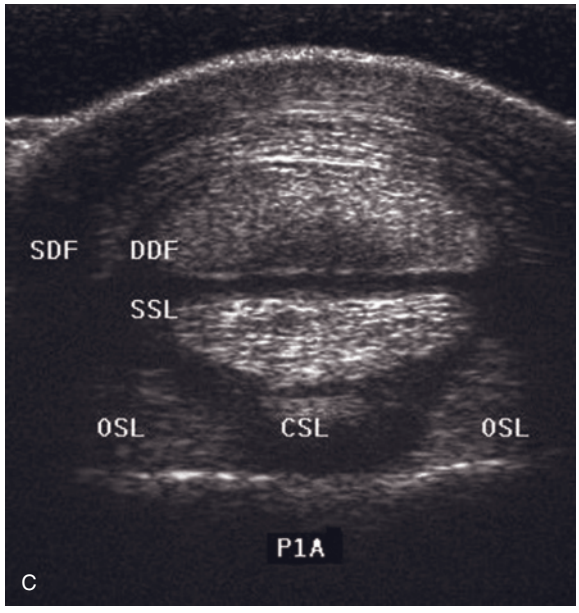




**Figure 4.35.** (A) In ZP1A the contour of the palmar border of the proximal P1 is a V-shaped hyperechoic line. From the midline position the OSLs are positioned on the abaxial surfaces of P1 (palmar tuberosities). The SDFT encircles the oval DDFT with a uniform palmar to dorsal dimension. The SSL is triangular in shape at its origin from the intersesamoidean ligament (ISL) and PSBs. (B) Angling the transducer proximad allows the distal aspect of the midsagittal ridge to be seen. The DDFT is oval at this level while the SSL is triangular at its origin with the cruciate sesamoidean ligament (CSL) seen dorsal to the SSL. (C) At the level of proximal of P1 the DDFT and the SSL are easily imaged but the OSL will require a more abaxial position of the transducer. Courtesy of Rich Redding.

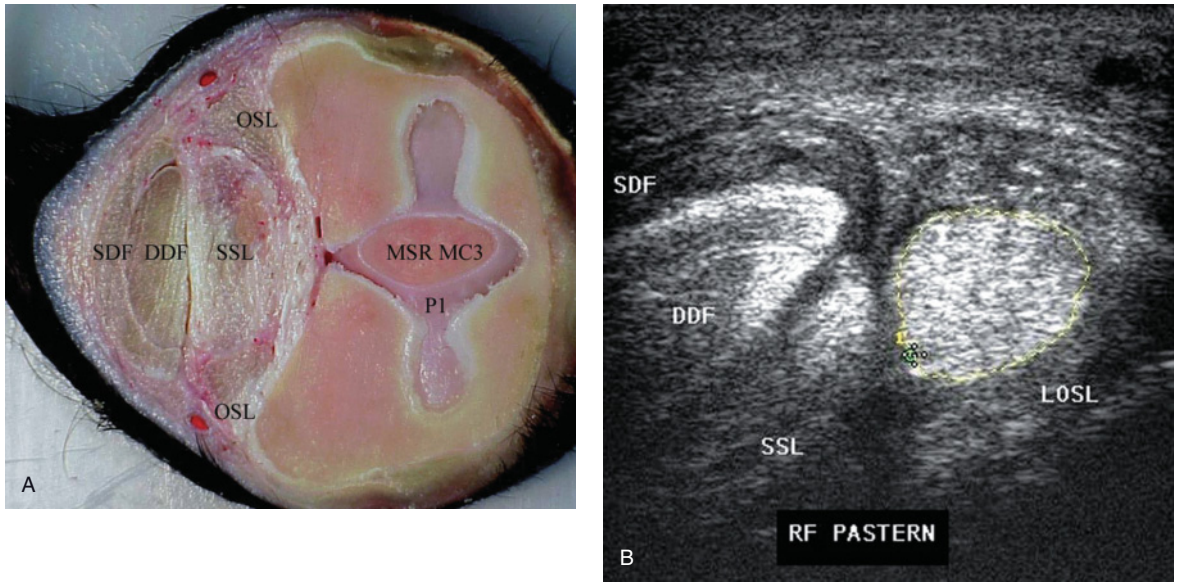



**Figure 4.36.** (A) To best image the origin of the OSLs, the transducer should be placed more abaxially and directed toward the base of the PSBs. (B) In transverse section, the OSLs appear somewhat rounded at this level. The transducer also should be placed longitudinally to image the origin of the OSLs to their respective PSBs. Courtesy of Rich Redding.

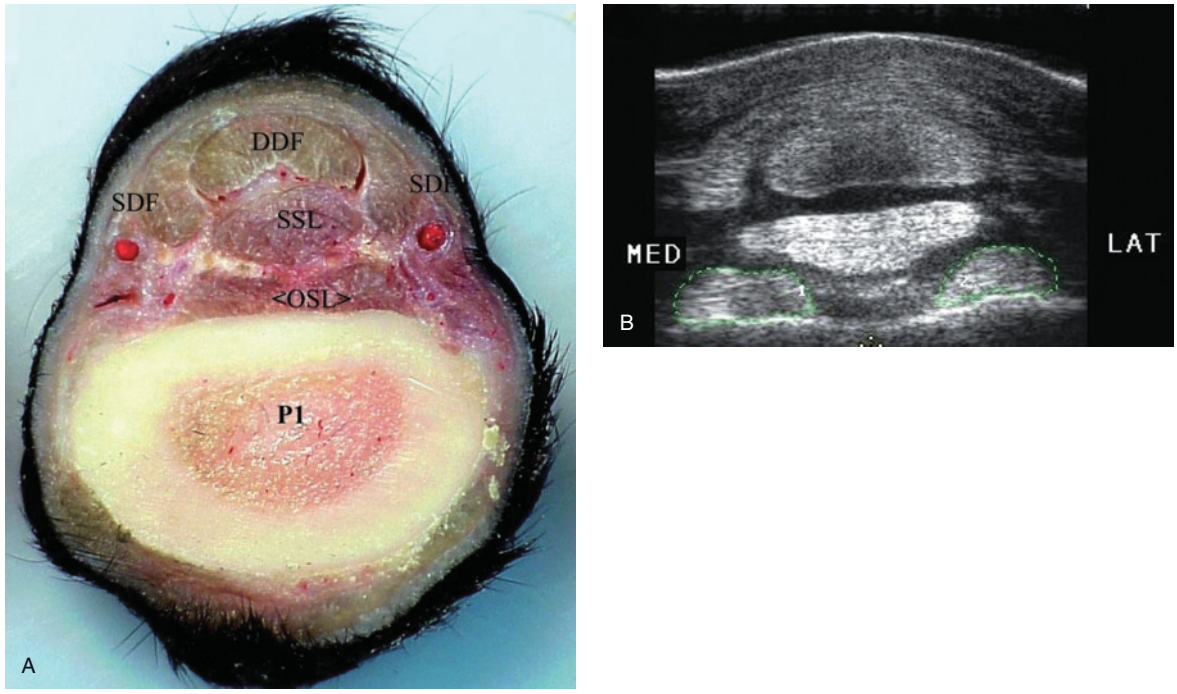



**Figure 4.37.** (A) Zone P1B is considered the middle of P1. The bone contour of P1 is flat at this level. The fibers of the SDFT are inclining abaxially to the medial and lateral aspect of the DDFT as they begin to divide into branches. The DDFT develops a central depression on its dorsal surface and is becoming bi-lobed in appearance. The SSL becomes more rounded in appearance. The OSLs incline more axially on P1, blending together to form a rectangular structure. (B) The ultrasound image shows that the SSL appears hyperechoic relative to other structures. When the SSL is in focus the DDFT is hypoechoic, necessitating that each structure be imaged independently. The SDFT cannot be adequately imaged with this transducer orientation. Courtesy of Rich Redding.

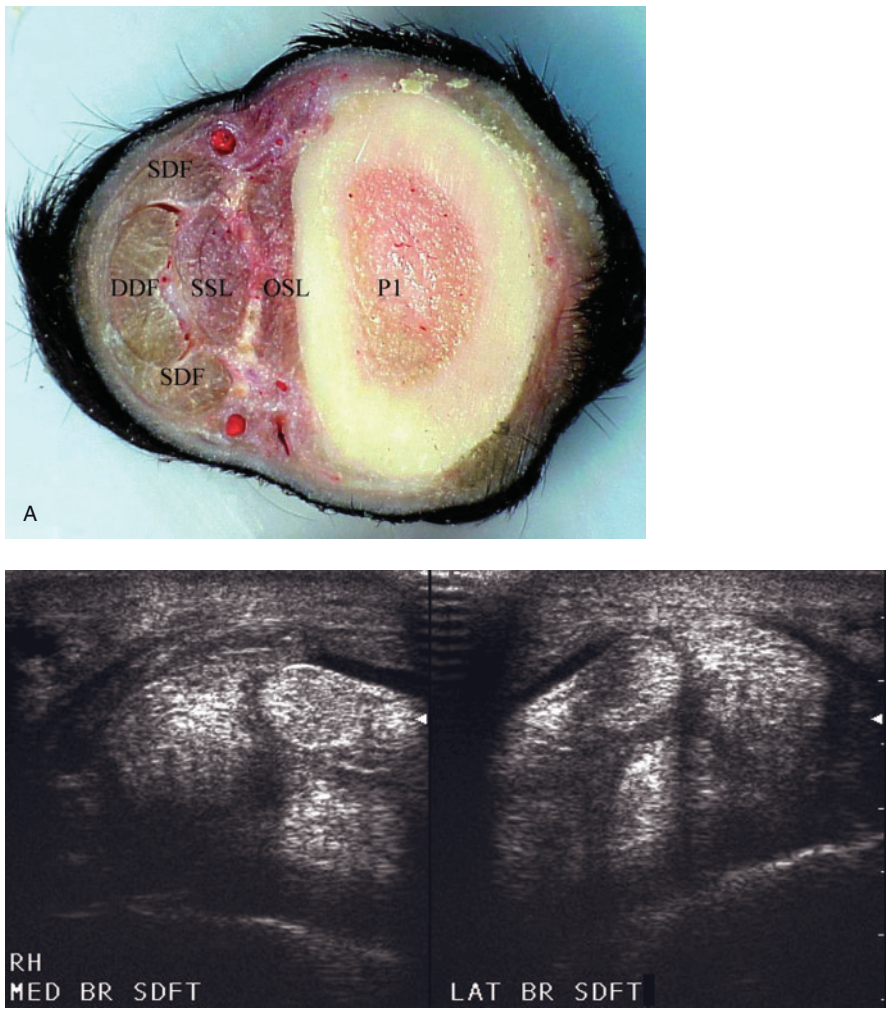



**Figure 4.38.** (A and B) To examine the SDFT branches the transducer must be placed more abaxially, which allows the individual SDFT branches to be imaged as they incline to their insertion onto the medial and lateral aspects of proximal P2. Courtesy of Rich Redding.

most common foreign bodies include wood, lead (bullets or buckshot), metallic objects (such as wire or fencing materials), glass, plant material, hair, and suture material (Figure 4.49). Wood appears as a linear hyperechoic structure that casts a strong acoustic shadow. The most common wood foreign bodies are associated with fencing materials that splinter after penetrating the skin. It is important to carefully evaluate the wounded area for multiple wood splinters before initiating retrieval because air introduced into the wound either at wounding or during surgery can block ultrasound transmission, further limiting the evaluation of tissues deep to it. Bullets and metallic structures can appear to have variable shapes and contours; but, like wood, these objects can cast strong acoustic shadows. Plant material and hair appear to have small hyperechoic shadows that may or may not cast acoustic shadows. This hyperechoic material usually is seen within a hypoechoic tract. Metal such as surgical instruments appear similarly and cast strong acoustic shadows that can assist the clinician when ultrasonographically guided retrieval is used. Placement of an instrument, such as a mosquito forceps around the foreign body, can be seen easily . If retrieval of the foreign body is more complicated, then the area should be mapped out with ultrasonography before retrieval.

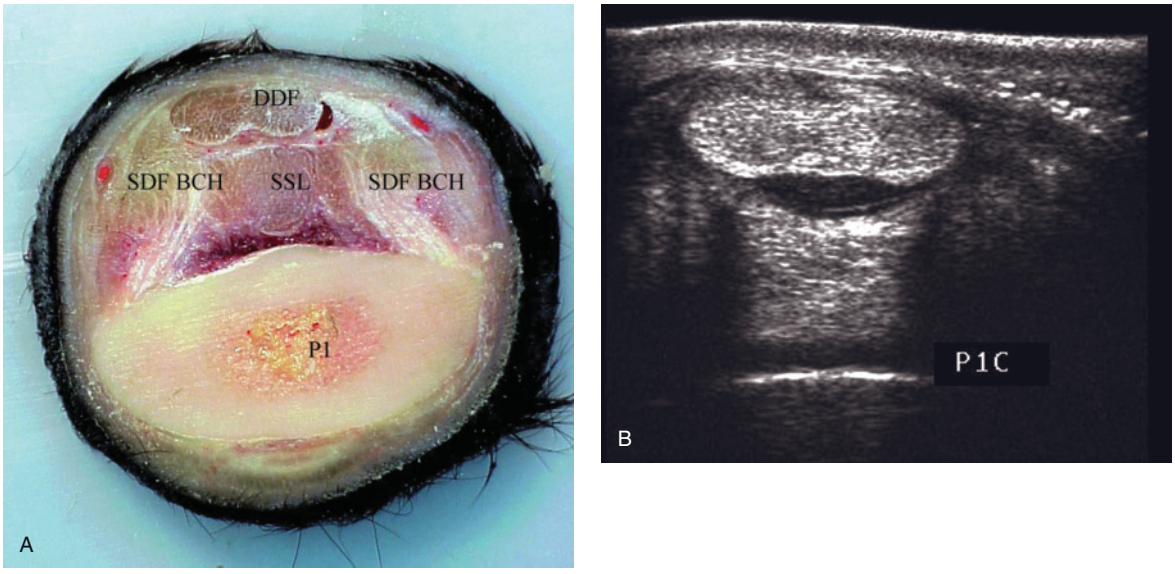



**Figure 4.39.** (A) In ZP1C the DDFT and the SSL are the only structures easily imaged when the transducer is on midline in a palmar position. The DDFT is bi-lobed and now expanding in a medial to lateral direction. The SSL is rounded just prior to insertion onto palmar P2 and the middle scutum. (B) The SDFT branches are more abaxial and difficult to image with this transducer orientation (see Figure 4.38). Courtesy of Rich Redding.

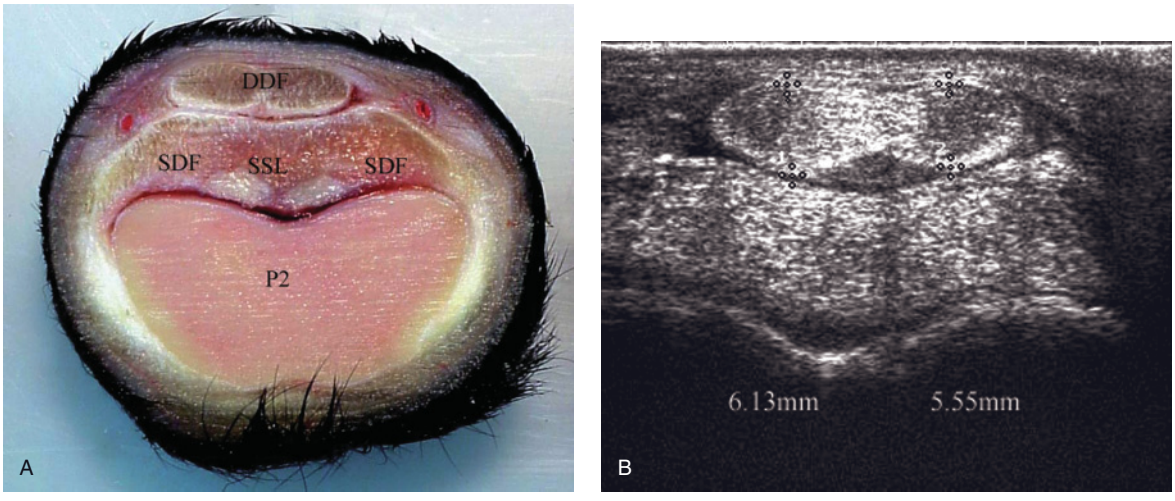



**Figure 4.40.** (A) Zones P2A and P2B are associated with the middle phalanx (P2). Zone P2A begins when the ultrasound examination visualizes the structures of the pastern crossing the PIP joint. The SSL, medial and lateral SDFT branches, and axial and abaxial ligaments of the pastern joint blend together to form the cartilaginous attachment onto P2, called the middle scutum. (B) There is a normal hypechoic area within the SSL just prior to insertion onto P2. Courtesy of Rich Redding.

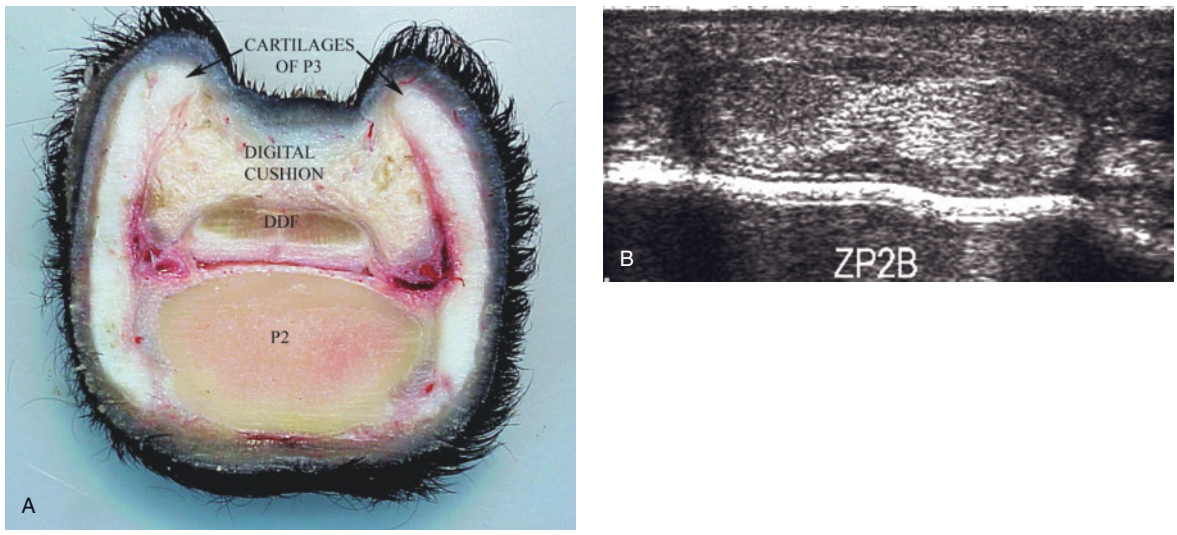



**Figure 4.41.** (A) Ultrasound imaging of Z2PB in the distopalmar aspect of P2 can be difficult due to the presence of the collateral cartilages of the distal phalanx (arrows). (B) The collateral cartilages interfere with proper placement of the transducer distal enough to assess the DDFT with a perpendicular orientation of the sound beam to the fiber pattern of the tendon. Courtesy of Rich Redding.

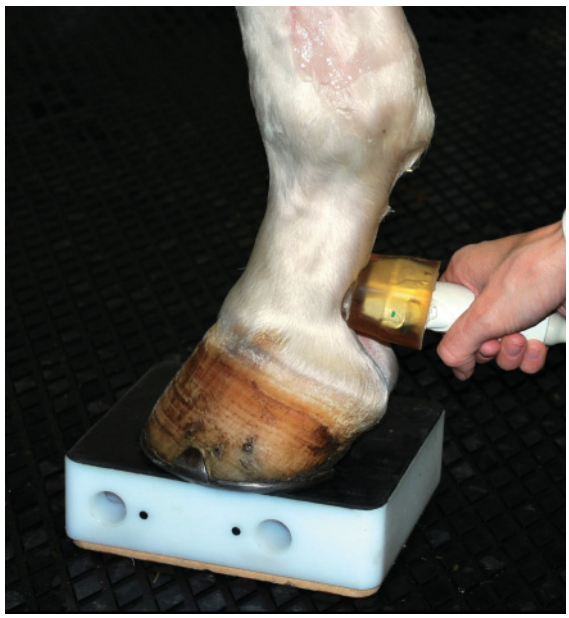


**Figure 4.42.** Placing the limb on a small block with the leg more caudal allows a more comfortable examination of the structures of the pastern region. Notice the stand-off pad placed over the probe to move the near field artifact away from the superficial structures. Courtesy of Rich Redding.

## Evaluation of Joints

Ultrasonography of joints has improved the imaging capabilities of articular surfaces, synovium, IA soft tissue structures such as the menisci in the stifle, and extra-articular structures such as the collateral ligaments. Ultrasound serves to complement the radiographic study in many synovial structures. The MFT joint is probably one of the most common joints on which ultrasound is performed, looking specifically for abnormalities in the medial meniscus (Figure 4.50). However, ultrasound can be used in most every joint, depending on the presenting problem. See Chapter 4 in *Adams and Stashak's Lameness in Horses, Sixth Edition*, for a detailed description of joint ultrasonography.

### *Limitations of Ultrasonography*

Ultrasonography has many limitations that must be recognized. The quality of the image is directly related to the operator, the equipment, and the anatomical area being examined. This imaging tool is influenced by the skill of the operator more than any other imaging technique. The operator is responsible for positioning and steering the sound beam as well as determining the equipment settings during image acquisition. Artifacts are easily produced and can create inaccuracies in the image, which can

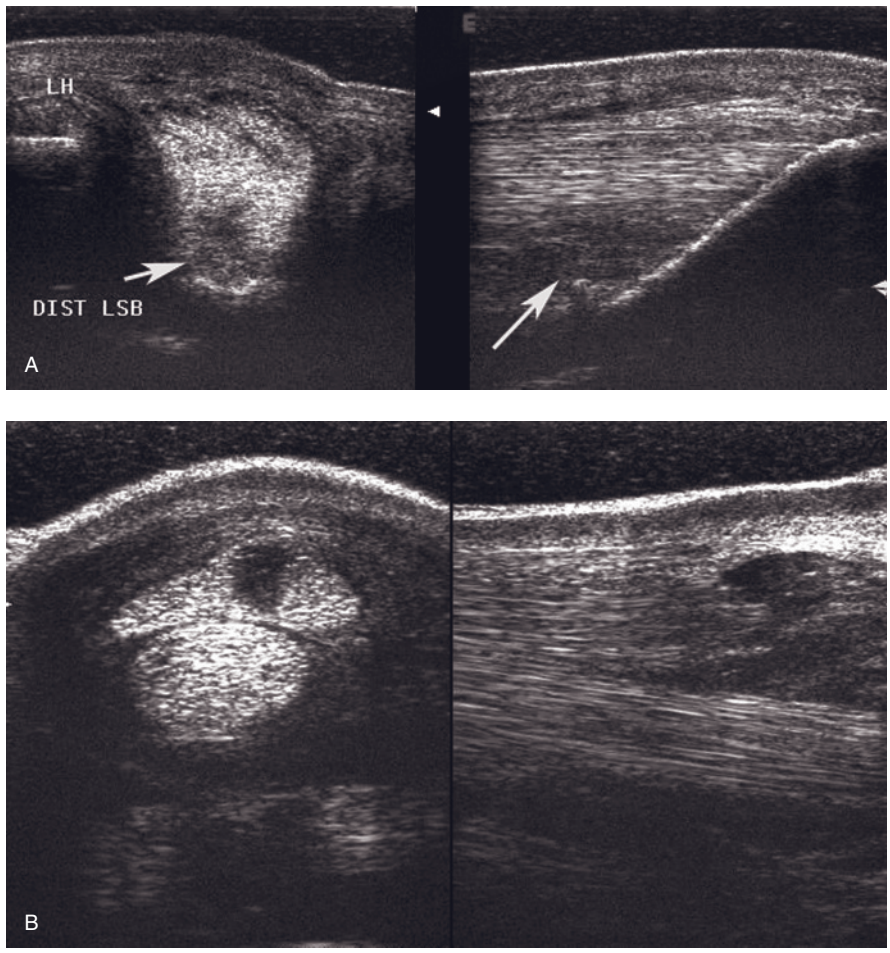



**Figure 4.43.** (A) Longitudinal and cross-sectional ultrasound image of a suspensory ligament attachment injury. Notice the loss of fiber pattern and the calcification at the attachment to the proximal sesamoid bone on the transverse and longitudinal sections. (B) Longitudinal and cross-sectional ultrasound image of the SDFT with a core type defect of the central aspect of the tendon. Courtesy of Rich Redding.

significantly compromise interpretation. Artifacts most often involve operator error and an assortment of sound-tissue interactions that may or may not be controllable.

One common but easily correctible artifact is created by inadequate skin preparation, which leads to poor transmission of sound and a corresponding dark image. High-frequency transducers produce better images but often require shaving the area to be examined with a razor. Improving skin-transducer contact is critical to obtaining the best images possible. Another common artifact due to operator error occurs when the ultrasound beam is off incidence to tissue interfaces and tendinous structures. Off-incidence artifact occurs when the ultrasound beam is not at 90° to the fibers of the target structure, which reflect the returning echoes away from the transducer. This creates a hypoechoic area that mimics a lesion(s) within the targeted structure. Improper gain and power settings and inappropriate focal zone position also can lead to suboptimal images. Near gain and power settings that are set too high reduce the ability to differentiate the tissues. Gain settings should be adjusted to produce a uniform gray scale across the entire image. Focal zones are variable in number and position and should be adjusted to the level of the specific structure(s) to optimize image quality.

Despite these limitations, ultrasound remains a practical, inexpensive, and readily accessible

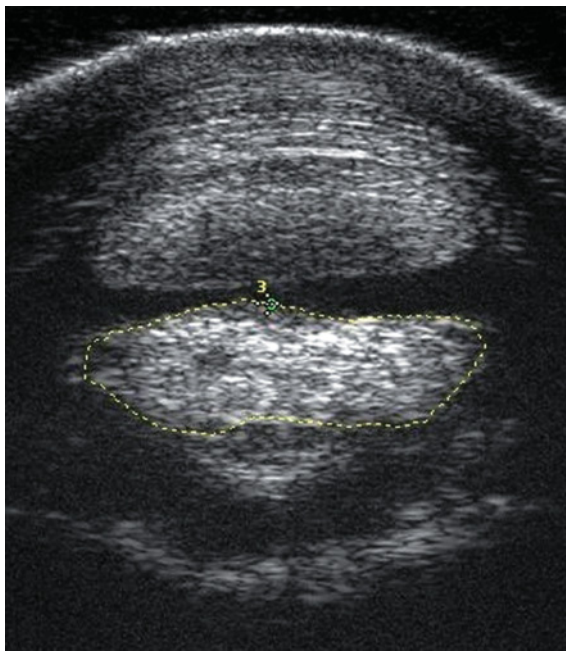


**Figure 4.44.** This is a straight sesamoidean ligament (SSL) injury. There is enlargement with an irregular outline associated with the heterogeneous appearance of the ligament. Tracing of the cross-sectional area is helpful to follow the rehabilitation process. Tendon and ligament damage is represented by changes in size, shape, architecture, position (with respect to surrounding anatomy), and fiber alignment. Courtesy of Rich Redding.

imaging technique for soft tissue injuries of the horse. However, with the recent introduction of magnetic resonance imaging (MRI) to equine musculoskeletal imaging, soft tissue and bone injuries can be evaluated in detail not provided by any other imaging technique. MRI is now considered the gold standard to assess lameness originating from the carpus and tarsus distally (especially of the foot within the horny hoof capsule), but it is not always practical or necessary to make a diagnosis. Lesion(s) seen with standard ultrasonographic imaging typically are not candidates for MRI examination.

While ultrasonography and MRI remain the optimal choices for soft tissue imaging, it remains unclear in all cases what is the most effective imaging tool. In humans, many studies have been performed that compare the diagnostic accuracy and utility of ultrasound to MRI for a variety of orthopedic problems. These studies are lacking in horses and need to be performed. A limited number of studies have been performed and have looked at the collateral ligaments of the DIP joint and the proximal plantar metatarsal region. In those studies, a negative ultrasound exam did not rule out an abnormality and positive ultrasonographic findings should be regarded with caution. It is hoped that further experience comparing ultrasound with MRI, along with the completion of more comparative studies between MRI and ultrasound, will identify specific indications for each imaging modality.

## NUCLEAR MEDICINE

Radiography, ultrasound, computerized tomography (CT), and magnetic resonance imaging produce images that reveal anatomic detail. Nuclear medicine techniques, on the other hand, image the blood flow to bone as well as the function or the physiological activity of bone. Nuclear medicine imaging is a very sensitive tool that augments, but does not replace, the basic lameness examination. Most academic institutions and several private clinics have nuclear medicine imaging facilities, making this modality available to many equine practitioners. This section discusses the principles, techniques, and indications for nuclear medicine imaging in the evaluation of the musculoskeletal system of horses.

### *Principles of Nuclear Medicine*

Nuclear medicine imaging, also known as scintigraphy, is based on the functional distribution of a radio-pharmaceutical in the body. The radiopharmaceutical is made of a radionuclide, most commonly technetium-99m ($^{99m}$Tc). It is labeled to a pharmaceutical which determines the target tissue of the radio-pharmaceutical in the body. $^{99m}$Tc decays by emitting a 140-kEv γ-ray. A γ-ray is identical to an X-ray, except that it originates from the nucleus of an unstable atom ($^{99m}$Tc in this case) as the atom strives toward a more stable state. Nuclear medicine imaging also can be described as an emission imaging technique because the image is made by γ-rays that are being emitted by the $^{99m}$Tc inside the horse. Radiography is considered a transmission imaging technique because the X-rays that produce the image are transmitted through the patient. $^{99m}$Tc can be produced on site using a molybdenum-99m generator, or it can be ordered from a nuclear pharmacy when needed. $^{99m}$Tc has a relatively short natural half-life ($T_{1/2}$) of 6 hours; for example, 100 mCi of $^{99m}$Tc will decay to 50 mCi in 6 hours, or, as an example, an

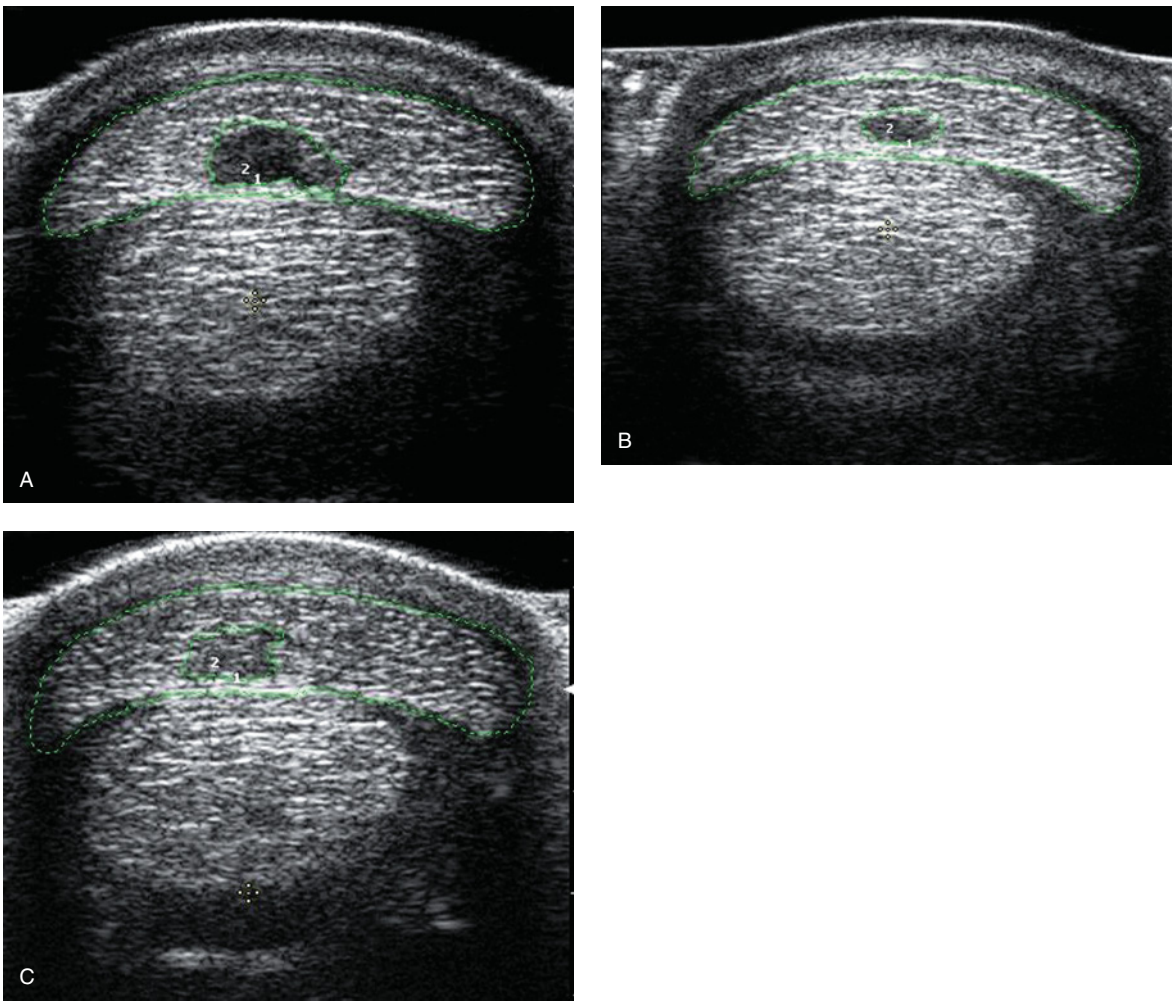



**Figure 4.45.** (A to C) Sequential scans acquired over 12 weeks (four, eight, and 12 weeks) of an iatrogenic tendon lesion created in the central aspect of the SDFT. Acute tendinitis initially appears as a decrease in echogenicity with corresponding increased tendon volume (cross-sectional area) which is frequently represented as a rounding of the structure. Early in the repair process cellular infiltration with fibroblasts and vascular cells fill the damaged area and begin to deposit collagen and form granulation tissue, which can increase the echogenicity of the lesion. Collagen is laid down randomly and cross-links are produced between the fibers. Random disorganized tissue may appear hypoechoic on ultrasound and can persist for some time post injury (into the remodeling phase of healing). Rehabilitation with increasing levels of exercise precipitates remodeling of the collagen and a return of the echogenicity and alignment to normal. The lesion size and cross-sectional area tend to progressively decrease over time. Courtesy of Rich Redding.

exposure rate of 4 mrem/hour (0.04 millisievert-[mSv]) will decrease to 2 mrem/hour (0.02 mSv) in 6 hours. However, the effective $T_{1/2}$ of a radiopharmaceutical is generally shorter than the natural $T_{1/2}$ due to biological excretion of the tracer.

The pharmaceutical portion of the radiopharmaceutical determines the distribution of the tracer radionuclide in the body. Various molecules or cells can be labeled. Red blood cells can be labeled for the evaluation of the circulating blood compartment, most commonly done to study cardiac function. Intra-venous administration of Tc-99m-pertechnetate ($^{99m}Tc0_4$) or Tc-99m-labeled red blood cells ($^{99m}$TcRBCs) are scintigraphic techniques for looking at the perfusion (blood flow) of soft tissue structures such as the joints of the distal limbs. White blood cells

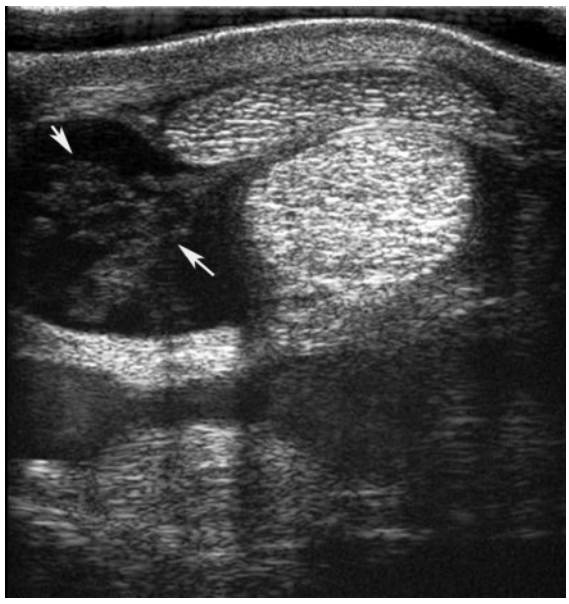


**Figure 4.46.** This horse developed an acute tenosynovitis of the DFTS following a traumatic injury to the distal limb. Ultrasound examination is indicated in acute tenosynovitis cases due to the potential for several structures to be affected, all of which can instigate an intense and prolonged inflammatory response. In this case there is a large clot within the DFTS adjacent to the DDFT (arrows) but little evidence of structural damage to the flexor tendons or DSLs. Courtesy of Rich Redding.

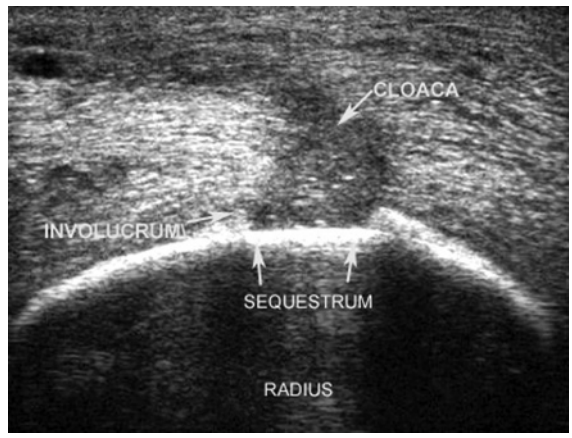



**Figure 4.47.** This horse had a draining tract of the medial radius. Radiographs were suggestive but not conclusive for a sequestrum but ultrasonography proved to be diagnostic. There are small gas shadows within the middle of the tract (cloaca) which courses from the surface of the radius to the skin. New bone is being deposited at the periphery of the sequestrum. The margins of the sequestrum have raised periosteal new bone production consistent with involucrum formation. Sequestra appear as hyperechoic structures that cast acoustic shadows. These periosteal changes can be seen earlier with ultrasound than radiographs. Courtesy of Rich Redding.

can be selectively labeled with $^{99m}$Tc-HMPAO to look for areas of active inflammation/infection. $^{99m}$Tc-labeled biotin ($^{99m}$TcEB1) also has been used to detect soft tissue inflammation in horses. Although each of these techniques uses $^{99m}$Tc, the distribution of the radiopharmaceutical will vary, based on the biokinetics of the pharmaceutical or cell to which the $^{99m}$Tc has been labeled.

Bone scans are done using radiolabelled polyphosphanates that have a high affinity for the Ca-hydroxy-appetite molecules in bone. Images made at 2 to 4 hours post injection are a representation of the uptake pattern in the bones. A very predictable uptake pattern is seen in normal animals, and increased radiopharmaceutical uptake (IRU) is seen with increased blood flow or increased osteoblastic activity. Either $^{99m}$Tc-oxidronate (HDP) or $^{99m}$Tc-methylene diphosphanate (MDP) is administered intravenously at a dose of about 0.35 mCi/kg bodyweight. $^{99m}$Tc-HDP has the advantage of faster soft tissue clearance, thus allowing image acquisition to begin sooner after injection. A second advantage of $^{99m}$Tc-HDP is improved visualization of bones surrounded by large amounts of muscle, (e.g., spine, pelvis, and hips). An average 450-kg horse will receive about 160 mCi (5.92 GBq) of the radiolabel. The dose rate can be adjusted for age (i.e., increased by about 10% in older patients and decreased by about 10% in juveniles) because of the difference in metabolic activity of bone tissue. Approximately 50% of the injected radiolabel is excreted in the urine, which results in the effective $T_{1/2}$ being shorter than the natural $T_{1/2}$.

A nuclear medicine evaluation of the musculoskeletal system may consist of three phases. Phase 1, known as the blood flow or vascular phase, represents the radiopharmaceutical in the blood vessels before diffusion into the extracellular fluid. It lasts for one or two minutes after injection. The body region to be evaluated must be positioned in front of the gamma camera at the time of injection, and dynamic rapid frame acquisition is made as the radiolabel perfuses the vasculature. Multiple images are acquired over the first few minutes while the radiolabel is within the vascular system, before diffusion into the extravascular space occurs. The vascular

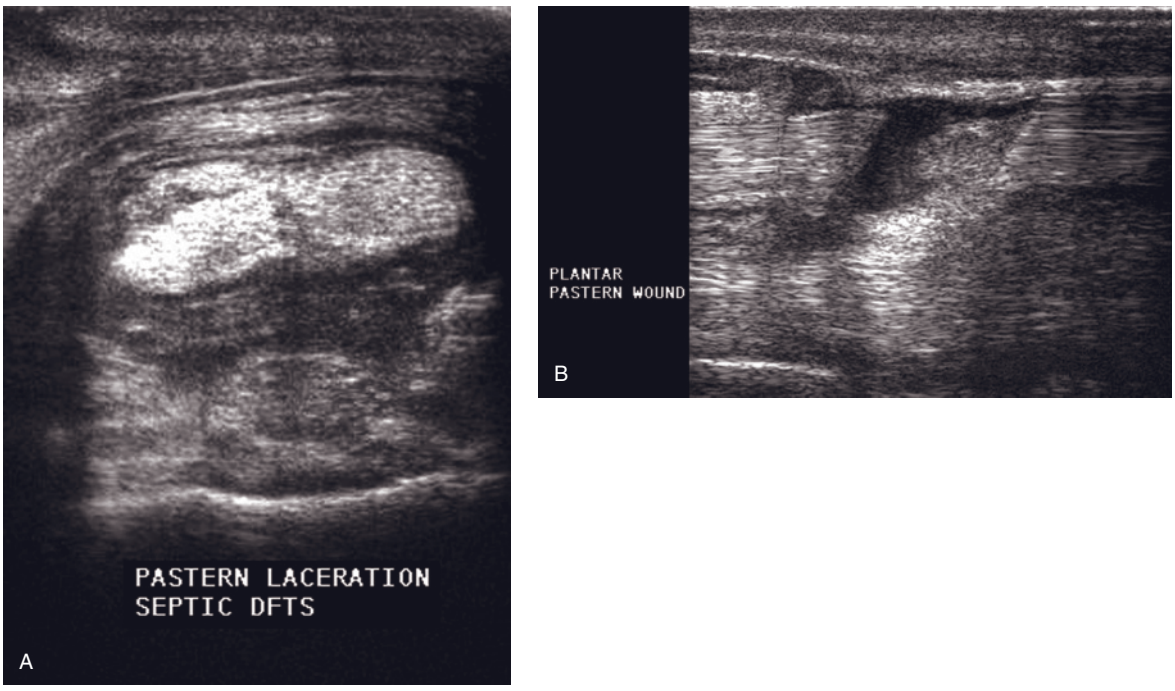



**Figure 4.48.** Lacerations over the extensor or flexor tendons of the distal extremities require careful examination of the tendons and any associated sheaths/bursas to determine the extent of damage incurred at wounding. (A) Transverse ultrasound image of a DDFT injury in the pastern. There is a proliferative response within the DFTS that suggests an active inflammatory process. Due to the penetrating injury, sepsis is likely. (B) Longitudinal ultrasound image of a laceration which demonstrates a severe DDFT injury. There is complete transaction of the DDFT with retraction of the proximal stump of the tendon. Courtesy of Rich Redding.

phase is used to compare the blood flow, especially to the distal limbs (e.g., in cases of degloving injuries), but also can be used to document perfusion deficits in different anatomical regions.

Phase 2, known as the pool or soft tissue phase, represents the radio-pharmaceutical distribution in the extracellular fluid. It is visualized from three to approximately 10 minutes post injection. This phase is used to evaluate blood flow to soft tissues. An increased signal will be observed with hyperemia due to edema, inflammation, etc. Increased radioactivity during the pool or soft tissue phase is best used in the distal limbs, and has been associated with navicular syndrome as well as inflamed joints and tendinitis or desmitis. Early intense bone uptake of the radiopharmaceutical ($^{99m}$Tc-HDP or $^{99m}$Tc-MDP) can sometimes be seen as soon as 5 minutes after injection, especially in cases of intense delayed phase bone uptake (e.g., fractures or infectious processes). This can sometimes make the evaluation of soft tissues challenging at best, if not impossible. A scintigraphic technique for looking at soft tissues only, without any possibility of bone uptake, is the use of $^{99m}$Tc-$O_4$ (pertechnetate, unlabeled to a pharmaceutical) administered IV with images made as soon as radiopharmaceutical equilibrium in the extracellular space is achieved. A similar dose to that of the bone scanning agents is recommended. $^{99m}$Tc-RBC is another alternative for evaluating blood perfusion to soft tissues without the risk of bone uptake overlap.

Phase 3, known as delayed or bone phase, occurs several hours later, when approximately 50% of the injected radiopharmaceutical has attached to bone. The remainder of the tracer is excreted by the kidneys in the first one or two urine voids post injection. The uptake pattern of normal bone is quite predictable and must be recognized for accurate interpretation of scintigrams. The diaphysis of long bones has the least uptake, and greatest uptake of the tracer occurs in the juxtaphyseal and subchondral bone in normal subjects. Increased uptake by or near the joints during the delayed (bone) phase has been related to OA (Figure 4.51), various enthesiopathies, and a variety of other pathological conditions.

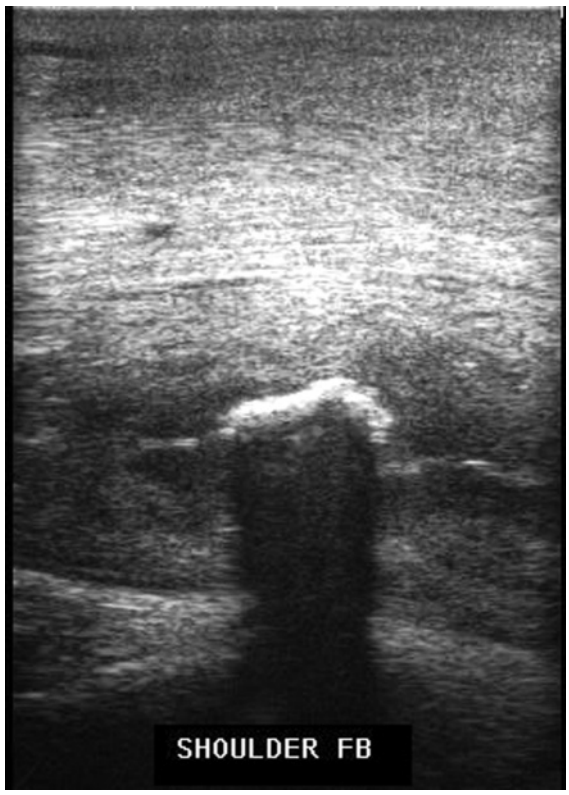



**Figure 4.49.** This horse had a draining tract at the shoulder region. There appears to be a dense foreign body several centimeters deep, consistent with a bullet. The surgical approach to the foreign body was mapped out with ultrasonography intra-operatively. Courtesy of Rich Redding.

## *Imaging Equipment*

The gamma camera contains a collimator made of small holes in a lead plate that allows only perpendicular γ-rays through (Figure 4.52). This reduces scatter, thereby improving image resolution. The γ-rays interact with a fluorescent crystal (a thallium-activated sodium iodide crystal is commonly used), changing the γ-energy to light photons. The light photons interact with a photocathode, generating electrons which are amplified by an array of photomultiplier tubes. The XY co-ordinates of the electrons are then recorded and the image is reconstructed. Thus, the image represents the geographic distribution of the radiopharmaceutical in the horse. Images are acquired in a 256-×-256 matrix to optimize image resolution without requiring excessive computer storage capabilities.

**Table 4.1.** Minimum image acquisition counts.

| Body Region | Number of counts × 1,000 |
|---|---|
| Foot | 100–150 |
| Carpus | 100–150 |
| Elbow | 150–200 |
| Shoulder | 200–300 |
| Tarsus | 150–200 |
| Stifle | 150–200 |
| Sacroiliac area | 200–300 |
| Spine | 200–300 |
| Soft tissue (pool) phase image | 75–100 |

Various techniques have been devised to suspend the gamma camera, including stationary systems such as forklifts or hydraulic systems, or systems that are available to move anywhere around the nuclear medicine suite such as track-and-hoist-, or track-and-column-mounted detectors (Figure 4.53). The advantage of the track systems is that the horse can be positioned anywhere in the room and the detector moved around it to obtain the different views. The gamma camera and collimator weigh approximately 1,500 lbs (680 kg) and must be kept very still for the acquisition time of about 30 to 90 seconds. The gamma camera computer acquires the data and reconstructs the images, and then sends the digitized images to the processing computer, where they are processed and stored. With recent advances in technology, DICOM® images can be produced with most software products and sent to a computer station with a picture archiving and communication system (PACS) for reviewing and storage.

Image acquisition is determined by the number of counts or the acquisition time. The number of counts/image is the *most* critical factor in terms of image quality. Although a certain minimum number of counts is needed for a diagnostic image, more counts result in a superior image. Table 4.1 offers suggestions for minimum counts/image. A longer image acquisition time is needed for more counts/image, although at some stage a long image acquisition time becomes impractical.

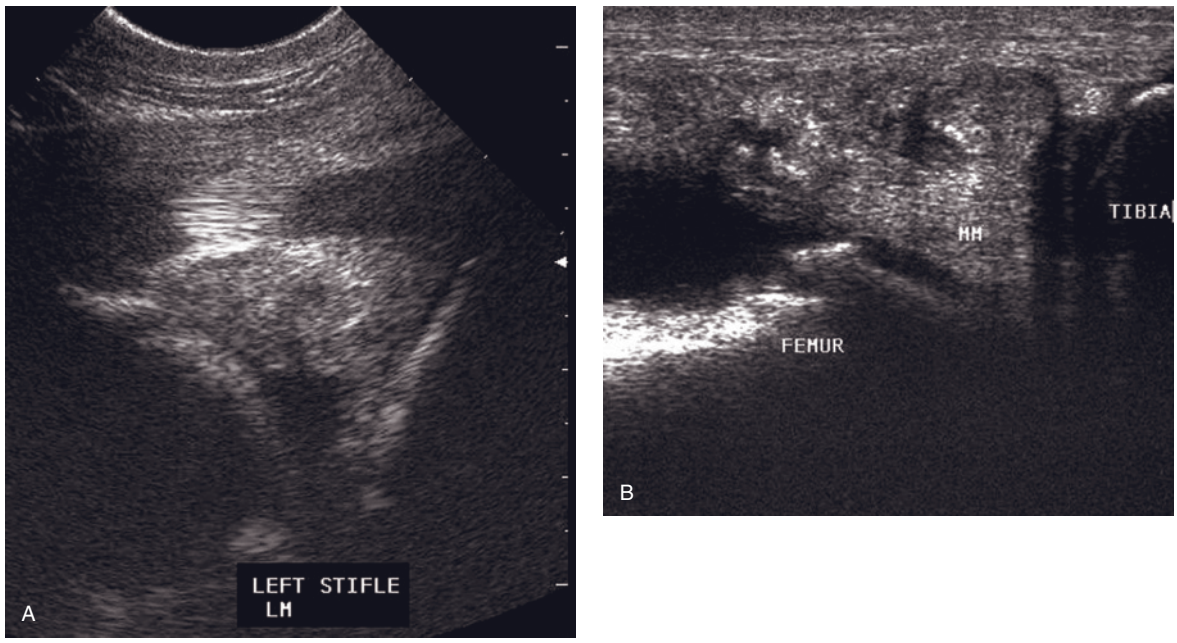



**Figure 4.50.** Damage to the medial meniscus is manifested ultrasonographically by a change in size, shape, echogenicity, or position relative to the femoral condyles and proximal tibia. (A) This ultrasound image of a damaged meniscus demonstrates an axial injury of the meniscus. (B) This image represents a more severe injury of the meniscus with prolapse of the meniscus. Courtesy of Rich Redding.

Because most horses will stand still for about 60 seconds when sedated with IV butorphanol and detomidine, the authors rarely acquire an image for less than 60 seconds. For example, if a 60-second image of the foot results in 250,000 counts (minimum 100,000 to 150,000 counts needed), we will acquire the image for 60 seconds rather than 30 seconds, which still would have given a diagnostic image, albeit with less resolution.

The risk with limiting the acquisition to a certain number of counts (as opposed to time) is that if there is urine contamination under a foot, or another limb in the field of view, or if the urinary bladder is in the field of view, then the counts recorded by the acquisition computer will include these aberrant γ-rays, which do not contribute to image quality. In fact, they reduce image quality by diminishing the number of γ-rays used for image reconstruction. It is therefore better to use acquisition time than counts/image when scanning, given the premise that the number of counts is the more critical factor in image quality. The object is to have sufficient radioactivity in the skeletal system to acquire sufficient counts/image in an appropriate length of time. Remember that the more counts acquired/image, the better the image resolution.

### *Method for a Scintigraphic Exam of the Musculoskeletal System*

The radiolabelled pharmaceutical, either $^{99m}$Tc-MDP (methylene diphosphanate) or $^{99m}$Tc-HDP (oxidronate), is generally used at a dose of 0.35 mCi/kg (0.16 mCi/lb). The radiolabel must be given intravenously or else slow release of the $^{99m}$Tc will result in sub-optimal images due to continuous release and thus high levels of circulating radioactivity. Patient control is very important because each image generally takes about 60 seconds to acquire. Chemical restraint is useful to prevent patient motion. Newer image viewing and processing software packages have a motion correction tool that allows some degree of motion with minimal or no image deterioration.

#### Imaging Technique

The tracer is given IV and the blood flow phase images are acquired immediately if

**Figure 4.51.** Delayed phase lateral view of the right tarsus of a horse, showing focal areas of IRU (arrows) on the dorsal aspect of the DIT and TMT joints (arrow), consistent with osteoarthritis. Courtesy of Erik Bergman.

required. Pool phase images are acquired within the next 10 minutes if desired. Pool phase images must be limited to about three or four anatomical regions for them to be completed before significant bone uptake occurs. Delayed phase images are acquired 2 to 4 hours after injection to allow an optimal bone-to-soft-tissue ratio. Furosemide is given IV 60 to 90 minutes before the delayed phase starts if lumbar spine, pelvis, and stifle images are being acquired. This increases the chances of an empty bladder, since the $^{99m}$Tc-HDP is excreted by the kidneys, and urine in the bladder obscures visualization of the stifles, lumbosacral junction, sacroiliac (SI) joints, and the coxofemoral joints. Lateral images of the limbs are made, being careful to position the camera lateral to the region being imaged (which is not necessarily lateral to the horse).

Dorsal views of the carpi are generally performed. Orthogonal views of a lesion always should be attempted to help document the third dimension. Lead sheets are used to shield scatter radiation from the other limbs (Figure 4.54). Lead also should be placed medial to the olecranon and the stifle to shield the sternum and the penis/urinary bladder, respectively. Slightly overlap the views so that no area is left unscanned. Be aware that increased soft tissue uptake during

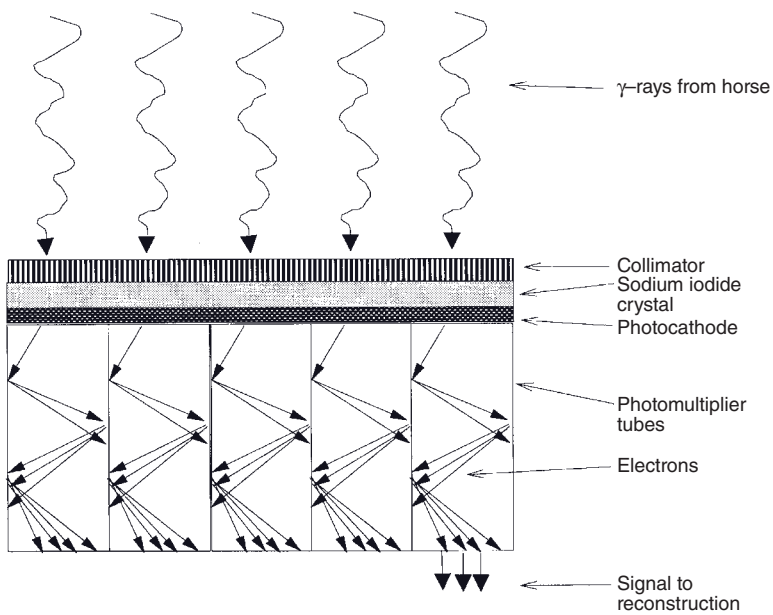



**Figure 4.52.** In the gamma camera, the γ-photons from the $^{99m}$Tc in the horse are changed to light photons by the sodium iodide crystal and then to electrons by the photocathode. The electrons are amplified by the photomultiplier tubes, and the signal is used for image reconstruction by the computer.


**Figure 4.53.** The gamma camera is mounted on a lift system with a yoke to move the camera in different directions. Both cameras can be positioned anywhere around the horse using a track system. Courtesy of Alejandro Valdés-Martínez.

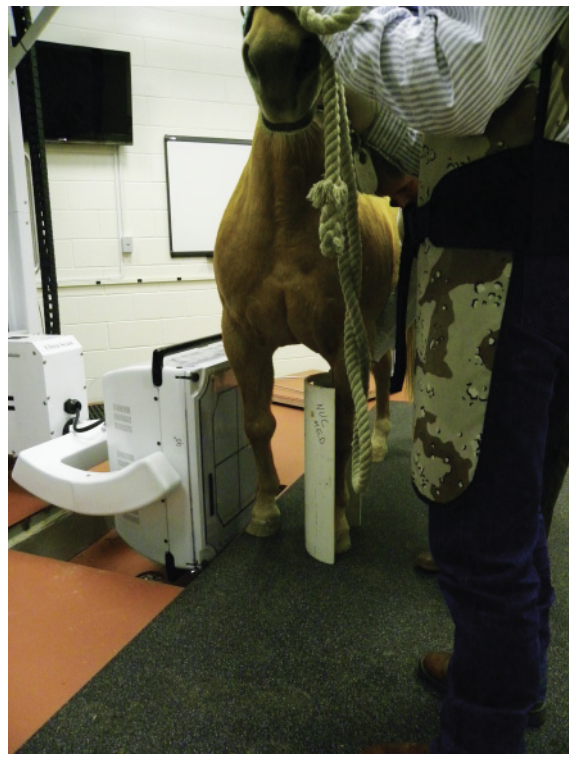


**Figure 4.54.** Gamma camera positioned in a pit below floor level for the lateral view of the right fore distal limb. Lead shielding is used to block out the contralateral limb. Courtesy of Alejandro Valdés-Martínez.

the soft tissue (pool) phase can be detected for up to 14 or 17 days after intra-articular or perineural anesthesia, respectively. Local nerve blocks (intra-articular or perineural) will not affect bone uptake in the delayed phase.

## *Indications for Nuclear Scintigraphy of the Skeletal System in Horses*

### Vascular Phase (Phase 1)

The vascular phase is excellent for evaluating blood flow to specific areas, and it is particularly important in evaluating trauma to the distal extremities or areas where inadequate blood perfusion is suspected. Decreased blood flow in the foot region may be seen in cases of laminitis. The vascular phase also can help document aortoiliac thromboembolism. Increased blood flow to a particular region may be associated with acute inflammatory conditions or infectious processes. In most institutions, the vascular phase is not included as part of a routine scintigraphic exam of the musculoskeletal system.

### Soft Tissue Phase (Phase 2)

Soft tissue phase (pool phase) images provide more useful information in cases of acute lameness, particularly in the distal limb, because of the ability to identify changes (especially increases) in blood flow to local areas. For example, hyperemia of the synovium or joint capsule secondary to acute synovitis/capsulitis, or at the proximal attachment of the suspensory ligament due to acute desmitis, may be detected during the soft tissue phase. Focal areas of trauma also can be evaluated for altered soft tissue perfusion. Sometimes it can be difficult to differentiate between early bone uptake by a lesion and increased blood flow to an area. Therefore, the more accurate soft tissue phase images are those done with pertechnetate ($^{99m}TcO_4$) or labeled RBCs ($^{99mm}Tc$-RBC), and not with a bone-seeking radiolabel. The practitioner should be aware that detection of increased blood flow to a specific region is more common in acute conditions, and a negative result on a

soft tissue phase image does not rule out a subtle or more chronic injury.

### Delayed Phase (Phase 3)

Delayed phase images provide information to evaluate the skeleton. The high sensitivity of this phase to detect early changes in bone metabolism before these changes are radiographically evident makes this part of the study most useful for evaluation of acute lameness (e.g., incomplete or stress fractures in racehorses or performance horses). The delayed phase also may help in the diagnostic work-up of horses presenting for other reasons such as ill-defined lameness or lameness that is difficult to diagnose with regional anesthesia, multiple-cause lameness in the same limb or different regions of the body, acute lameness of unknown origin, re-check of known lesion (to follow progress of healing), evaluation of the physiologic activity of radiographic lesions, and evaluation of areas that are difficult to radiograph such as the proximal thoracic and pelvic limb, the spine, and the pelvis including the sacroiliac and coxofemoral joints. Delayed phase imaging is useful for the assessment of bone viability, and as a general survey in prepurchase examinations. Soft tissue uptake in the muscles, seen during the delayed phase, can be seen in cases of rhabdomyolysis or dystrophic mineralization of soft tissues.

## *Scintigraphic Signs of Disease*

### Soft Tissue Phase

Increased activity in soft tissues is a good method to document increased blood flow to specific regions, for example joints with synovitis/capsulitis. In these cases, nuclear scintigraphy is very sensitive in detecting increased periarticular blood flow around inflamed joints before radiographic changes of OA are evident. A fetlock joint capsulitis will have a region of IRU over the joint when compared with the distal metacarpal region and the proximal phalanx (Figure 4.55).

Increased soft tissue phase uptake with a normal delayed phase image is compatible with a more acute degenerative condition, whereas if it is accompanied by increased uptake in the delayed phase, the condition is probably more chronic in nature (Figure 4.56). Soft tissue phase imaging is helpful in diagnosing desmitis or avulsion type injuries of the proximal attachment of the suspensory ligament, which are not necessarily evident on ultrasound or radiographic studies. Deep lying regions, for example the coxofemoral and SI joints, are difficult if not impossible to evaluate during the soft tissue phase because of the meager nature of the signal and the large amount of other tissues between the hip and the camera. These tissues make up several half value layers (a layer of tissue resulting in the reduction of the signal by one-half) which attenuate the beam significantly before reaching the gamma camera.

**Figure 4.55.** Soft tissue (pool) phase image of the left fore foot, showing increased blood flow to the fetlock region (arrow), which is suggestive of hyperemia associated with joint synovitis and/or capsulitis. Courtesy of Alejandro Valdés-Martínez.

Care must be taken to not over-interpret soft tissue phase images when severe increased uptake also is seen in the delayed phase images; often, soft tissue phase "hotspots" in these cases represent early bone uptake. For example, the soft tissue image in Figure 4.57A probably represents early bone uptake of the radiopharmaceutical by the proximal phalanx because of the intense uptake seen in the proximal phalanx in the delayed phase (Figure 4.57b)

### Delayed Phase

Regions with increased blood flow and osteoblastic activity demonstrate increased uptake of the radiopharmaceutical. The severity or intensity of the increased uptake can vary, and is often

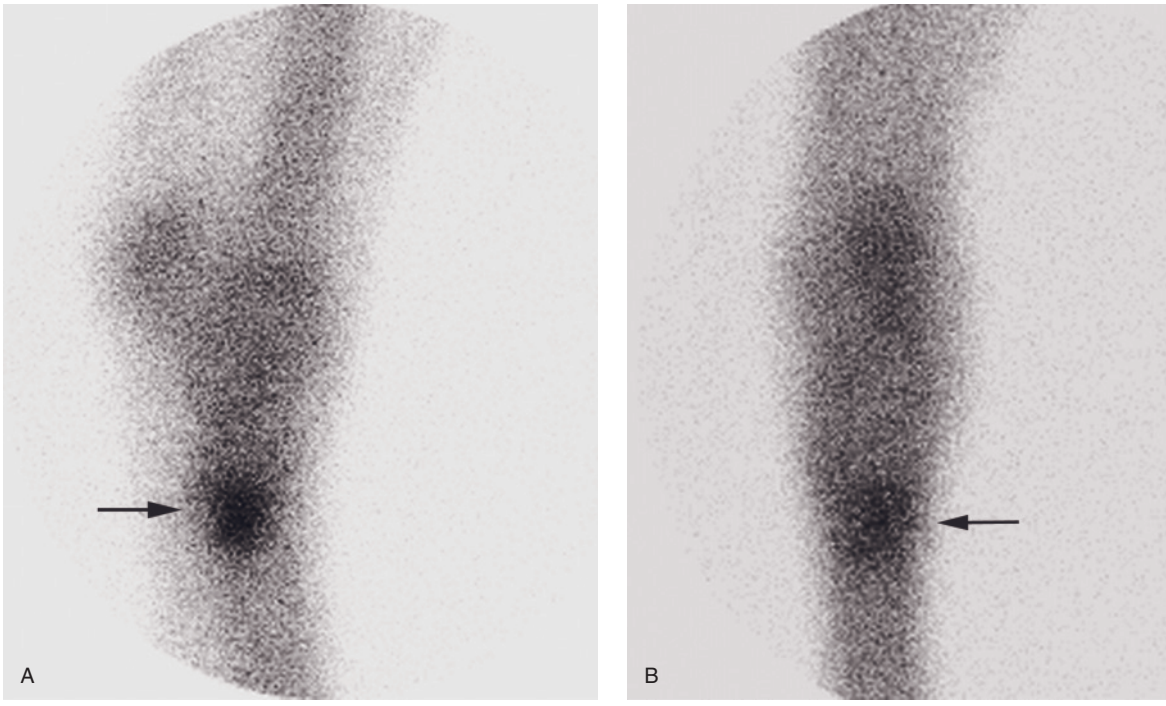



**Figure 4.56.** Delayed phase lateral (A) and plantar (B) views of the right tarsus of a horse, showing focal and intense IRU at the origin of the suspensory ligament on the proximal and plantar aspects of MTIII (arrow), compatible with origin of the suspensory desmitis and enthesiopathy. Courtesy of Erik Bergman.

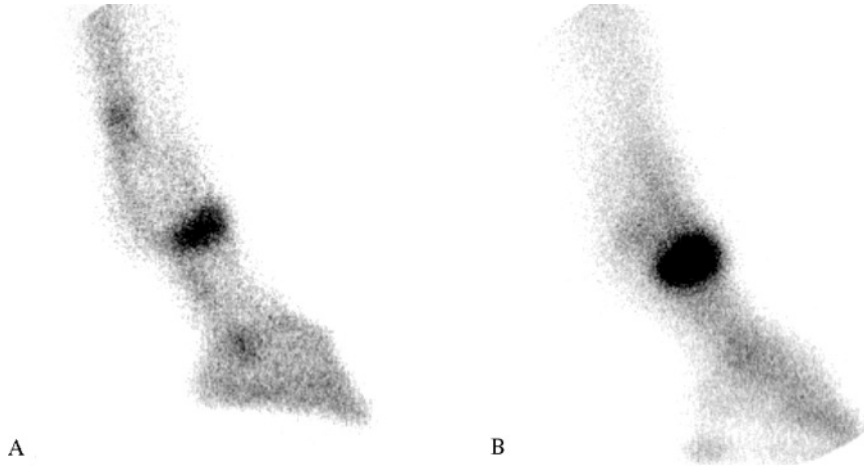



**Figure 4.57.** Right fore foot of a horse suffering from a chronic proximal P1 fracture. (A) Soft tissue (pool) phase image showing marked increased blood flow to the proximal portion of P1. (B) Delayed phase image showing marked IRU by the proximal portion of the first phalanx due to a sagittal stress fracture.

associated with conditions such as fractures, stress fractures, OA, enthesiopathy, osteomyelitis, and neoplasia. Fractures and infectious processes have similar scintigraphic behavior in most bones and in some cases it is difficult to differentiate between the two conditions. Therefore, correlation with clinical signs and other imaging findings is extremely important for making the diagnosis.

The amount of tracer uptake that a fracture demonstrates may help determine the time of onset (acute vs. chronic) or the nature (pathologic vs. traumatic) of the fracture (Figure 4.58). Chronic and sub-acute fractures (older than 48 hours) have intense increased uptake due to the considerable osteoblastic activity (Figure 4.59 and 4.60). Acute fractures have less radiopharmaceutical uptake because it takes approximately 24 hours for the osteoblastic activity to be greater than surrounding bone. In fact, acute traumatic fractures of less than 24 hours duration may fail to show increased tracer uptake when compared with adjacent bone. Fracture uptake in humans is expected at about 24 hours post injury (although it takes longer in older patients) and is expected to last for six to 12 months or longer in older patients. The uptake by a fracture should decrease over time as fracture healing occurs.

**Figure 4.58.** Delayed phase dorsal view of the distal forelimbs of a horse with a focal and intense IRU on the mid-distal diaphysis of the right MCIII, consistent with a stress fracture. Courtesy of Alejandro Valdés-Martínez.

Multifocal areas of IRU have been described with different diseases such as enostosis-like lesions (Figure 4.61), hypertrophic osteopathy, neoplasia, and horses with a bone fragility disorder, a recently reported condition of unknown etiology that affects the axial and proximal appendicular skeleton.

Localized delayed phase uptake of the radiopharmaceutical by soft tissues is not commonly seen, but can occur with various conditions (e.g., dystrophic mineralization of ligament and tendon injuries, regional anesthesia, and rhabdomyolysis), and is seen as linear or diffuse uptake patterns in the muscles, such as the gluteals, semimembranosus, and semitendinosus (Figure 4.62).

### *Limitations of Nuclear Medicine*

As mentioned above, nuclear medicine traces physiologic processes in the different body systems and therefore has a high sensitivity in detecting early changes in the metabolism. This is the main reason why nuclear scintigraphy is an invaluable diagnostic tool in equine lameness. However, the specificity of bone scintigraphy is very low in the majority of cases. During the evaluation of the bone scintigraphy results, the practitioner should be aware that an area of IRU only indicates an area of increased osteoblastic activity in a particular region. If the IRU is considered pathologic, in most situations a list of differential diagnoses should be made and further imaging performed to gain a better idea of the anatomical changes of the affected region. In general, bone scintigraphy is used to localize an area of abnormal bone metabolism that may explain the source of lameness and not necessarily the specific pathologic change. However, in some cases the diagnosis can be made from the bone scintigraphy results, such as stress fractures in racehorses.

False negative results occur secondary to many different reasons and can be considered as a limitation of bone scintigraphy. For example, a bone lesion with minimum uptake in the proximal region of a limb may not be apparent due to the significant γ-ray attenuation produced by the surrounding musculature. As in cases of subchondral bone cysts, cartilage or meniscal

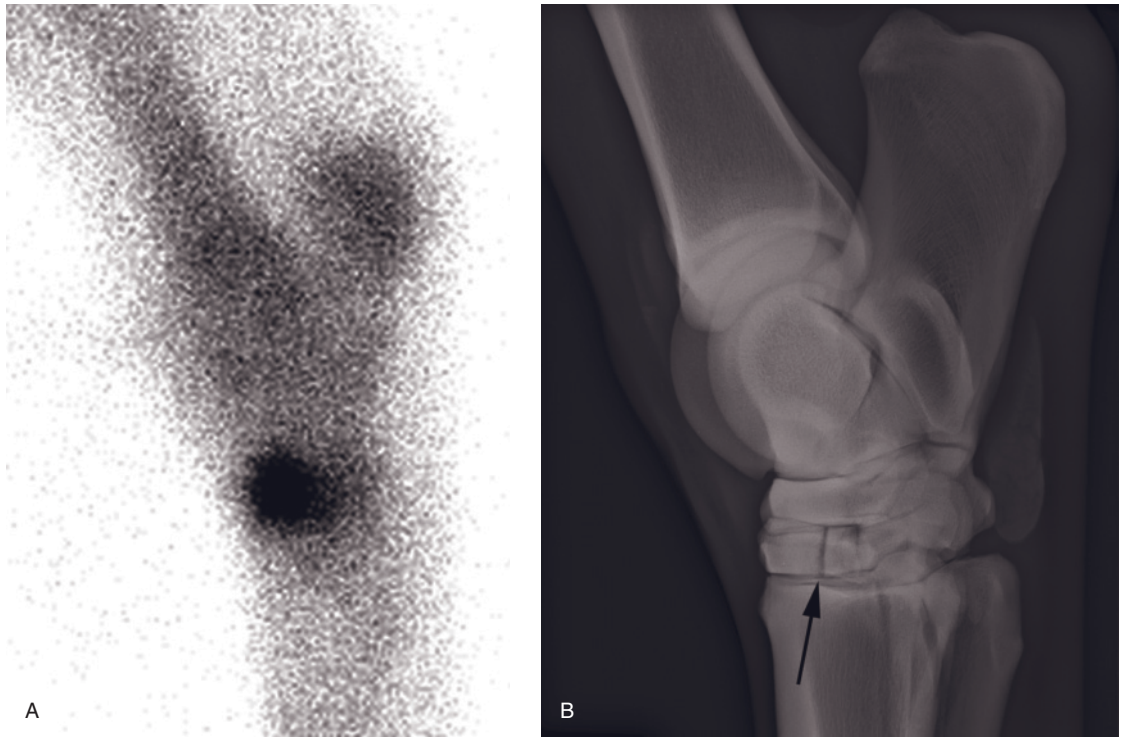



**Figure 4.59.** (A) Delayed phase lateral view of the left tarsus of a horse with focal and intense IRU in the region of the distal tarsus corresponding to a third tarsal bone fracture. (B) Lateromedial radiograph of the same tarsus showing the well-defined radiolucent fracture line extending from the proximal to the distal articular surfaces of the third tarsal bone consistent with a slab fracture (arrow). Courtesy of Erik Bergman.

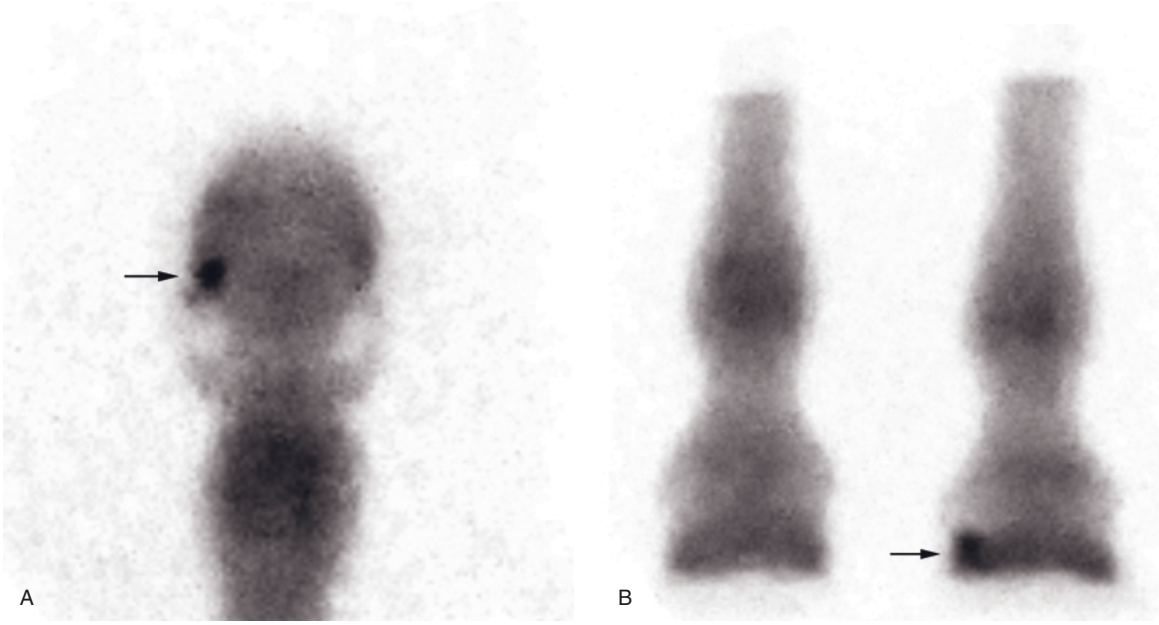



**Figure 4.60.** Delayed phase solar (A) and dorsal (B) views of the left fore foot of a horse with a focal and intense IRU on the medial aspect of P3 (arrows), representing a palmar process fracture. Courtesy of Kent Allen.

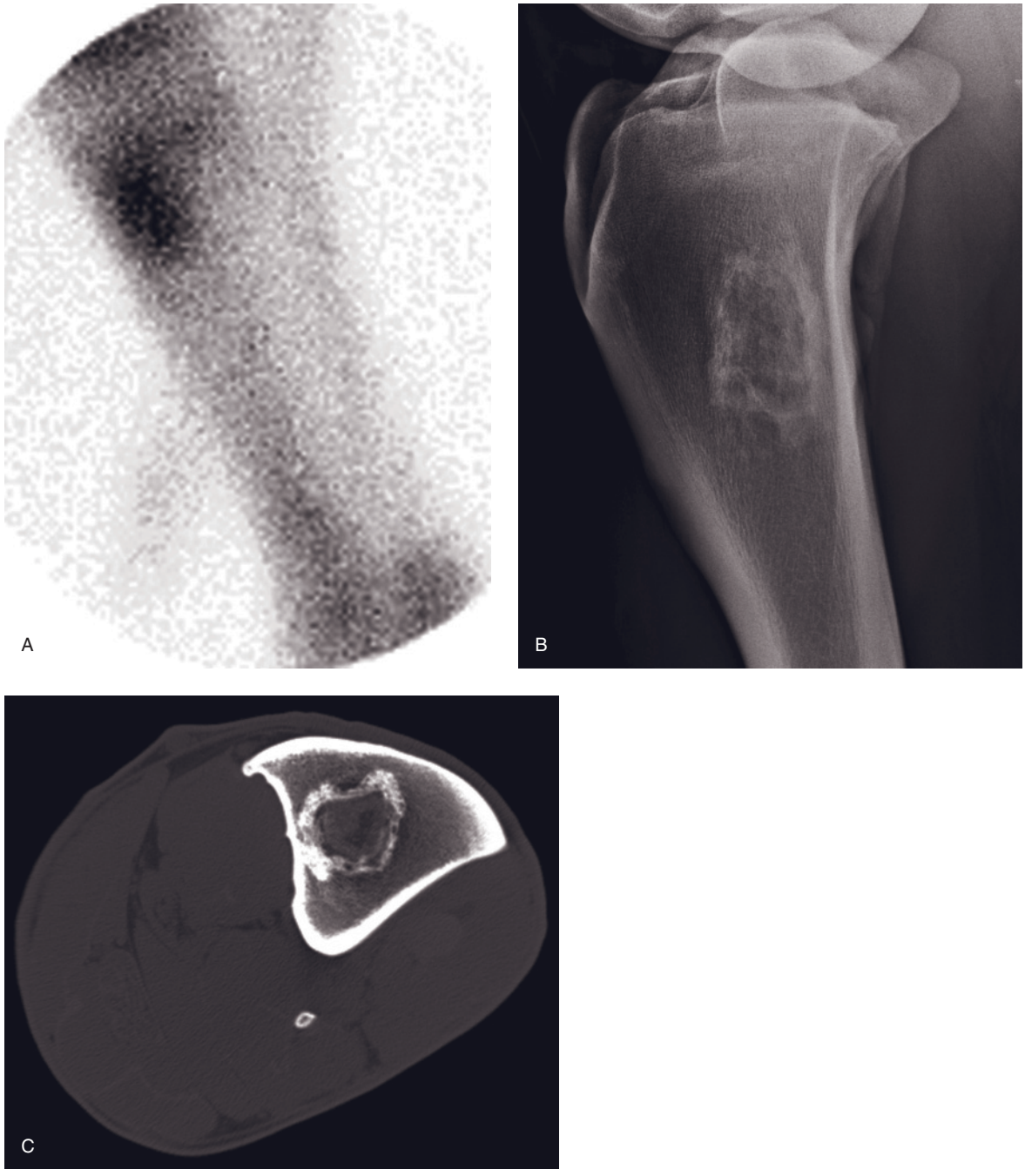



**Figure 4.61.** (A) Delayed phase lateral view of the left tibia of a horse, showing a focal and intense IRU in the medullary cavity of the proximal diaphysis, suggestive of enostosis-like lesion. Radiographic (B) and computed tomographic (CT) image (C) showing the presence of ill defined, irregular medullary sclerosis, compatible with enostosis-like lesion. Courtesy of Erik Bergman.

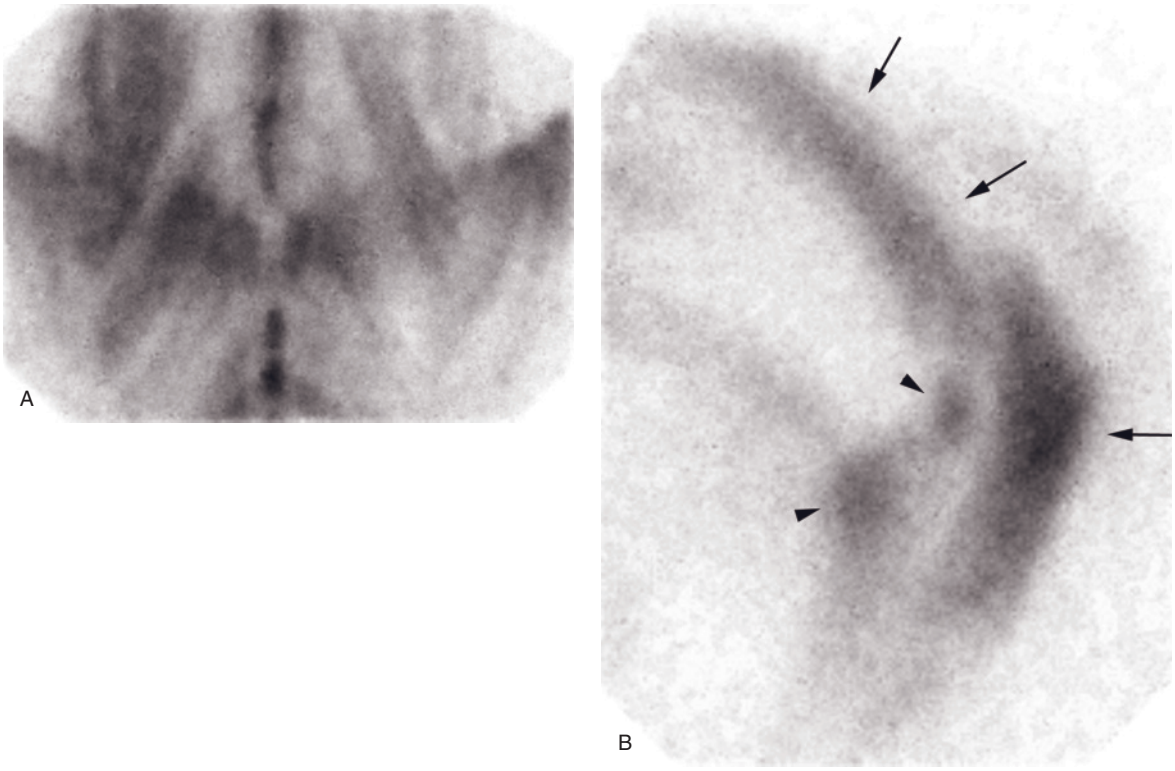



**Figure 4.62.** Delayed phase dorsal (A) and lateral (B) views of the pelvis, showing linear IRU in the muscles of a horse with rhabdomyolysis (tying-up syndrome). Courtesy of Kent Allen.

injuries will likely give a false-negative result if the adjacent bone is not affected.

## MAGNETIC RESONANCE IMAGING

### *Introduction*

Magnetic resonance imaging is a relatively new multiplanar cross–sectional imaging modality in horses that is fast becoming the gold standard for diagnosis of musculoskeletal injury of the distal limbs. MRI allows soft tissue and bone structures to be evaluated in ways not previously possible. It provides superior contrast and detail, especially of soft tissue structures, and some physiological information on both soft tissue and osseous injuries.

As with any new imaging modality, it is essential to understand the factors that influence the signal characteristics that produce the diagnostic images and to learn the strengths and weaknesses of the technique. The clinician must understand the basic physics of MRI and the MRI sequences used for acquisition in order to comprehend the relationship between signal abnormalities and pathological abnormalities and between signal and normal anatomical variations. As more and different MRI systems, both high- and low-field, become available for imaging equine patients, it is also important to remain constantly critical of image quality. The increasing use of MRI in equine sports medicine requires every equine practitioner to have a basic knowledge of MRI interpretation concepts.

Accurate knowledge of the pros and cons of MRI will help clinicians make a careful selection of horses that undergo MRI. MRI is not a substitute for in-depth clinical investigation and more conventional imaging techniques, and many diagnoses can and will continue to be made without MRI. Nonetheless, the use of MRI has highlighted the potential shortfalls of both radiography for imaging bone and ultrasonography for imaging soft tissue lesions.

### *General Principles and Physics of MRI*

MRI produces a gray-scale image of tissue hydrogen protons by placing tissues in a large

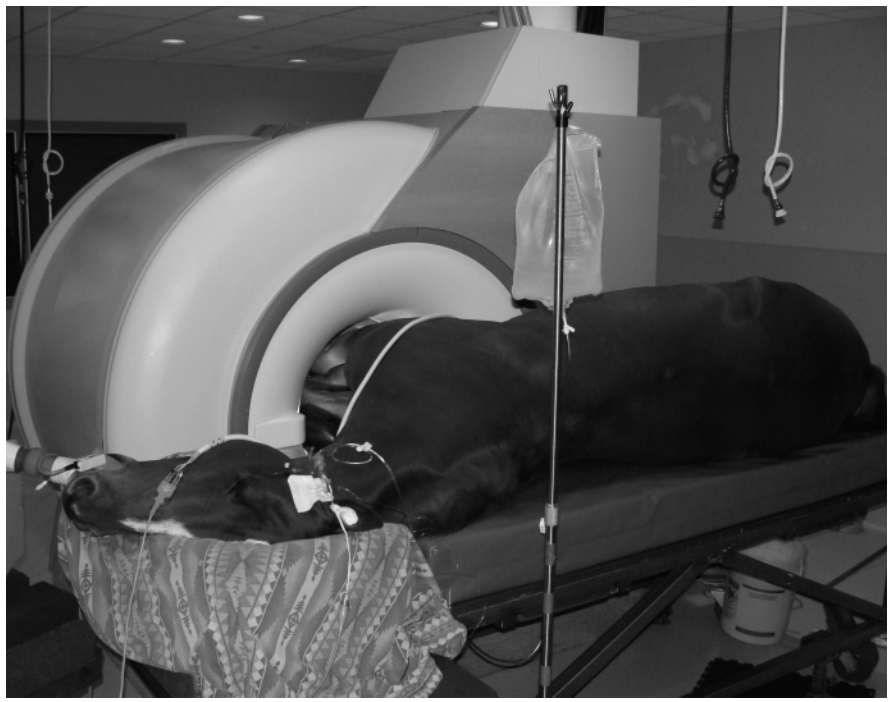


**Figure 4.63.** The 1.5-Tesla Siemens Symphony high field magnet (Siemens, Malvern, PA) used for MRI of horses under general anesthesia at the Veterinary Teaching Hospital of North Carolina State University. The horse is positioned in lateral recumbency with the lame (r) limb lowermost. The region of interest in the limb, in this case the foot, is positioned in the isocenter of the magnet. Courtesy of Michael Schramme.

magnetic field, exposing them to a radiofrequency pulse, and measuring the magnetic resonance caused by this pulse. A computer interprets the data and creates images that display the different resonance characteristics of different tissue types.

The resonance that is measured originates from the magnetic properties of the positively charged proton in the nuclei of hydrogen atoms in biological tissues. The positively charged proton spins, resulting in a magnetic moment. This magnetic moment allows it to interact with the external magnetic field. Normally, hydrogen atoms are randomly oriented in tissues.

However, when tissues are placed in the bore of a large magnet (Figure 4.63) and exposed to an external magnetic field, all hydrogen atoms align parallel with this field. Subsequently a radiofrequency coil (Figure 4.64) is applied to the anatomical area of interest within the large magnet. Radiofrequency coils are made for human shoulders, knees, and other body parts. The coil is a large inductor with a defined wavelength that can transmit the short radiofrequency pulse that makes MRI possible. The coil also may detect (receive) the magnetic resonance from the tissues.

When hydrogen nuclei are exposed to a short radiofrequency pulse specific for hydrogen by the radiofrequency coil, they absorb this pulse and change their alignment within the main magnetic field according to the direction of the radiofrequency pulse. Following discontinuation of the radiofrequency pulse, the hydrogen nuclei resume their previous orientation of parallel alignment with the main magnetic field, thereby making the transition from a high-energy state to a low-energy state. This transition results in energy release that is used to generate a signal. The exchange of energy between spin states is called the resonance, thus the name magnetic resonance imaging.

The time required for the hydrogen nuclei to resume equilibrium within the main magnetic field is the relaxation time, measured in milliseconds. The relaxation of hydrogen nuclei can be divided into two separate components, the longitudinal and transverse relaxations. Longitudinal relaxation is influenced by interaction of spinning protons with the tissue lattice.

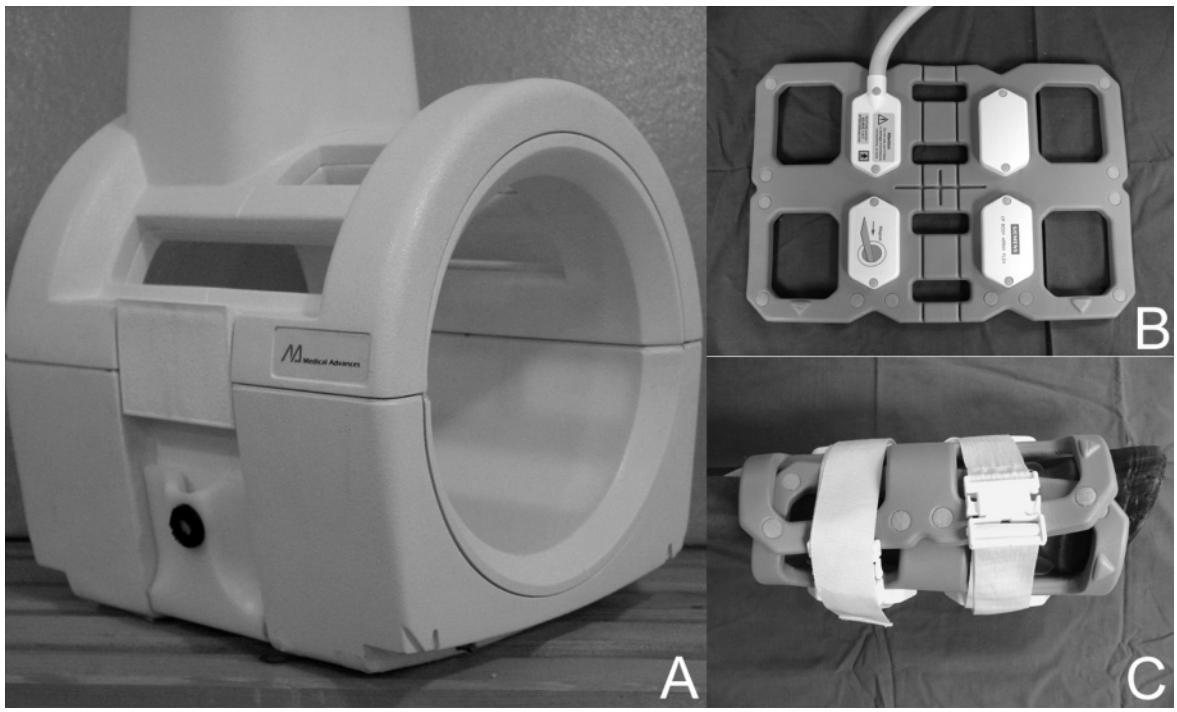



**Figure 4.64.** Radiofrequency coils manufactured for human MRI are used in equine MRI. (A) Knee coils are transmit-receive quadrature coils (volume coils) that surround the entire body part and have good field homogeneity. A human torso array coil is a receive-only flexible phased array coil (B) that can be wrapped around the horse's limb and secured with Velcro straps (C). Courtesy of Michael Schramme.

The transverse relaxation refers to the interaction of adjacent spinning protons with each other. Spin-lattice, or longitudinal relaxation time, is called T1. Spin-spin, or transverse relaxation time, is called T2. T2 is much shorter than T1. Most tissues can be characterized by their T1 and T2 signal properties. Images are constructed with the different signals obtained from the various tissue parts. The collected signal is called an echo and it is detected by the radiofrequency receiver coil placed around the area of interest in the limb. Because fat and water both contain a high concentration of hydrogen atoms, the strength of the resonance signal depends on the amount of fat and water in the tissue. High-signal areas are white and low-signal areas are black.

Within the main magnetic field, field gradients can be created by three additional gradient coils that surround the bore of the large magnet and change the intensity of the static magnetic field in transverse, dorsal, and sagittal directions. As a result, the intensity of signal produced by a given hydrogen proton can be used to position it precisely in space and create a 3D image. Hence, the magnetic field gradients allow tissue sampling as a slice in any chosen orientation or as 3D data sets. Three-dimensional signal acquisition allows thinner slice thickness and improves the signal-to-noise ratio.

### *Equipment and Practicalities of MRI of Horses*

Magnetic field strength is expressed in tesla units. One tesla (T) is approximately 20,000 times the strength of the earth's magnetic field. High-strength magnetic fields measure in excess of 1 T, low strength fields less than 0.5 T, and mid-strength fields between 0.5 and 1 T. Superconducting, closed, cylindrical bore magnets (Figure 4.63) generate high-strength magnetic fields, whereas resistive or permanent, open magnets (Figure 4.65) produce low-strength fields. Signal strength is proportional to the strength of the magnetic field. Consequently, lower field systems generate less tissue signal, require longer acquisition times, and produce

**Figure 4.65.** The Hallmarq Equine Limbscanner® (Hallmarq Veterinary Imaging, Guilford, UK) is an open, low-field magnet, mounted vertically at floor level that allows MRI to be performed on standing, sedated horses. Courtesy of Michael Schramme.

lower resolution images. The uniformity or homogeneity of the main magnetic field also is higher in closed magnets than in open magnets. Image quality and resolution increase with increasing magnetic field strength. Both high-field and low-field MRI units are currently in clinical use for horses.

Though a clinical 3 T magnet has been introduced recently, 1.5 T is considered the gold standard for high-field imaging of horses (Figure 4.63). The area of the limb to be imaged must be positioned in or near the isocenter of the cylindrical bore of the magnet, which lies at the intersection of all three gradient coils and is in the optimal position for imaging. This requires the horse to be recumbent, and therefore under general anesthesia, which increases the cost and, to some extent, the risk of the procedure. Although high-field magnets are generally capable of imaging limbs of horses from the carpus and tarsus distally, this capability is limited by how far the horse can be pulled into the bore of the magnet. Therefore, not all high-field magnets are equal in this respect. Positioning in the isocenter is more difficult in longer and narrower cylindrical bores than in some newer short-bore magnets with flared ends. In addition, some high-field magnets have a much tighter imaging window around the isocenter than others, which makes it harder to pull areas of interest further proximal than the fetlock region into the imaging window.

Low-field MRI of horses is currently performed with one of two permanent, open magnets with a field strength ranging from 0.20 to 0.26 T. One open low-field MRI scanner designed specifically for imaging distal limbs of horses (Hallmarq Equine Limbscanner®, Hallmarq Veterinary Imaging, Guilford, UK) is mounted vertically at floor level and allows imaging to be performed on standing, sedated horses (Figure 4.65). However, imaging of areas proximal to the foot is susceptible to motion artifact in these units. Other low-field magnets (Vet MR® and Vet MR Grande®, Universal Medical Systems, Inc. Solon, OH) are oriented horizontally on a pedestal and require horses to be placed under general anesthesia. Low-field magnets produce a lower signal-to-noise ratio resulting in reduced image resolution and detail.

Even so, low-field magnets are capable of producing diagnostic quality images of the distal limb and many examples of pathology visible with these magnets are available.

Radiofrequency coils designed for human imaging are used in equine MRI. The choice of coil is dictated by manufacturer availability. Most distal limb studies are performed with human knee coils or torso array coils. Knee coils are transmit-receive quadrature coils (volume coils) that surround the entire body part and have good field homogeneity over a large area. A human torso array coil is a surface or flexible phased array coil that can be wrapped around the limb and secured with Velcro straps (Figure 4.64). Surface array coils are receive-only coils that have a good signal-to-noise ratio with an additional 20% signal gain over volume coils. They allow faster scanning with finer detail and have the greatest sensitivity nearest to the coil surface.

Horses undergoing MRI must have all metal and ferrous material removed to avoid interference with the magnetic field and generation of susceptibility artifacts. This includes shoes, nails, metallic debris in the nail holes and sole, and the occasional metallic implant. Other ferromagnetic materials such as horse transport tables and anesthetic equipment may interfere with the homogeneity of the magnetic field and should be avoided in the radiofrequency-shielded MRI room. Customized non-ferrous tables and MRI compatible anesthetic machines, ventilators, and monitoring equipment are available.

### *Indications, Case Selection, Advantages, and Disadvantages of MRI*

MRI is indicated when a lameness problem has been localized to an anatomical area and other imaging modalities have failed to provide an unequivocal diagnosis. Lameness should first be localized to an anatomical area because unlike nuclear scintigraphy, MRI is not a screening technique. The examination is costly and time-consuming and should be focused and completed within a fixed time period sufficient to scan one area of interest in the lame limb and the contralateral limb for comparison. Such protocol will avoid problems associated with prolonged anesthesia in high-field magnets or motion during a prolonged standing examination in a low-field magnet. Moreover, the overwhelming detail seen with MRI can make it difficult to decide which signal abnormalities are clinically significant. Therefore, accurate knowledge of the localization of the cause of lameness with diagnostic analgesia is indispensable when interpreting MR images.

MRI is particularly useful in anatomical areas where conventional imaging modalities have limitations, such as the foot, palmar/plantar soft tissues, and joints of the distal limbs. Ultrasonographic findings may be equivocal in lameness associated with the palmar/plantar soft tissues of the distal limb and in the foot. Radiography is incapable of detecting subtle or early cartilage or subchondral bone abnormalities in joint lameness. Radiographic abnormalities also may not be present in some cases of lameness associated with scintigraphic abnormalities. Although MRI is the only imaging modality that can assess all tissues in a single examination, the availability of MRI should not result in the omission of radiographic and ultrasonographic examinations. MRI should not replace, but rather complement, radiographic and ultrasonographic findings. Radiography in particular has better bone vs. soft tissue contrast when compared to MRI and may therefore be more sensitive to subtle bone contour changes such as osteophytes and enthesiophytes.

MRI has numerous advantages over other imaging modalities. It does not use ionizing radiation. It has high intrinsic contrast and resolution, particularly for soft tissues, resulting in good anatomic separation between different tissues. As a 3D cross-sectional imaging modality, MRI can scan an object in any image plane.

The main disadvantages of MRI are its cost (installation and running expenses), still limited availability, limited accessibility restricted to examination of the distal limbs and head only, need for general anesthesia with high-field magnets, relatively lower tissue signal and interference of patient movement with low-field magnets, and the need for specialist training. MR image acquisition involves some complicated physics and a bewildering choice of pulse sequences of which the nomenclature varies between manufacturers. There is a lack of uniformity or consensus regarding the most appropriate sequences to use. Image quality can be influenced by many different parameters, including time, signal-to-noise ratio, size of the object of interest, slice thickness, field of view, and other imaging specifications. In addition, MRI gives rise to a number of unfamiliar imaging artifacts that may mimic the presence of lesions or render a scan non-diagnostic. It is

important to know how signal characteristics are influenced by all of the above mentioned parameters, so that the clinician can assure image quality. The large number of images generated with each study also makes interpretation time consuming.

### *Sequences and Protocols for Equine MRI*

MRI examinations rely on the use of several different acquisition sequences. Each sequence name describes the radiofrequency pulse applied, the weighting of that pulse, and the associated magnetic field gradients. Different sequences used in conjunction to image a given anatomical area define the imaging protocol. It is necessary to use several sequences in multiple image planes within a protocol to identify pathological conditions accurately. The common categories of conventional MRI sequences are spin echo (SE), turbo spin echo (TSE), gradient echo (GE), and inversion recovery (IR). The difference between these MR sequences relies on the method and timing of how the radiofrequency signals are pulsed into the tissues and how the resonance is collected to generate an image. Two parameters that define the sequence type are the repetition time (TR) and the echo time (TE). TR defines the time interval between radiofrequency pulses, and TE the time interval between the introduction of a radiofrequency pulse and the collection of resonance signal. These time intervals determine the tissue contrast of spin echo and fast spin echo images.

Spin echo sequences have long acquisition times and are impractical for live horse imaging. Fast or turbo spin echo sequences are used as a more practical alternative to reduce acquisition times while maintaining signal-to-noise ratio. The purpose of gradient echoes is to decrease acquisition times further and allow 3D acquisitions. However, these advantages of gradient echoes are accompanied by disadvantages such as decreased soft tissue contrast and increased susceptibility to magnetic field inhomogeneities and susceptibility artifacts. Gradient echoes can be T1-weighted or T2-weighted. Whereas some clinicians routinely use a clinical imaging protocol based on dual echo fast spin echo sequences, others prefer 3D gradient echo sequences because of the faster scanning times, thinner slices, higher detail, and increased sensitivity to hemorrhage in 3D acquisitions.

Inversion recovery sequences are produced using a similar method to that of spin echo sequences. However, the first radiofrequency pulse applied causes rotation of protons over 180° with a selected time of inversion, which suppresses returning signal from a specific tissue, usually fat. During this acquisition, it is useful to adjust the location of the digital fat suppression process manually to exactly 220 MHz away from the water peak. Fat suppression also can be achieved by a high detail spectral presaturation technique, although results of this technique are less consistent than those of inversion recovery. As a result of fat suppression, adipose tissue appears black, making fluid the only remaining source of hyperintense signal on an inversion recovery image, amidst dark bone, soft tissues, and fat. The short tau inversion recovery (STIR) sequence is commonly used in orthopedics for the detection of abnormal fluid in bone. However, STIR sequences have an increased acquisition time, decreased signal-to-noise ratio, and decreased resolution. Fat suppression techniques require good homogeneity of the system's main magnetic field and are therefore more easily obtained with high-field systems.

Depending on whether T1 or T2 relaxation is measured during acquisition, sequences are called T1-weighted, T2-weighted, or intermediate between both T1 and T2, which is referred to as a proton density sequence (PD). PD images display any change in the density of protons as a change in signal intensity. T1 and T2 have characteristic values for each type of tissue and can be used to describe the magnetic properties of all tissues (Table 4.2 and Figure 4.66). On T1-weighted images, adipose tissue has high signal (white), muscle has low to intermediate signal (dark to medium gray), and fluid has low signal (dark gray). On T2-weighted images, fat signal intensity is lower than in T1 and is bright to medium gray, muscle is still medium gray, and fluid has high signal (white). On PD images, fat is white or light gray, muscle is medium gray, and fluid has intermediate signal and is medium to lighter gray. In general, T1-weighted and PD images show anatomical detail well, whereas T2-weighted and inversion recovery images show less detail but have higher fluid contrast and are thus more likely to demonstrate pathology characterized by accumulation of fluid.

A range of time intervals is used to produce PD, T1, or T2 weighting of sequences. T1-weighted images are produced using short TR and TE values. T2-weighted images have long TR and TE values. PD images have long TR and short TE values. Manipulation of TR and TE values can produce more or less T1 or T2 weighting of any sequence and thereby result in

**Table 4.2.** The signal intensity of different tissues in different contrast weightings.

| Sequence | T2 | T1 | Proton density | Inversion recovery |
|---|---|---|---|---|
| Cortical bone | Black | Black | Black | Black |
| Cancellous bone | Light gray | Light gray | Light gray | Light gray |
| Cartilage | Dark gray | Light gray | Gray | Gray |
| Tendon | Black | Black | Black | Black |
| Ligament | Black | Gray to black | Black | Black |
| Fat | Light gray | White | White | Black |
| Fluid | White | Dark gray | Light gray | White |

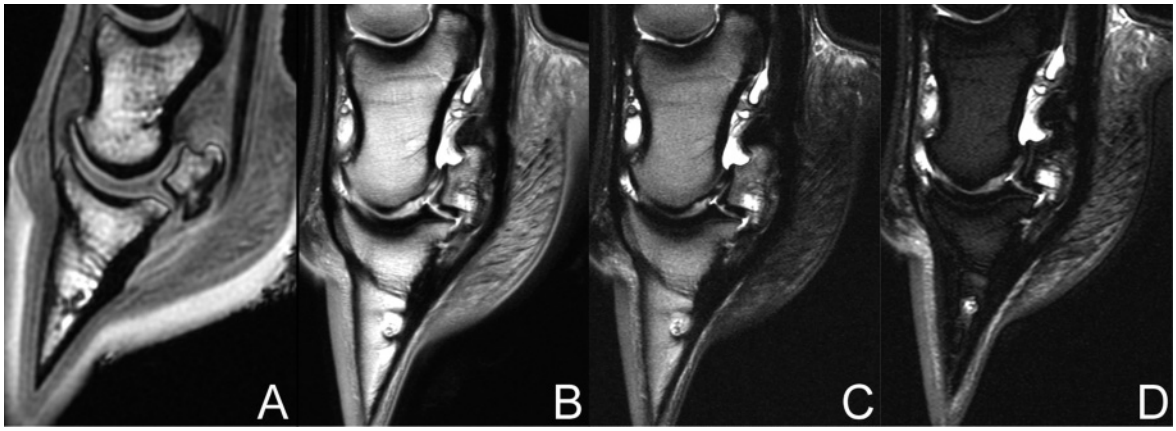



**Figure 4.66.** T1-weighted (A), proton density (B), T2-weighted (C), and short tau inversion recovery (STIR) sagittal images of the foot of a horse with navicular bone disease. Cortical bone, tendons, and ligaments are black on all sequences. On the T1-weighted image (A), fat is white and fluid is dark gray. On the proton density image (B), fat is white and fluid is light gray. On the T2-weighted image (C), fat and fluid are white. On the STIR image (D), fat is black and fluid is white. In general, T1-weighted and PD images show anatomical detail well, while T2-weighted and inversion recovery images show less detail but have higher fluid contrast. Courtesy of Michael Schramme.

different signal intensities for the same tissue within the same sequence definition. This feature may result in identical sequences with a different appearance between different MRI systems.

Standard protocols consist of proton density fast spin echo, T2-weighted fast spin echo, short tau inversion recovery (STIR) fast spin echo, and 3D gradient echo (3D GE) sequences. Because T2-weighted scans can take a long time to acquire, T2 and PD echoes can be collected concurrently as dual echoes with identical TR but different TE to minimize scanning times. PD images are easiest for evaluating ligament and tendon margins and symmetry. T2-weighted images have high fluid contrast, making them useful for looking at fluid in soft tissues. However, they have minimal shades of gray resulting in poor definition of soft tissue margins. STIR images show inflammatory fluid in bone and soft tissues most easily, but have a low resolution. T2-weighted or spoiled T1-weighted gradient echo sequences are used in 3D acquisitions for evaluation of the fine anatomical detail in thin tissue slices.

The choice of protocol is determined by the type of scanner, the region under scrutiny, and

the preference of the attending clinician. Consequently, protocols may differ between hospitals. With an endless number of combinations of sequences, weightings, and image planes available, it is tempting to continue scanning every patient until the clinician feels all questions have been answered. However, this approach lacks consistency and may cause problems. Sequences and image planes should be standardized to allow for consistent comparison between limbs and between horses. Deviation from the standardized protocol complicates interpretation and may lead to misdiagnosis. Moreover, excessive scanning times and prolonged recumbency within a confined magnet space with limited padding can result in post anesthetic complications. Table 4.3 shows the routine protocols with a typical MRI examination taking approximately one hour to complete. For optimal assessment of an anatomical region, images are generally obtained in three planes: sagittal, dorsal, and transverse. Imaging the area of interest in the lame(r) limb as well as the contralateral limb for comparison is often performed. This permits recognition of the presence of normal anatomical variations and bilateral pathology and is essential for determining the significance of signal abnormalities (Table 4.3).

## *Artifacts of MRI*

MRI produces a wide range of artifacts and variations that can confuse the interpreter. In addition, MRI is susceptible to artifacts that are created by acquisition of images at oblique angles. Even slightly asymmetric positioning of image slices can create significant problems of image interpretation. Consequently, MRI is an imaging technique that can easily lead the examiner to over interpret images as well as miss lesions. Knowledge of the regional anatomy and familiarity with the image acquisition process are necessary to understand the origin of MRI artifacts. More than with other imaging modalities, practice and experience are essential

**Table 4.3.** Siemens Symphony 1.5 Tesla magnetic resonance imaging protocols for horses.

| Slice plane | Sequence | TR msec | TE msec | FA | Matrix size pixels | NEX # | FOV mm | Slice # | Slice width mm | Gap mm | Scan pime min |
|---|---|---|---|---|---|---|---|---|---|---|---|
| **Foot** | | | | | | | | | | | |
| Sagittal | STIR | 5840 | 26 | 180 | 256 × 192 | 1 | 140 | 23 | 3.5 | 0.5 | 2:51 |
| Sagittal | PD TSE | 3800 | 14 | 180 | 320 × 224 | 2 | 140 | 23 | 3.5 | 0.5 | 5.47 |
| Sagittal | T2 TSE | 3800 | 95 | 180 | 320 × 224 | 2 | 140 | 23 | 3.5 | 0.5 | |
| Transverse | PD TSE | 3940 | 14 | 180 | 320 × 203 | 2 | 150 | 30 | 4.0 | 0.5 | 3:07 |
| Transverse | T2 TSE | 4750 | 81 | 180 | 320 × 203 | 2 | 150 | 30 | 4.0 | 0.5 | 3:07 |
| Transverse | STIR | 7610 | 26 | 180 | 256 × 192 | 1 | 150 | 30 | 4.0 | 0.5 | 3.19 |
| Transverse | 3D FLASH FS # | 36 | 10 | 40 | 256 × 256 IP | 1 | 120 | 40 | 2.0 | −2.0 | 4.09 |
| Dorsal | 3D FLASH # | 18 | 10 | 40 | 256 × 256 IP | 1 | 140 | 32 | 2.0 | −2.0 | 2:30 |
| Oblique transverse | PD TSE FS | 2760 | 30 | 180 | 320 × 224 | 1 | 150 | 26 | 3.0 | 0.6 | 2.08 |
| **Fetlock** | | | | | | | | | | | |
| Sagittal | STIR | 5490 | 28 | 180 | 256 × 256 | 1 | 170 | 21 | 3.0 | 0.5 | 2.30 |
| Sagittal | PD TSE | 4370 | 14 | 180 | 384 × 189 | 2 | 170 | 21 | 3.0 | 0.5 | 4.02 |

(*Continued*)

Table 4.3. (*Continued*)

| Slice plane | Sequence | TR msec | TE msec | FA | Matrix size pixels | NEX # | FOV mm | Slice # | Slice width mm | Gap mm | Scan pime min |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Sagittal | T2 TSE | 4370 | 112 | 180 | 384 × 189 | 2 | 170 | 21 | 3.0 | 0.5 | |
| Transverse | PD TSE | 5740 | 12 | 180 | 320 × 224 | 2 | 144 | 30 | 4.0 | 0.5 | 6.26 |
| Transverse | T2 TSE | 5740 | 124 | 180 | 320 × 224 | 2 | 144 | 30 | 4.0 | 0.5 | |
| Transverse | STIR | 7610 | 26 | 180 | 256 × 192 IP | 1 | 144 | 30 | 4.0 | 0.5 | 3.42 |
| Dorsal | 3D FLASH # | 18 | 10 | 40 | 256 × 218 | 1 | 160 | 36 | 2.0 | −2.0 | 2.31 |
| Sagittal | 3D FLASH FS # | 37 | 10 | 40 | 256 × 256 | 1 | 120 | 40 | 2.0 | −2.0 | 4.14 |
| **MC/MT** | | | | | | | | | | | |
| Sagittal | STIR | 5490 | 28 | 180 | 256 × 256 | 1 | 170 | 21 | 3.0 | 0.5 | 2.30 |
| Sagittal | PD TSE | 3800 | 14 | 180 | 384 × 189 | 1 | 170 | 21 | 3.0 | 0.5 | 2.30 |
| Sagittal | T2 TSE | 3800 | 112 | 180 | 384 × 189 | 1 | 170 | 21 | 3.0 | 0.5 | |
| Transverse | PD TSE^ | 3020^ | 12 | 180 | 384 × 269 | 1 | 144 | 30 | 4.0 | 0.5 | 3:39 |
| Transverse | T2 TSE^ | 3020^ | 117 | 180 | 384 × 269 | 1 | 144 | 30 | 4.0 | 0.5 | |
| Transverse | STIR | 7900 | 29 | 180 | 256 × 192 IP | 1 | 144 | 30 | 4.0 | 0.5 | 3.51 |
| Transverse | 3D FLASH FS # | 36 | 10 | 40 | 256 × 256 IP | 1 | 120 | 30 | 2.0 | −2.0 | 4.09 |
| Dorsal | 3D FLASH # | 18 | 10 | 40 | 256 × 256 | 2 | 140 | 36 | 2.0 | −2.0 | 3.45 |
| **Carpus/ tarsus** | | | | | | | | | | | |
| Sagittal | STIR | 5490 | 28 | 180 | 256 × 256 | 1 | 170 | 21 | 3 | 0.5 | 2:30 |
| Sagittal | PD TSE | 3800 | 14 | 180 | 384 × 189 | 1 | 170 | 21 | 3 | 0.5 | 2:30 |
| Sagittal | T2 TSE | 3800 | 112 | 180 | 384 × 189 | 1 | 170 | 21 | 3 | 0.5 | |
| Transverse | PD TSE | 3060^ | 13 | 180 | 384 × 269 | 1 | 144 | 30 | 4 | 0.5 | 4:06 |
| Transverse | T2 TSE | 3060^ | 119 | 180 | 384 × 269 | 1 | 144 | 30 | 4 | 0.5 | |
| Transverse | STIR | 7900 | 29 | 180 | 256 × 192 | 1 | 144 | 30 | 4 | 0.5 | 3:51 |
| Dorsal | 3D FLASH # | 36 | 10 | 40 | 256 × 256 IP | 1 | 120 | 30 | 2.0 | −2.0 | 4.09 |
| Sagittal | 3D FLASH # FS | 18 | 10 | 40 | 256 × 256 | 2 | 140 | 36 | 2.0 | −2.0 | 3.45 |

**TR, repetition time; TE, echo time; FA, flip angle; FOV, field of view; NEX, number of excitation; 2D, two-dimensional; PD, proton density; TSE, turbo spin echo**
**sequence; 3D, three-dimensional; FLASH, fast low angle shot sequence; FS, fat saturated; STIR, short tau inversion recovery sequence; IP, interpolated; #, RF spoiling; ^, 2 concatenations.**

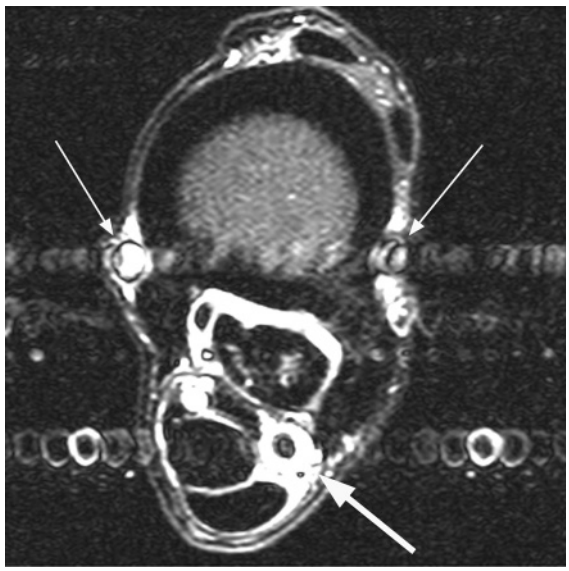


**Figure 4.67.** Transverse short tau inversion recovery (STIR) image of the right hind proximal metatarsal region. Multiple motion ghosting caused by blood flow results in displaced reduplications of the images of the lateral and medial dorsal metatarsal arteries (narrow arrows) and the medial metatarsal artery (broad arrow) in the phase encoding direction. Courtesy of Michael Schramme.

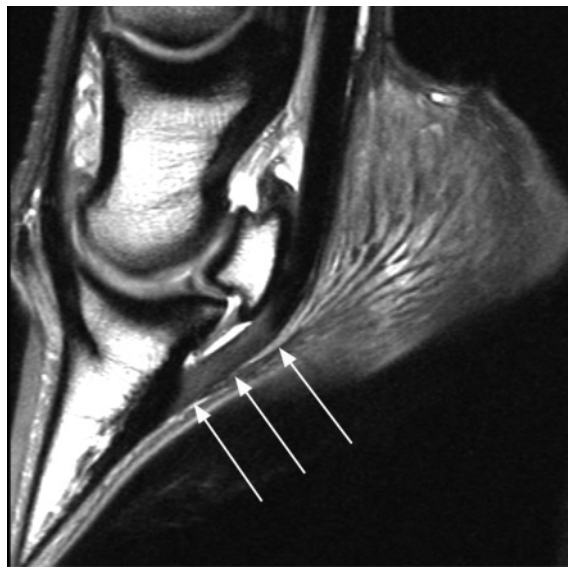


**Figure 4.68.** Sagittal proton density image of the central part of the foot of a forelimb. The magic angle effect causes an abrupt increase in signal intensity of the DDFT from the distal border of the navicular bone distally to its insertion to the distal phalanx (arrows). Courtesy of Michael Schramme.

to becoming proficient at evaluating MRI studies.

Artifacts can be classified as motion artifacts, magnetic field heterogeneity artifacts and digital imaging artifacts. Motion artifacts generally result in ghosting, which results from displaced reduplications of the image in the phase encoding direction, or in marked blurring of the image.

Respiration and blood flow can cause multiple motion ghosting (Figure 4.67). Respiratory motion is most often manifested in the upper limbs of anesthetized horses and can be reduced with sandbags or by interrupting mechanical ventilation for the duration of the more sensitive sequences. Ghost images of blood flow in vessels occur in the phase-encoding direction and it is important to select a phase encoding direction in which motion artifacts are not superimposed on areas of particular interest (e.g., the proximal part of the suspensory ligament).

Artifacts from magnetic field heterogeneity lead to image distortion or alterations in signal intensity and are more common in low-field than high-field magnets. Magnetic susceptibility artifacts are caused by the presence of ferrous material in metal or blood breakdown products from hemorrhage in tissues. Susceptibility artifacts result in an area of zero signal around the ferrous object and create distortion of the rest of the image. Gradient echoes are particularly susceptible to these artifacts.

Chemical shift artifacts are caused by the presence of fat and water adjacent to each other. This causes the position of the fat signal to shift in the frequency-encoding direction of the image. In areas of medullary bone (e.g., navicular bone) with an equal amount of water and fat, signals from both fat and water cancel each other out and are replaced with an area of zero signal (black) in the medullary cavity of the affected bone. This can lead to an erroneous diagnosis of medullary sclerosis.

Magic angle artifact causes a sudden increase in signal in tendons and ligaments where collagen is orientated at an angle of around 55° to the main magnetic field (Figure 4.68). This is most obvious at the insertion of the DDFT to the distal phalanx but can also appear in the oblique distal sesamoidean ligaments (DSL) and collateral ligaments of the joints of the distal limb. Magic angle artifact is particularly noticeable in sequences with a low TE and is less evident on T2-weighted images.

Partial volume-averaging artifacts occur when the different signal intensities of more than one tissue type within the same voxel are digitally averaged, resulting in a misleading shade of gray. This artifact results in blurring of the margins of structures and image inaccuracies. Curved and thin structures are most susceptible to the volume-averaging effect. The volume-averaging effect can be reduced by using thinner slices. Phase-wrap artifacts occur when a portion of the object that lies outside the field of view is represented out of position in the image. Gibbs truncation artifacts, or Gibbs ringing artifacts, are seen when lines of bright signal are repeated parallel with an interface of abrupt signal change between two objects of markedly different signal intensity in the image. This is an effect of undersampling and disappears with use of a higher acquisition matrix.

### *Types of MRI Abnormalities*

MRI has become the imaging modality of choice for many lameness conditions of the foot. It is most commonly used to image specific areas of the distal limb such as the foot, pastern, fetlock, or metacarpus/metatarsus when other imaging modalities have been unable to determine the cause of the lameness. Abnormalities in both bone, such as edema (Figures 4.69, 4.70) and sclerosis, and soft tissue structures, such as core lesions (Figure 4.71) and surface fibrillation (Figure 4.72), are detectable with MRI. Specific abnormalities found with MRI in the foot, fetlock, and metacarpal/metatarsal regions are included in tables 4.4 to 4.6. Common lesions identified in the foot include DDFT injuries (Figures 4.71, 4.72), navicular bone edema/disease (Figure 4.73), and lesions within the podotrochlear apparatus. Subchondral bone trauma (Figure 4.74) and injuries to the DSLs (Figure 4.75) are common lesions identified in the fetlock region. Proximal suspensory ligament abnormalities often are identified in the metacarpal/metatarsal region (Figure 4.76). Further information on specific MRI abnormalities can be found in Chapter 4 of *Adams and Stashak's Lameness in Horses, Sixth Edition*.

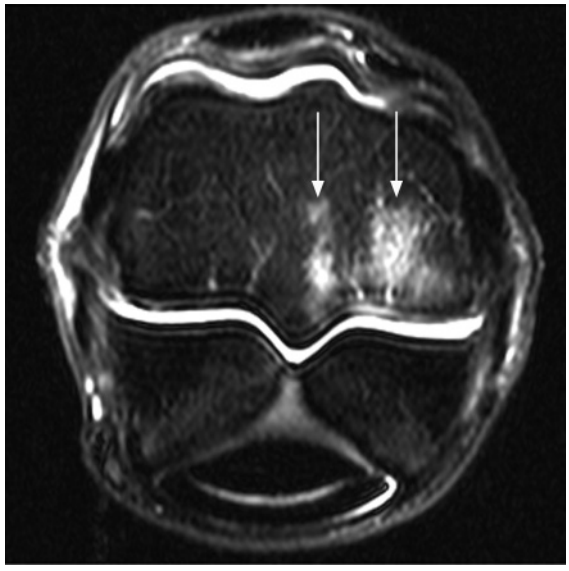


**Figure 4.69.** Transverse short tau inversion recovery (STIR) image of the distal aspect of the right MCIII at the level of the proximal sesamoid bones. There are hyperintense areas of abnormal intra-osseous fluid in the palmar aspect of the lateral condyle and sagittal ridge of the third metacarpal bone (arrows). Courtesy of Michael Schramme.

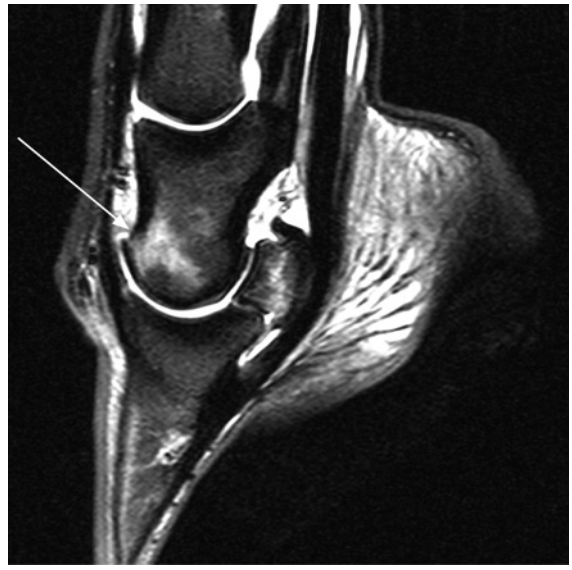


**Figure 4.70.** Sagittal short tau inversion recovery (STIR) image of the central part of the foot of a horse with acute onset foot lameness. There is an area of marked signal hyperintensity at the dorsodistal aspect of the P2 adjoining the articular surface of the DIP joint (arrow). This appearance suggests the presence of bone edema or a localized bone bruise of the middle phalanx. Courtesy of Michael Schramme.

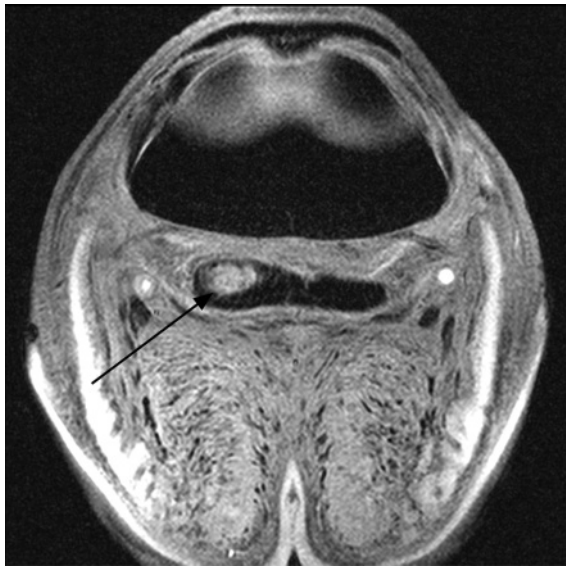


**Figure 4.71.** Transverse T1-weighted fast low angle shot (FLASH) image with fat saturation of the right front foot at the level of the middle phalanx of a horse with acute onset foot lameness. There is abnormal signal hyperintensity in a large core lesion of the medial lobe of the DDFT (arrow). Courtesy of Michael Schramme.

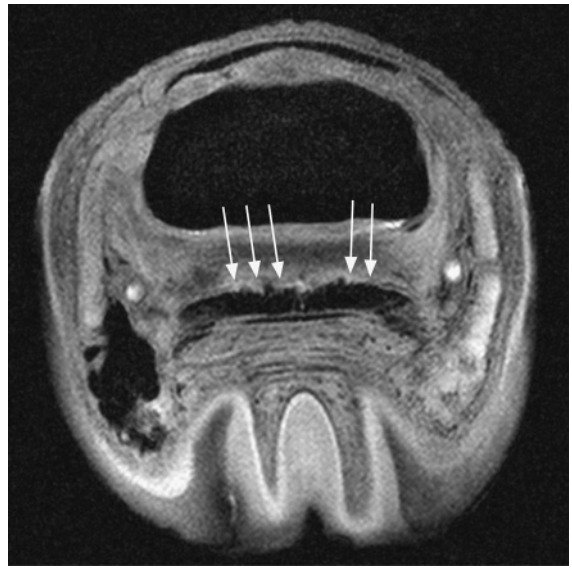


**Figure 4.72.** Transverse fast low angle shot (FLASH) image with fat saturation of the foot at the level of the middle phalanx of a horse with foot lameness. The dorsal surface of the DDFT is irregular due to the presence of fibrillations and short, incomplete sagittal splits disrupting the smooth dorsal contour of the tendon (arrows). Courtesy of Michael Schramme.

**Table 4.4.** Incidence of MRI findings in seven retrospective MRI studies of foot lameness. The total incidence of each lesion is represented, rather than only the incidence of lesions considered as the primary cause of lameness. Several horses had simultaneous incidents of more than one lesion.

| MRI lesions % total incidence | Mair et al. 2003 N = 35 | Sherlock et al. 2007 N = 41 | Dyson et al. 2005 N = 199* | Dyson et al. 2007 N = 347* | Boswell et al. 2006 N = 170 | Mitchell et al. 2006 N = 98 | Schramme and Redding 2009 N = 172* |
|---|---|---|---|---|---|---|---|
| DDFT tendinitis | 49 | 29 | 60 | 48 | 52 | 64 | 30 |
| Navicular disease | 31 | 32 | 19 | 29 | 8 | 77 | 50 |
| CL desmitis DIP joint | 6 | 7 | 31 | 43 | 23 | 21 | 16 |
| Navicular bursitis | — | 17 | — | — | — | 49 | 1 |
| Collateral sesamoidean desmitis | 14 | 10 | — | 10 | — | 13 | 8 |
| Distal sesamoidean impar desmitis | 9 | — | 10 | 15 | 1 | 4 | 6 |
| DIP joint synovitis/OA | 23 | 37 | 3 | 2 | 9 | 68 | 9 |
| Bone bruising middle or distal phalanx | — | 5 | 7 | 4 | 6 | 2 | 3 |
| Distal sesamoidean desmitis | 6 | — | 1 | 2 | 1 | — | 2 |
| Distal annular desmitis | — | — | — | — | — | — | 4 |
| Multiple injuries | NS | 15 | 17 | 33 | 4 | NS | 24 |
| No abnormalities detected | 14 | 12 | NS | NS | 6 | NS | 10 |

*, High-field MRI system; DDFT, deep digital flexor tendon; CL, collateral ligament; DIP, distal interphalangeal; OA, osteoarthritis; NS, not specified

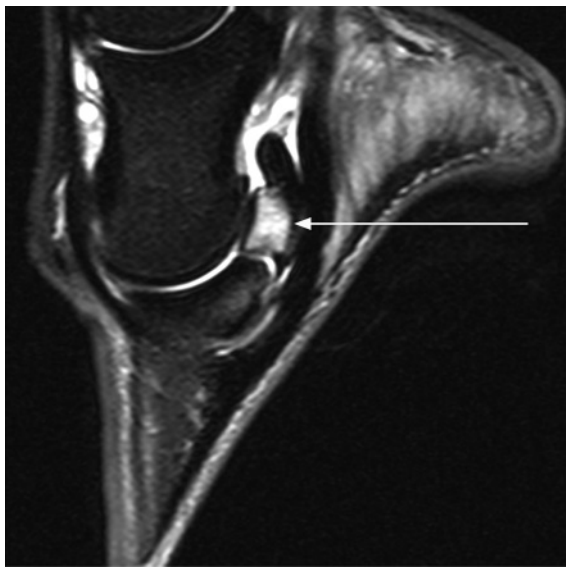


**Figure 4.73.** Sagittal short tau inversion recovery (STIR) image of the foot of a horse with lameness that is abolished by anesthesia of the palmar digital nerves. There is marked STIR hyperintensity of cancellous bone in the medullary cavity of the navicular bone (arrow), indicating the presence of abnormal medullary fluid, medullary fibrosis, or medullary fat necrosis. Courtesy of Michael Schramme.

**Table 4.5.** Lesions identified with MR imaging in 40 horses with metacarpo(tarso)phalangeal region lameness in order of incidence. Twenty-five horses had simultaneous occurrence of two or more injuries.

| MRI Diagnosis (n = 40) | Incidence % |
|---|---|
| Subchondral bone injury | 47.5 |
| Distal sesamoidean desmitis | 32 |
| OA and cartilage injury | 20 |
| Suspensory branch desmitis | 20 |
| Osteochondral fragmentation | 18 |
| Proximal sesamoid bone injury | 18 |
| Intersesamoidean desmitis | 10 |
| DDFT tendinitis within DFTS | 10 |
| Collateral desmitis | 7.5 |
| SDFT tendinitis | 5 |
| Enostosis-like lesion | 5 |
| Palmar annular desmitis | 2 |
| Proximal digital annular desmitis | 2 |
| Dystrophic mineralization of LDET | 2 |

**OA, osteoarthritis; DDFT, deep digital flexor tendon; SDFT, superficial digital flexor tendon; DFTS, digital flexor tendon sheath; LDET, lateral digital extensor tendon**

## COMPUTED TOMOGRAPHY

### *Introduction*

Computed tomography (CT) imaging, also known as "CAT" scanning (computer axial tomography), combines the use of a digital computer with a rotating X-ray device to create detailed cross-sectional images or "slices" of the different organs and parts of the body. Tomography is from the Greek word "tomos" meaning slice or section and "graphia" meaning to describe. Computed tomography is based on the principals of X-ray generation and their interaction with tissue. X-rays are directed through the body and are absorbed or attenuated at differing levels, creating a matrix or profile of X-ray beams of different strengths.

In standard radiographs generated by film-screen systems, the X-ray passes through the body part to an intensifying screen which emits light that interacts with emulsion on film. However, in the case of CT, the film-screen system is replaced by a banana-shaped detector which measures the X-ray profile of the differential X-ray absorption by each tissue. This CT technology allows the detection of thousands of differences of X-ray absorption of tissues, called Hounsfield units or CT numbers, which can be used to differentiate fat from fluid and even soft tissue. CT is so sensitive that it is capable of discriminating between tissues with density differences of as little as 0.5%, while at least 10% change in density difference is necessary before radio-opaque differences can be seen radiographically. This ability is termed contrast resolution and is far superior to film-screen combinations. In addition, rather than having a stationary X-ray tube with conventional radiography, the X-ray tube is rotated around the patient to allow for a cross-sectional image to be produced, eliminating superimposition.

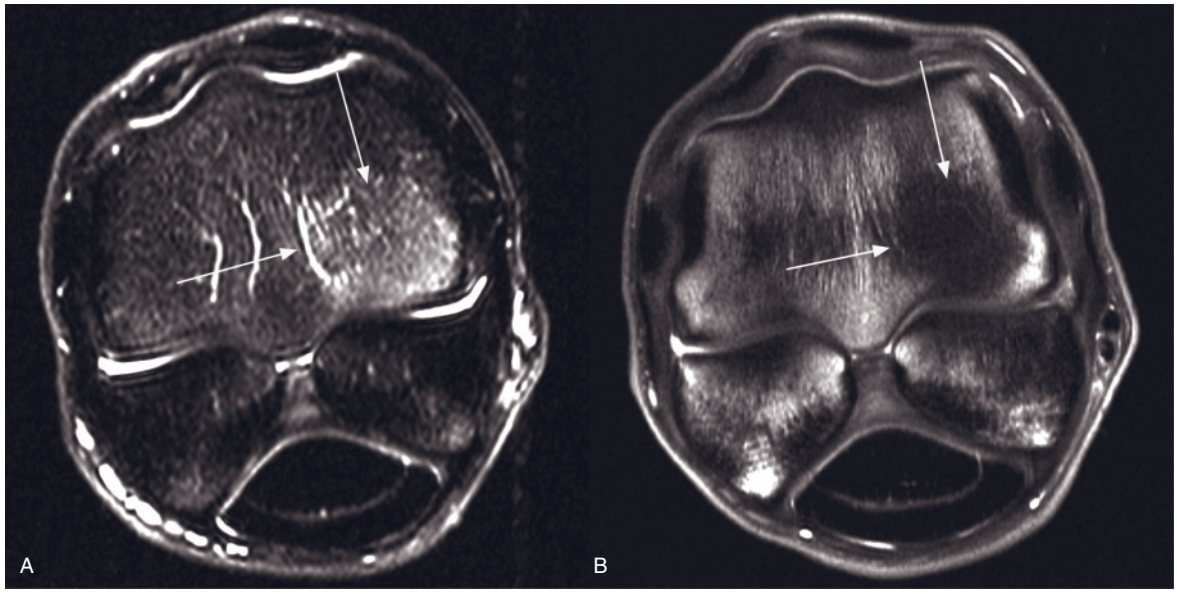



**Figure 4.74.** Transverse short tau recovery (STIR) image (A) and transverse proton density image (B) at the level of the proximal sesamoid bones of the right fore MCP joint of two 3-year-old racehorses with fetlock lameness and abnormal MR signal in the palmar aspect of the lateral condyle of MCIII. STIR signal hyperintensity suggestive of abnormal bone fluid is present in (A) (arrows), while an area of signal hypointensity indicates the presence of palmar osteosclerosis in (B) (arrows). Courtesy of Michael Schramme.

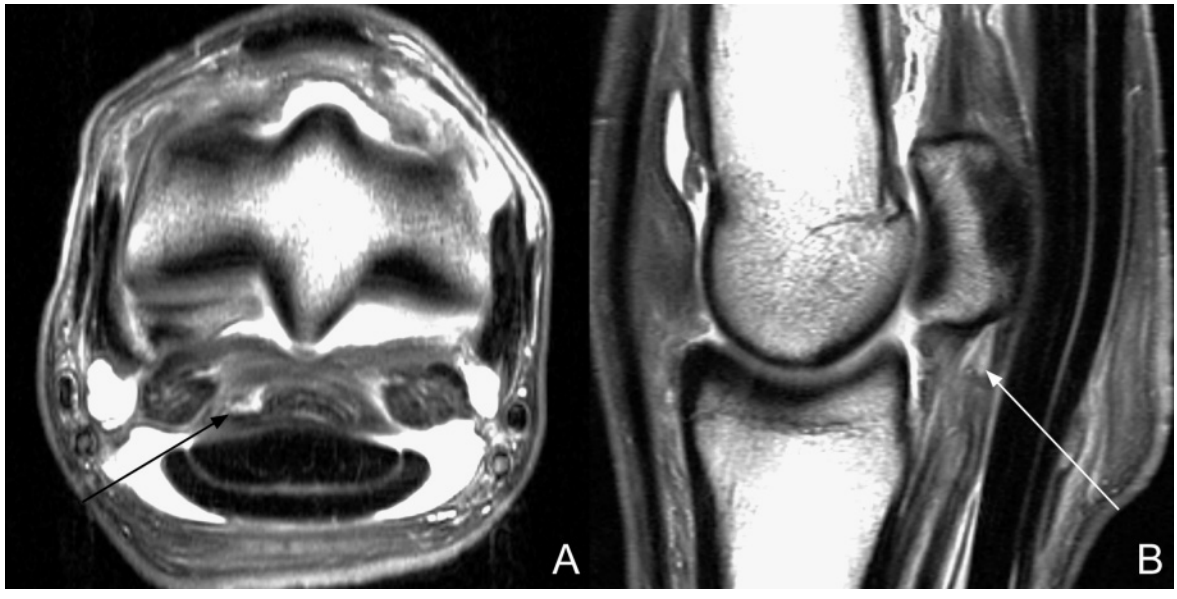



**Figure 4.75.** (A and B) Transverse and sagittal proton density images of the MCP joint of a horse with chronic fetlock lameness. There is abnormal focal signal hyperintensity within the proximolateral aspect of the straight distal sesamoidean (black arrow) ligament indicative of local fiber disruption. The lesion originates at the distal border of the lateral proximal sesamoid bone (white arrow) and extends 11 mm distally. Courtesy of Michael Schramme.

**Table 4.6.** The primary MRI diagnoses made in horses with lameness localized to the proximal metacarpal/metatarsal region.

| Primary MRI diagnosis % incidence | Schramme and Redding 2009 N = 56 | Brokken et al. 2007 N = 45 |
|---|---|---|
| Proximal suspensory desmitis without bone injury | 37 | 27 |
| Proximal suspensory desmitis and bone injury proximal MC3/MT3, MC4/MT4 | 11 | 24 |
| Desmitis of the accessory ligament of the DDFT | — | 33 |
| Distal tarsal OA | 13 | — |
| Bone injury proximal MC3/MT3, MC4/MT4 | 9 | 2 |
| Splint bone injury with focal suspensory desmitis | 5 | — |
| Central tarsal bone cyst | 5 | — |
| Focal tarsal bone edema | 4 | — |
| Enostosis-like lesions MC3/MT3 | 4 | — |
| Proximal suspensory desmitis and desmitis of the accessory ligament of the DDFT | — | 4 |
| Desmitis of the accessory ligament and tendinitis of the DDFT | — | 4 |
| Dorsal injury MC3/MT3 | 2 | — |
| Tendinitis of the DDFT | 1 | 2 |
| Effusion of the distal tarsal sheath | | 2 |
| No obvious abnormalities | 9 | 2 |

**OA: osteoarthritis; DDFT: deep digital flexor tendon; MC3/MT3: third metacarpal/metatarsal bone; MC4/MT4: fourth metacarpal/metatarsal bone**
**Brokken et al. 2007; Schramme and Redding 2009**

## *Imaging Benefits of CT*

Of all the various imaging techniques, CT is unique in its ability to image a combination of soft tissue, bone, and blood vessels at a high resolution. However, in equine medicine it is probably most frequently used to demonstrate bony abnormalities. For example, conventional X-ray imaging of the head can only show the bone structures of the skull, though all the structures are superimposed such as the nasal cavity and sinuses. CT easily shows the bone structure, but this is portrayed in cross section which allows better anatomic definition and delineation of pathological conditions. When IV contrast medium is used, soft tissue structures can be delineated from fluid.

MR imaging, which is another cross-sectional imaging technique, does an excellent job of showing soft tissue and blood vessels, but does not provide as much bony detail of structures such as the skull. MRI, when compared to CT, has much better contrast resolution for soft tissues due to the fact that it images hydrogen,

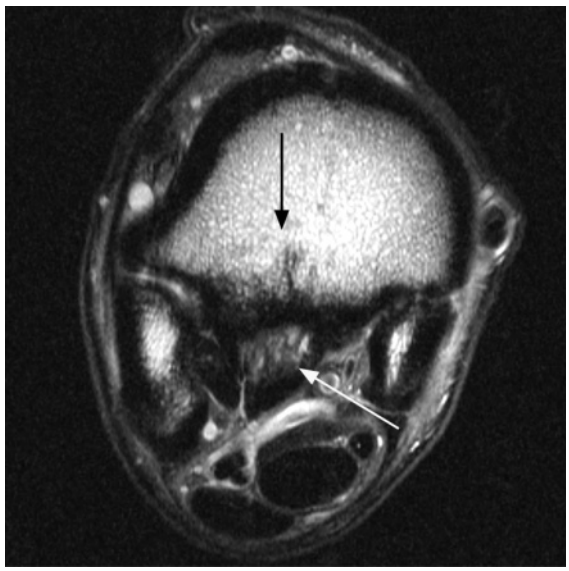


**Figure 4.76.** Transverse proton density image of the proximal metatarsal region of the left hindlimb of a horse with marked proximal suspensory desmitis and enthesopathy of the proximal plantar metatarsal cortex. There is a large central area of abnormal signal hyperintensity in the suspensory ligament (white arrow). There are irregular areas of low signal in the medullary cavity of MTIII reflecting the presence of osteosclerosis (black arrow). The plantar metatarsal cortex is thickened and has an irregular endosteal margin. Courtesy of Michael Schramme.

does not use ionizing radiation, and the sequences yield additional pathophysiological information. However, the generally poor bone detail (due to the lack of water in bones) provided by MRI, as well as the longer acquisition time and limited availability, make CT an extremely valuable diagnostic method in horses.

Furthermore, CT is much less expensive on a per case basis, compared to MRI, has faster scan times (especially multidetector systems), provides better detection of acute hemorrhage and small calcifications, and provides better spatial resolution because images can be less than 1 mm in thickness without changing the acquisition time. In addition, the images can be reconstructed in any image plane as well as being made into 3D reconstructions. CT also has the ability to image patients with ferrous implants, in contrast to MRI. MRI will likely be used preferentially over CT in many applications; however, more modern CT machines (multislice) are now competing for many applications previously dominated by MR.

Cross-sectional imaging of a structure eliminates the superimposition of bone and soft tissue, which makes CT especially helpful to examine complex joints such as the foot, fetlock, carpus, and tarsus. The CT examination is made up of multiple parallel cross-sectional slices that are acquired in transverse orientation (in the case of an extremity or head when placed in the gantry of the CT).

Modifications of current CT systems have allowed evaluations of the horse in a few select institutions. However, CT availability is limited by numerous practical and economic considerations, including purchase price, installation cost, and service contracts which can be very expensive. CT tables are designed to carry humans weighing no more than 350 to 400 pounds. This is problematic since CT requires patients to move through the gantry as the examination is being performed, unlike MRI, in which the patient is just placed in the magnet field and the images are generated. As a result, use of CT in the horse requires a special table that interfaces with the CT table without hindering the CT table's motor. Many human CT systems are available at very affordable prices but these systems should be evaluated for the ability to obtain service as well as the modifications to allow for equine patients to be anesthetized, transported to the CT scanner, and then scanned and recovered. These systems also should be evaluated for gantry diameters that physically limit introduction of the proximal limb or caudal cervical area (50 to 60 cm). Smaller orthopedic CT units are available with small gantries (20 to 40 cm in diameter), but these units can only perform studies on the distal limbs and will have a difficult time imaging the adult equine head.

### *Interpretation of CT Images*

Interpretation of CT images is relatively straightforward, provided the clinician has an appreciation of the specific anatomy of the imaged region. Because CT uses the same technology as conventional X-rays, the concepts of attenuation (whites and blacks) are similar to those found during a radiographic examination. The five roentgen opacities (air, fat, soft-tissue/fluid, mineral, and metal) still hold true, but CT allows for a number (called the CT number or

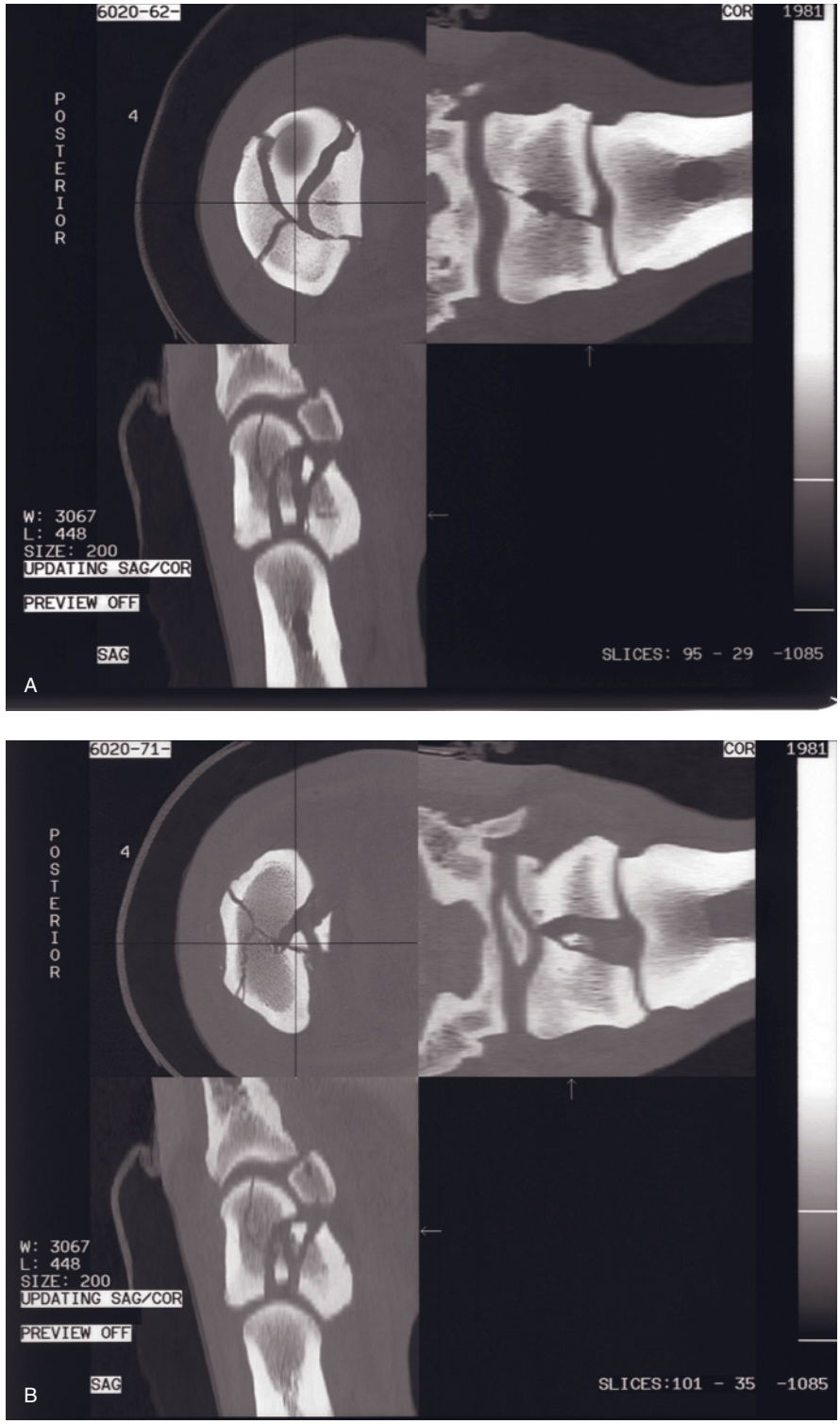



**Figure 4.77.** CT images of a horse with a comminuted P2 fracture that were obtained just before surgery to help with surgical reconstruction of the fracture. The fracture was repaired with two dorsally applied bone plates.

Hounsfield unit) to be assigned, which can be used to differentiate fluid and soft tissue. In addition, the administration of contrast medium IV allows for separation of soft-tissue (as it will enhance) compared to fluid in sinuses as well as evaluating tendon lesions.

In the examination of the extremities, images of both limbs are usually acquired at the same time, permitting critical comparison of suspected abnormalities. Attempts should be made to place the limbs in such a way as to acquire the slices of both limbs at roughly the same level. Alternatively, each limb can be imaged separately, most imaging viewer programs allow the user to match the images to provide a direct side-by-side comparison. In addition, after image acquisition, the data can be reconstructed and illustrated with 3D software programs imparting a perception of depth and volume to structures. 3D reconstruction of a structure provides the clinician with a better anatomic orientation of an area and, in the case of an extremity fracture, provides better characterization of fracture orientation (and/or disease extension). In middle phalangeal fractures, the use of CT permits better surgical planning and determination of prognosis due to the lack of superimposition as well as the assessment of joint involvement and direction of fracture lines (Figure 4.77).

### *Indications for CT in Equine Lameness and Surgery*

Historically, CT has been used to evaluate specific problems of complex joints (carpus, tarsus [Figure 4.78], fetlock, and occasionally foot) as well as the head and cranial cervical verterbra. The main reasons for using CT in horses have evolved into a quick evaluation of complex fractures (Figure 4.79) or other primary bone abnormalities that need further definition before surgery, which generally follows the examination (Figures 4.77, 4.80). Cost for the CT examination is usually half of an MR examination. Scan times are usually shorter than for MR (about 20 minutes with positioning using CT, whereas MRI can have imaging procedures up to 90 minutes in a high-field system and 2 to 3 hours in a low-field system). However, installation of new multislice CT scanners will reduce scan times dramatically. This new technology is becoming more affordable with the recent invention of the 300-slice CT scanner, making four-slice and 16-slice CT scanners obsolete in the human market and very affordable in the veterinary market.

The use of CT in the equine lameness population is probably underutilized. CT diagnoses, with or without the use of contrast medium can provide a rapid, highly detailed assessment of the distal limbs (also stifles depending on the machine). Though it lacks the ability to identify bone edema and subtle tendon lesions, these lesions are generally ones that will be treated similarly with stall rest and slow return to work. With injuries that require surgical intervention, CT is proving to be a modality of choice in the equine patient due to the quick acquisition time of a CT. With an average CT examination and contrast administration, the total CT time can be performed in 30 minutes or less. The images are relatively straight-forward to interpret and 3D reconstructions can be made in the time it takes the horse to be prepared for surgery. However, MRI will most likely remain a superior diagnostic modality to evaluate horses with subtle lameness or in cases where radiographs and rest have provided minimal benefit.

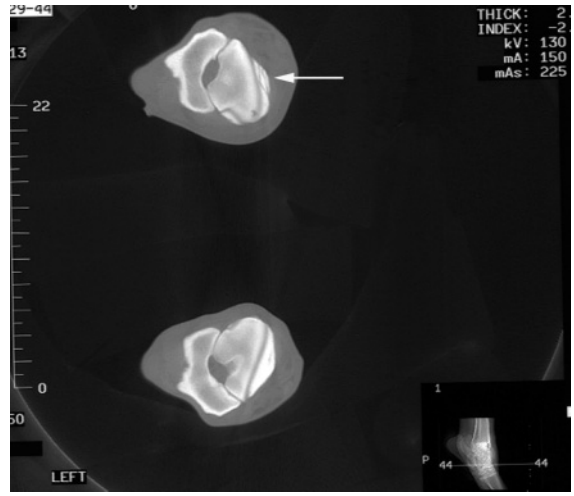



**Figure 4.78.** CT images of a horse with lameness and effusion of the tarsocrural joint. A fracture of the central tarsal bone was suspected but could not be seen on radiographs, and was confirmed with these CT images. The fracture was comminuted (arrow) and did not extend through the entire aspect of the bone.

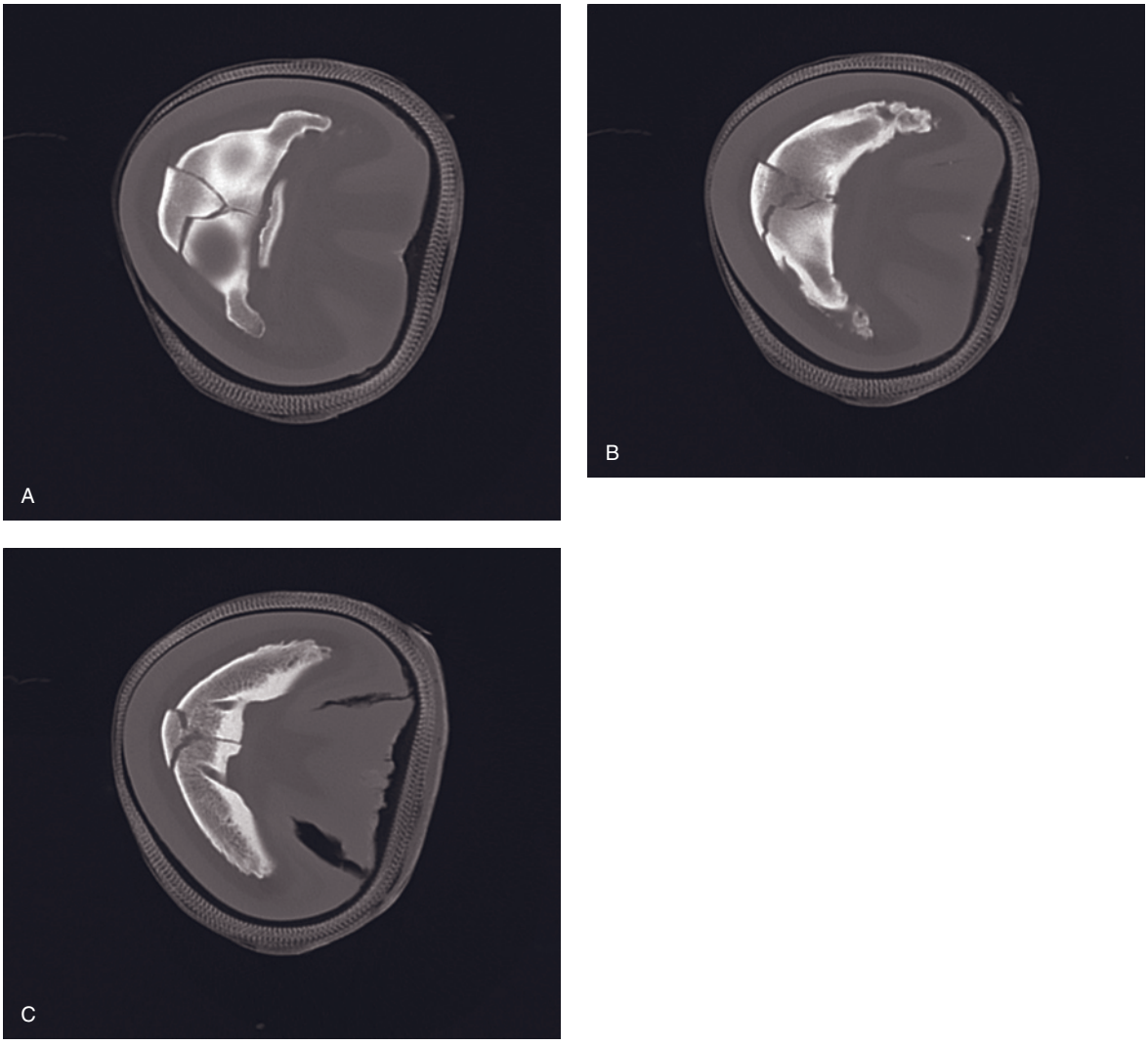



**Figure 4.79.** Proximal (A), middle (B), and distal (C) CT images of a comminuted fracture of the distal phalanx. The CT was used to rule out the possibility of lag screw repair and to further define the extent of comminution.

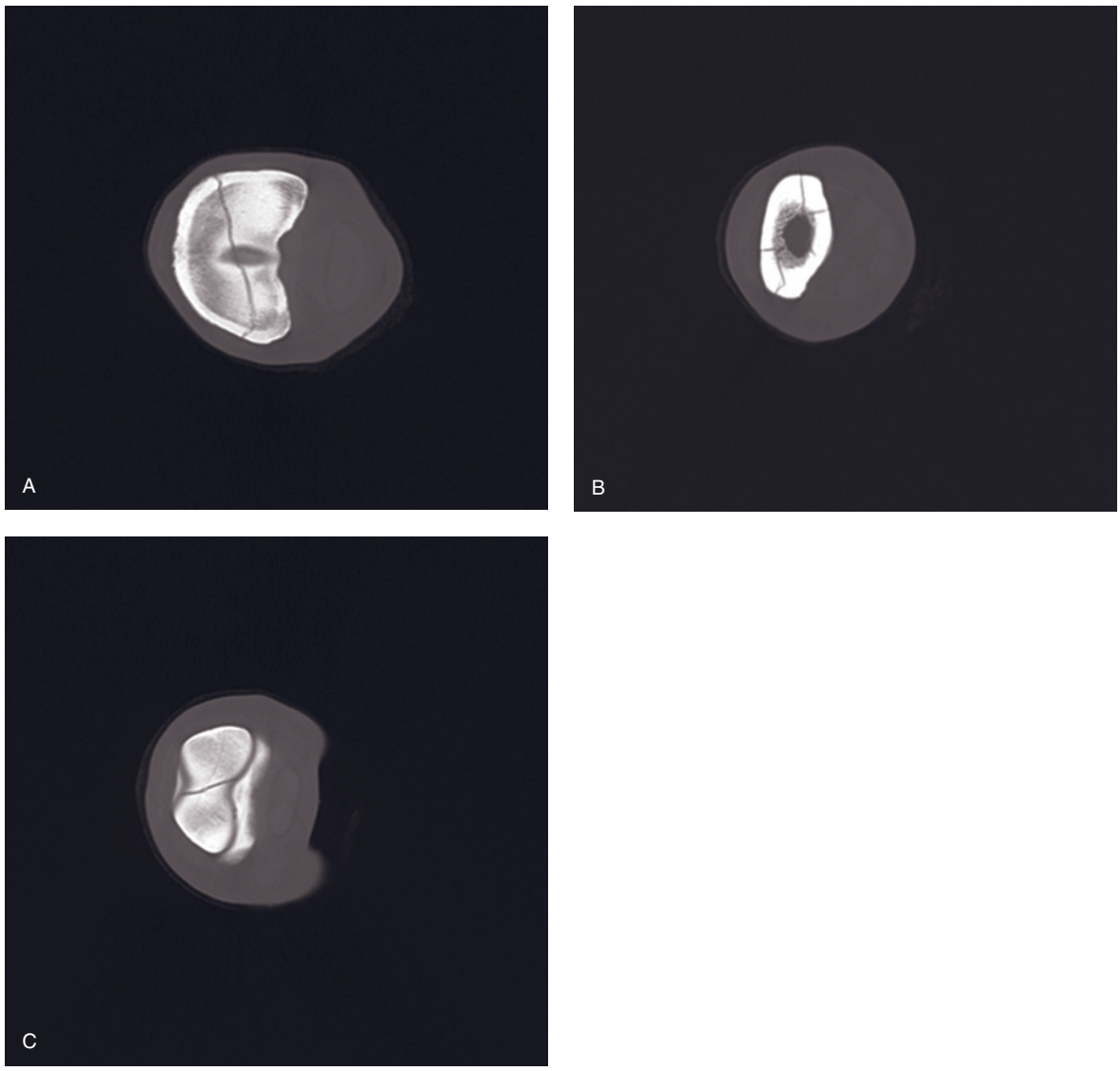



**Figure 4.80.** Proximal (A), middle (B), and distal (C) CT images of a comminuted fracture of the first phalanx that were used to help decide the location and direction for lag screw repair of the fracture.

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**Revised from "Diagnostic Procedures" in *Adams and Stashak's Lameness in Horses, Sixth Edition,* by Alejandro Valdés-Martínez, Richard D. Park, Philip F. Steyn, W. Rich Redding, Michael Schramme, and Anthony P. Pease.**

5

CHAPTER

# Common Conditions of the Foot

## NAVICULAR DISEASE/SYNDROME

### *Introduction*

- Navicular disease or syndrome is estimated to be responsible for one-third of all chronic forelimb lameness in horses.
- Quarter horses, Thoroughbreds, and Warmbloods, particularly geldings, are at greatest risk, whereas it is rarely diagnosed in ponies or Arabians.
- The definition of navicular disease/syndrome is controversial. "Navicular disease," "navicular syndrome," "palmar heel pain," or "palmar foot syndrome" often are used in horses that block to a low palmar digital (PD) nerve block.
- The disease/syndrome may be associated with pain arising from the navicular bone itself, collateral suspensory ligaments (CSLs) of the navicular bone, distal sesamoidean impar ligament (DSIL), navicular bursa, and deep digital flexor tendon (DDFT).
- A multitude of abnormalities within the foot can be desensitized with a PD block; therefore, a PD block is not specific for navicular disease/syndrome (Box 5.1).

### *Etiology*

- The two proposed causes of navicular disease are vascular compromise and biomechanical abnormalities leading to tissue degeneration. Wear and tear of the navicular apparatus from repetitive stress and biomechanical forces is considered the most likely cause.
- Excessive and repetitive force applied to the distal third of the navicular bone by the DDFT is a major contributor to the disease.
- Poor hoof conformation and balance, particularly the long-toe, low-heel hoof conformation accompanied by the broken-back hoof-pastern axis are considered major risk factors (Figure 5.1).
- Factors such as excessive bodyweight, small feet, broken pastern angles, long toes, low heels, hoof imbalances, work on hard surfaces, etc. are likely to increase the forces/unit area of the navicular bone and podotrochlear apparatus (Figure 3.1).
- Pathologic changes to the navicular bone include cartilage erosion, subchondral bone sclerosis associated with thickening of the trabeculae, focal areas of lysis, edema, congestion, and fibrosis in the marrow spaces. Damage to the fibrocartilage together with DDFT fibrillation may predispose to adhesion formation between the tendon and the bone. Other studies have identified superficial or deep sagittal or parasagittal crevices or splits of the DDFT, abrasions or fibrillation on the dorsal surface, and focal fibroplasia (Figure 5.2).

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

**Box 5.1. Abnormalities that may exist in horses classified as having navicular disease, navicular syndrome, palmar foot syndrome, or palmar heel pain.**

1. Navicular disease: radiographic, CT, or MRI abnormalities within the navicular bone
2. Desmitis/trauma of the podotrochlear apparatus
   a. Collateral ligaments of the navicular bone
   b. Desmitis of the distal sesamoidean impar ligament
   c. Desmitis of the distal digital annular ligament
3. Tendinitis of the DDFT: Usually at three locations
   a. The insertion
   b. Palmar to the navicular bone
   c. Proximal to the navicular bone
4. Desmitis of the collateral ligaments of the DIP joint
5. Navicular bursitis
6. Synovitis/capsulitis/OA of the DIP joint
7. Primary hoof imbalances (improper trimming or shoeing)
8. Hoof capsule or heel distortions

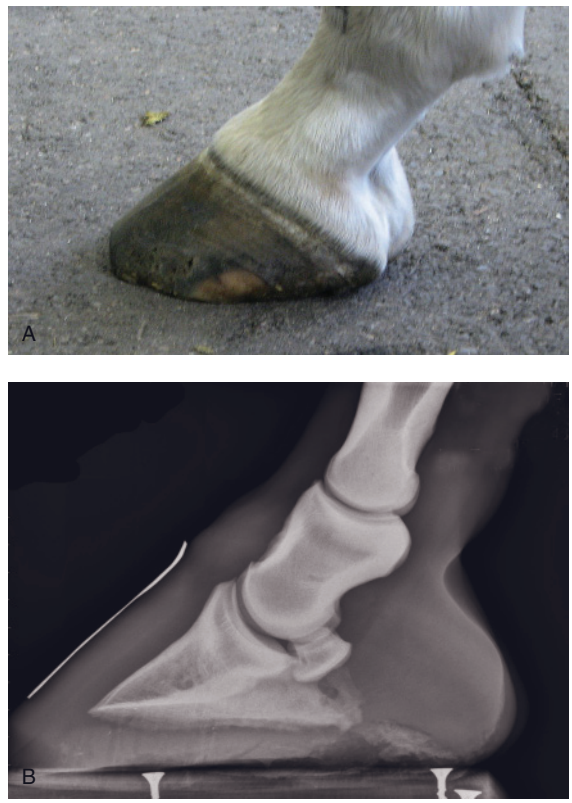



**Figure 5.1.** Front foot (A) and lateral radiograph (B) of a horse with a reverse or negative angle of P3 that is thought to predispose to problems in the palmar aspect of the foot.

- Abnormalities within the podotrochlear apparatus (CSL, DSIL) also have been described, but their clinical significance is not as well documented.

## *Clinical Signs*

- The classic signalment is a middle-aged or older Quarter horse gelding with a history of a progressive, chronic, unilateral or bilateral forelimb lameness.
- The history may include a gradual loss of performance, stiffness, shortening of the stride, loss of action, unwillingness to turn, and increased lameness when worked on hard surfaces.
- Unilateral lameness can especially occur with lesions that involve the flexor surface of the navicular bone and/or the DDFT.
- Most horses will be more lame in one forelimb, both at a straight trot and when circled on a hard surface, but will often demonstrate lameness on the opposite forelimb when circled with that limb on the inside. The lameness will often switch to the opposite (less lame limb) forelimb following a PD block of the lame foot.
- Common hoof problems seen in horses with navicular disease/syndrome include low, underrun heels, contracted or collapsed heels, medial to lateral imbalances, and long toes.
- At exercise, most horses will exhibit a mild to moderate lameness (2 to 3/5) that is worse on the inside limb when circled on a hard surface. Horses with bilateral lameness tend to have a stiff, shuffling gait and often carry their head and neck rigidly.
- Most horses will demonstrate pain with hoof testers over the central and occasionally the cranial third of the frog. However, a negative response to hoof testers does not rule out navicular syndrome/disease. Horses with very thick soles and hard frogs may not respond to hoof tester pressure.

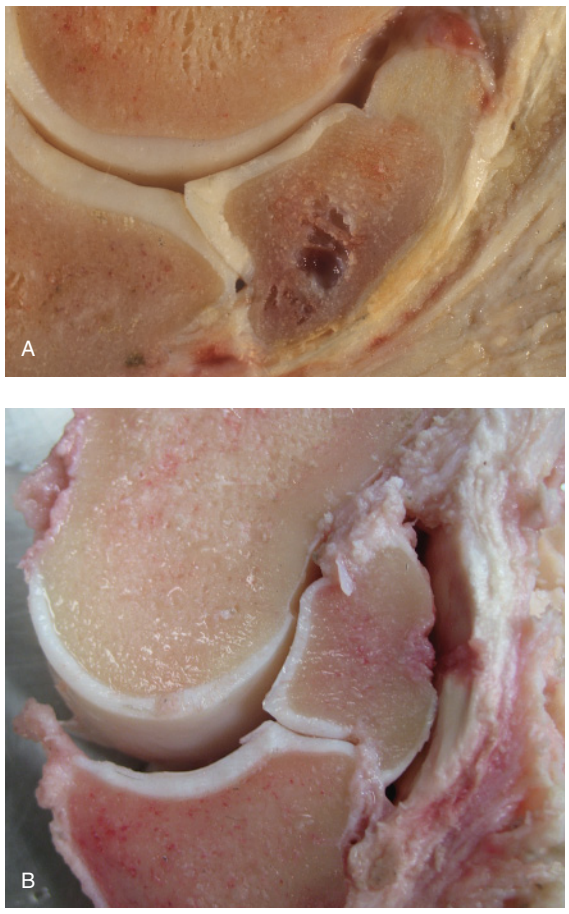



**Figure 5.2** Cross sections of the navicular bone at necropsy demonstrating cyst-like lesions within the body of the navicular bone (A) and degeneration of the flexor cortex (B). The horse in (B) also had surface fibrillation of the DDFT.

- Effusion of the distal interphalangeal (DIP) joint may be present in some horses but is not a consistent clinical feature.

## *Diagnosis*

- Diagnosis begins with localizing the site of lameness to the foot or more specifically to the palmar aspect of the foot using a low PD block. The majority of horses will improve substantially (80% to 100%) following a PD block and the lameness in the opposite forelimb will either worsen or become apparent if a unilateral lameness was initially found.
- Intrasynovial anesthesia of the DIP joint can be performed to further localize the site of pain but is not specific for the joint. The degree of improvement in lameness following DIP joint anesthesia is usually less than that following a PD block in most horses with navicular disease/syndrome.
- Anesthesia of the navicular bursa is probably the most specific nerve block that can be used to localize the site of pain but is not performed routinely by most clinicians.
- Radiography is usually the initial imaging tool, even though the lack of abnormalities in the navicular bone does not eliminate the bone as the site of the pain. Radiographic abnormalities include:
  1. Enthesiophytes at the proximomedial and proximolateral aspect of the bone
  2. Proximal or distal extension of the flexor border of the bone
  3. Distal border fragments
  4. Large and variably shaped distal border radiolucent zones
  5. Discrete radiolucent areas in the spongiosa with or without detectable communication with the flexor cortex
  6. Increased thickness of the flexor cortex
  7. Sclerosis of the spongiosa.

  Additional abnormalities that are considered important include flexor cortex defects or erosions, loss of corticomedullary distinction, and mineralization of the supporting ligaments of the navicular bone or the DDFT. (Figures 5.3 to 5.5)
- Ultrasonography can be used to help diagnose potential soft tissue injuries within the foot but has fallen out of favor with the advent of MRI. Negative findings with ultrasound do not rule out the presence of abnormalities in the navicular region.
- Scintigraphy does not provide a definitive diagnosis and the economics of performing multiple advanced imaging techniques on the same horse has made scintigraphy less useful. MRI is preferred
- Computed tomography (CT) is the best modality to detect and assess pathology within the cortex and trabeculae of the navicular bone. However, intra-arterial contrast-enhanced CT has been shown to improve the imaging of soft tissue structures within the foot, and may be an alternative to using MRI
- Magnetic resonance imaging (MRI) is currently the preferred diagnostic technique after radiography to assess most horses with navicular disease/syndrome. The different sequences permit accurate evaluation of soft tissues, cartilage, and bone within the digit in near anatomic detail (Figure 5.6). The MRI technique

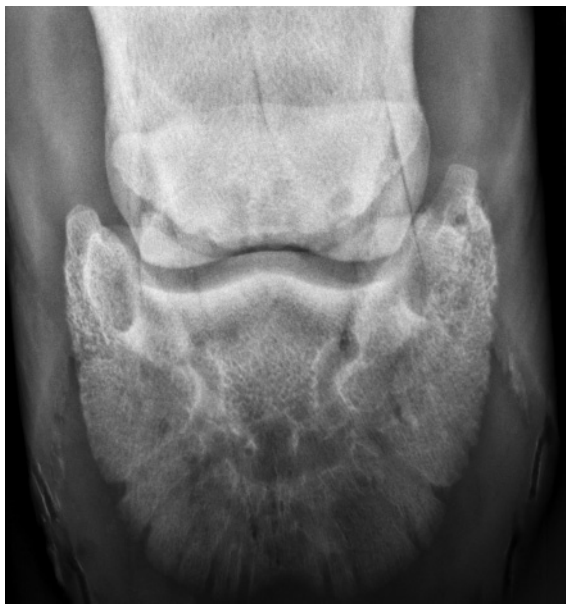


**Figure 5.3** Multiple abnormalities within the navicular bone as seen on an oblique radiograph. Abnormalities present include remodeling along the proximal border, multiple cystic lesions along the distal border, and enthesiophytes on the wings of the navicular bone.

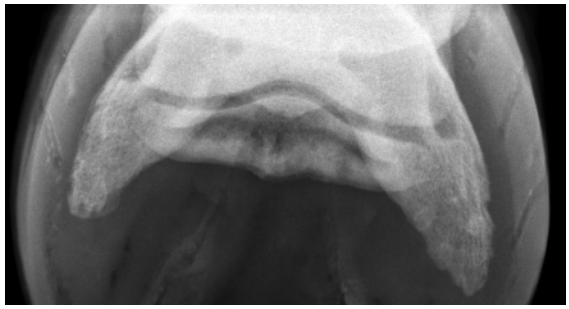


**Figure 5.4.** Skyline radiograph of the same horse as in Figure 5.3, demonstrating sclerosis of the medullary cavity of the navicular bone and erosions along the flexor surface.

can be performed in the standing or recumbent patient.
- Usually, multiple abnormalities within the foot most likely contribute to the pain in horses with navicular disease/syndrome.

### *Treatment*

- The treatment plan is usually tailor made for each individual horse based on the severity of lameness, intended use of the horse, wishes of the owner, results of diagnostics (or lack of diagnostics such as MRI), hoof conformation, previous treatments, and the most likely diagnosis.
- Horses with advanced radiographic abnormalities in the navicular bone will be problematic regardless of the treatments employed.
- The goal is to manage the disease. The cornerstone of treatment is corrective trimming and shoeing. The goals of trimming and shoeing are to:
  1. Restore normal foot balance
  2. Correct foot problems such as shearing of the quarters and heels, underrun heels, and heel bulb contraction
  3. Reduce biomechanical forces on the navicular region
  4. Ease break-over
  5. Support the heels
  6. Protect the injured areas of the foot (Figure 5.7).
- Non-surgical treatments include rest and controlled exercise; isoxsuprine; nonsteroidal anti-inflammatory drugs (NSAIDs); bisphosphonates such as tiludronate; and intrasynovial medications into the DIP joint, navicular bursa, or digital flexor tendon sheath.
- Horses with core lesions within the DDFT may benefit from intralesional regenerative therapies depending on their location.
- Surgical treatments include palmar/plantar digital neurectomy, inferior check ligament

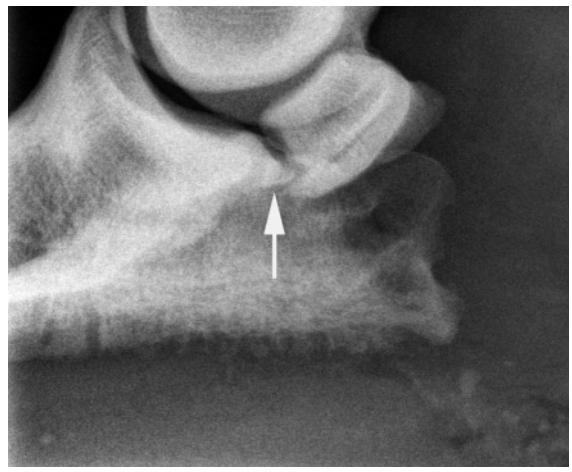


**Figure 5.5.** An avulsion fracture from the distal border of the navicular bone (arrow) can be seen on this lateral radiograph.

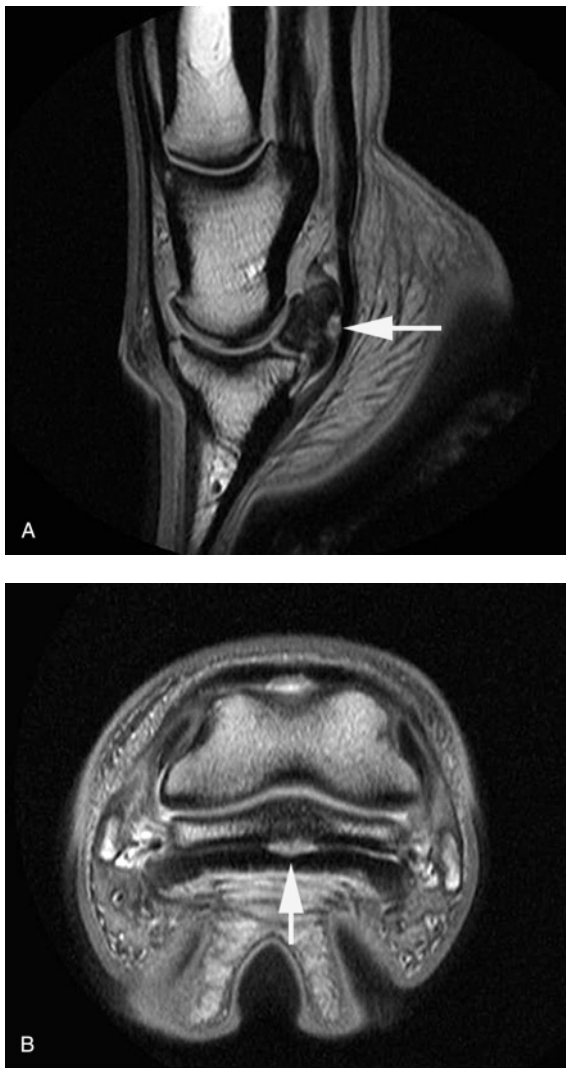



**Figure 5.6.** (A) Lateral STIR MR image of the horse in figure 5.2B demonstrating abnormal signal within the navicular bone (arrow) and (B) surface damage to the DDFT (arrow).

desmotomy, and endoscopy of the navicular bursa in select cases. (The reader is referred chapter 9 of this text and to Chapter 5 in *Adams and Stashak's Lameness in Horses, Sixth Edition*, for further details of treatment options).

### *Prognosis*

- In general, clinical resolution may occur in 40% to 50% of the horses but the optimal treatment and prognosis may differ depending on the specific pathologies that may be present.
- Horses with major radiographic abnormalities will most likely have a worse prognosis than horses with no radiographic changes or minor soft tissue pathology.
- Horses with navicular bone pathology demonstrated on radiographs or MRI together with concurrent DDFT lesions also tend to have a poor prognosis.
- Horses with primary soft tissue injuries are thought to have a guarded prognosis for return to full athletic function, and horses with major lesions in the navicular bone, *per se,* have a poor prognosis.

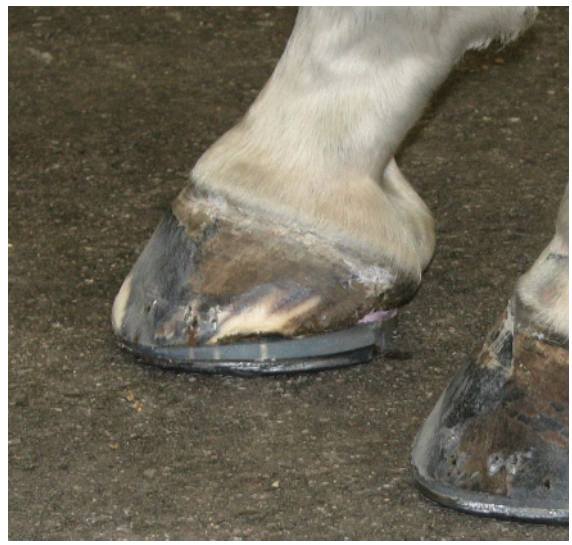


**Figure 5.7.** Natural balance shoes with a full wedge-pad and dental impression material placed in the palmar aspect of the foot can be used to treat horses with navicular disease/syndrome.

## FRACTURES OF THE NAVICULAR (DISTAL SESAMOID) BONE

### *Etiology*

- Acute trauma (concussion) to the foot is the most likely cause of simple and comminuted complete navicular bone fractures.
- Severe navicular bone osteolysis associated with navicular disease or sepsis may predispose to pathologic fractures.
- Avulsion fractures occur along the distal border of the bone and are considered part of the pathologic changes associated with

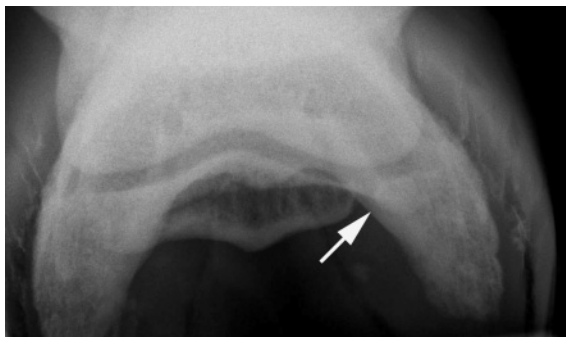


**Figure 5.8.** Bipartite navicular bone as seen on a skyline radiograph in a young horse with lameness isolated to the foot.

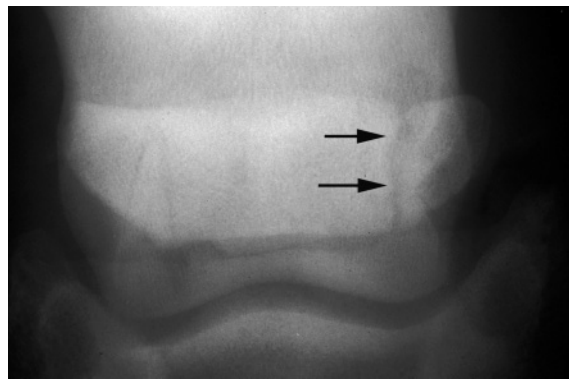


**Figure 5.9.** A wing fracture of the navicular bone (arrows) as seen on the oblique radiograph of the foot.

navicular disease, but they also may be trauma induced.
- Bipartate navicular bones are developmental abnormalities, are not a true fracture, and can be confused with a chronic fracture radiographically (Figure 5.8).

### *Clinical Signs*

- Navicular bone fractures have been reported in many breeds and in horses with varied use.
- Complete fractures can occur in any limb, but avulsion fractures are most common in the forelimb.
- The severity of lameness and clinical signs in horses with avulsion fractures are similar to those in horses with navicular disease.
- Horses with complete navicular bone fractures typically have a history of an acute, severe lameness in one limb that improves with time.
- Most horses with complete fractures have a painful response to hoof testers across the frog region and often have effusion within the DIP joint.
- A PD nerve block should improve the lameness in most cases, but regional anesthesia at a more proximal location may be required to completely eliminate the lameness. Intra-articular anesthesia of the DIP joint usually improves the lameness.

### *Diagnosis*

- Radiography of the foot is required to confirm the diagnosis. Careful packing of the frog is necessary to avoid confusing the lines from the lateral sulci of the frog that cross the navicular region with a fracture. Complete simple fractures are typically located in the sagittal plane medial or lateral to the midline and are typically not displaced (Figure 5.9).
- Most complete fractures are best identified on the skyline or 60° oblique views of the navicular bone, and should be present on multiple views. Navicular bone fractures must be differentiated from congenital bipartite or tripartite separation.
- Avulsion fractures can be difficult to identify on radiographs. Often, they are best seen on the 60° oblique view, but they also may be present at the distal aspect of the navicular bone on a lateromedial view (Figure 5.5) or within the medullary cavity of the navicular bone on the skyline view.

### *Treatment*

- There is no known specific treatment for avulsion fractures. Horses are treated similar to those with navicular syndrome but often benefit from heel elevation (provided the heels are not already too long) to relieve tension on the DSIL and DDFT.
- Horses with complete navicular bone fractures usually are treated non-surgically by confinement alone, confinement and corrective shoeing (usually with heel elevation of 3° to 12°), or external coaptation aimed at reducing hoof expansion. A minimum of four to six months of stall rest has been recommended because these fractures are very slow to heal.
- Surgical repair of simple complete navicular bone fractures using a single cortical bone screw placed in lag fashion has been reported.

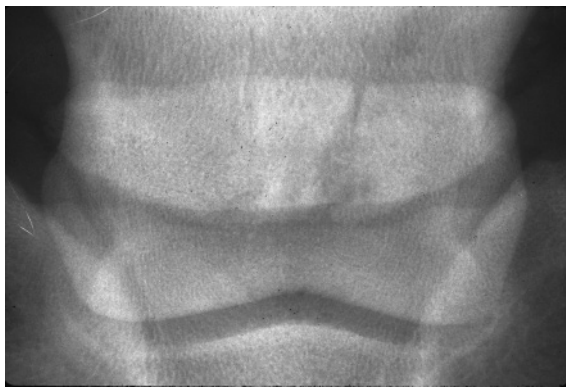


**Figure 5.10.** This oblique radiograph of the navicular bone was taken 23 months after the fracture occurred.

- PD neurectomy also can be performed to relieve pain in cases that have not responded to conservative treatment.
- Chronic lameness may result from poor fracture healing and adhesions that develop between the DDFT and the navicular bone. A non-calcified fibrous union can still be evident years after the fracture occurred in many horses.

### *Prognosis*

- The prognosis is considered guarded to poor for horses with complete navicular bone fractures to return to athletic performance.
- In general, horses with fractures in the hindlimb are considered to have a better chance to return to performance than those affected in the forelimbs.
- Complete radiographic healing seems to occur infrequently, even in horses that become sound (Figure 5.10).
- Horses with avulsion fractures and other radiographic signs of navicular disease have a fair to guarded prognosis to return to soundness.

## INJURIES TO THE DDFT AND PODOTROCHLEAR APPARATUS

### *Introduction*

- Advances in ultrasound, CT, and MRI have enabled improved recognition of these problems.
- It is not an either/or situation as it appears common to have soft tissue and bony abnormalities co-existing in many lame horses with foot problems.
- The podotrochlear apparatus consists of the DSIL and the collateral suspensory ligaments (CSLs) of the navicular bone.
- The DDFT is bi-lobed within the foot and is the most commonly injured soft tissue structure in the foot. Abnormalities have been identified at the DDFT insertion to the distal phalanx (least common), the level of the navicular bone or CSLs (most common), more proximally in the pastern, or a combination of sites.

### *Etiology*

- Similar biomechanical forces and repetitive trauma to the palmar aspect of the foot associated with navicular disease most likely contribute to these injuries.
- Horses that jump or have a low heel hoof conformation may be at risk for injuries to the DDFT.
- Single-event traumatic "tearing" of the DDFT causing a true tendinitis also may occur, but is less common.
- The most common location of DDFT injuries is at the level of the navicular bone, and these can be true core lesions or sagittal splits, erosions, or abrasions.
- Abnormalities of the podotrochlear apparatus often are present in association with abnormalities of the navicular bone.

### *Clinical Signs*

- Often there is a history of an acute onset of moderate to severe lameness that may improve with rest and worsen with exercise. The lameness usually is unilateral and may worsen in a circle or when exercised on soft ground.
- Pain may be elicited with deep palpation of the DDFT between the collateral cartilages of the heels. Increasing tension on the DDFT with the navicular wedge test may accentuate the lameness.
- The lameness will improve with a PD nerve block, but the lameness may not be completely abolished until a higher block is done. Anesthesia of the DIP joint was more effective in alleviating the lameness in horses with DDFT lesions than was the PD block in one study.
- The response to perineural anesthesia may depend on the location of the lesion(s) and whether concurrent problems exist in the foot.

### *Diagnosis*

- A definitive diagnosis of a DDFT injury of the foot is best determined with an MRI (Figures 4.71, 4.72, 5.6B).
- Typically, horses have minimal to no radiographic abnormalities. Ectopic mineralization may be seen in some horses with DDFT lesions but is not necessarily correlated with active tendinitis of the DDFT. Enthesiophytes involving the podotrochlear apparatus attachments to the navicular bone and erosive lesions of the flexor surface of the navicular bone may suggest concurrent injury but cannot provide a definitive diagnosis.
- Ultrasound can be used to diagnose some injuries to the DDFT and the podotrochlear apparatus, depending on the location of the lesion.
- Contrast CT also is an option to document abnormalities in the DDFT if an MRI is unavailable.

### *Treatment*

- The most important aspects of treatment of soft tissue injuries of the foot are rest, rehabilitation, and corrective foot care to correct hoof imbalances. Rest and rehabilitation is usually performed over a minimum of six months for DDFT injuries and may be necessary for even longer depending on the lesion.
- Types of shoes that may benefit horses with DDFT injuries include egg-bar shoes and shoes and/or pads to elevate the heels.
- Intrasynovial treatment of the navicular bursa with corticosteroids and hyaluronan (HA) can be performed if the lesion is within the bursa or if concurrent bursitis is present.
- Intralesional treatment with platelet-rich plasma (PRP), stem cells, and other biologics may be performed if the lesion is accessible to this technique.
- A technique to inject the insertion of the DDFT at the level of P3 has been described recently and can be used if the DDFT abnormality is in this location. A spinal needle is inserted at the depression between the heel bulbs at the level of the coronary band and is advanced until it contacts bone at the interface of the DDFT and P3, which is confirmed radiographically.
- Lesions at the level of the navicular bone may benefit from endoscopy of the navicular bursa and debridement of any torn tendon fibers (Figure 5.11).

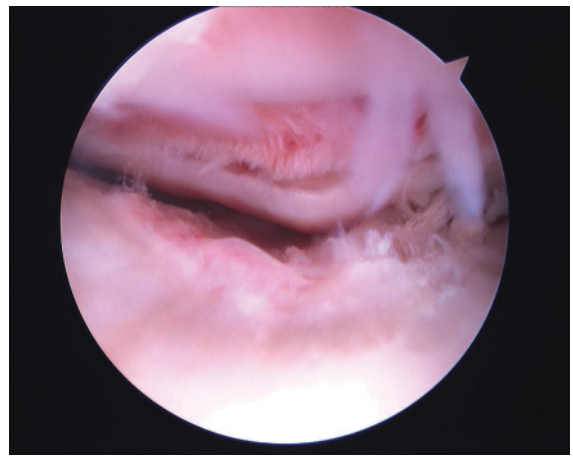


**Figure 5.11.** Endoscopic view of a flexor cortex lesion and fibrillation of the DDFT. The navicular bone is at the top of the image and the DDFT is on the bottom.

### *Prognosis*

- The prognosis for horses with soft tissue injuries of the foot is considered guarded to poor for return to athletic performance.
- Only 28% of horses with DDFT lesions returned to performance after six months rest in one study.
- Horses with DDFT injuries and concurrent navicular bone pathology are particularly problematic.
- Horses with DDFT lesions localized to the navicular bursa that are debrided endoscopically appear to have an improved prognosis (Figure 5.11).

## INJURIES TO THE COLLATERAL LIGAMENTS (CLs) OF THE DIP JOINT

### *Etiology*

- Lesions of the CL of the DIP joint were the second most common soft tissue injury in one MRI study and can occur alone or together with other injuries.
- Acute-onset or repetitive trauma is considered the most likely cause.
- Asymmetrical foot placement or foot imbalances may cause sliding and rotation of the distal phalanx relative to the middle phalanx, contributing to these injuries.
- Lesions within the CL of the DIP joint have been reported to be a primary degenerative process rather than inflammatory.

### Clinical Signs

- Often, there are few localizing clinical signs in horses with injuries to the CL of the DIP joint.
- The medial collateral ligament of forelimb is the most common site of the injury.
- Horses often have a history of a chronic forelimb lameness of variable severity that is worse in the circle. Phalangeal flexion usually is positive.
- Palpable swelling and pain of the ligament at its proximal attachment to the P2 may be present above the coronary band in severe cases.
- Effusion of the DIP joint is not a consistent clinical finding.
- Most horses improve with a PD nerve block but may not be completely sound until a more proximal block is performed. Only 40% of horses improved with intra-articular (IA) anesthesia of the DIP joint in one study.

### Diagnosis

- The best imaging modality to confirm an abnormality in the CLs of the DIP joint is MRI (Figure 5.12).
- Ultrasound can be used to diagnose some injuries depending on the location of the lesion. Negative findings do not rule out the presence of a lesion.
- Nuclear scintigraphy can be used to document focal increased radiopharmaceutical uptake at the insertion of the CL, suggesting an abnormality.
- Bone exostosis, lysis, or sclerosis may be seen with radiography at the insertion sites of the CL on the distal or middle phalanges but appear to be uncommon (Figure 5.13).

### Treatment

- Some form of rest, rehabilitation, and corrective trimming and shoeing are usually necessary.
- Intralesional treatment using platelet-rich plasma (PRP) and other biologicals can be used if the lesion is accessible above the hoof wall.
- Additional treatments include extracorporeal shock wave therapy, application of a half-limb or foot cast, and medication of the DIP joint.

### Prognosis

- The reported prognosis is variable. Two studies have reported that only 29% of horses treated with rest and rehabilitation returned to athletic function.
- A more recent study indicated that 60% of treated horses (12 of 20) returned to their previous level of exercise.

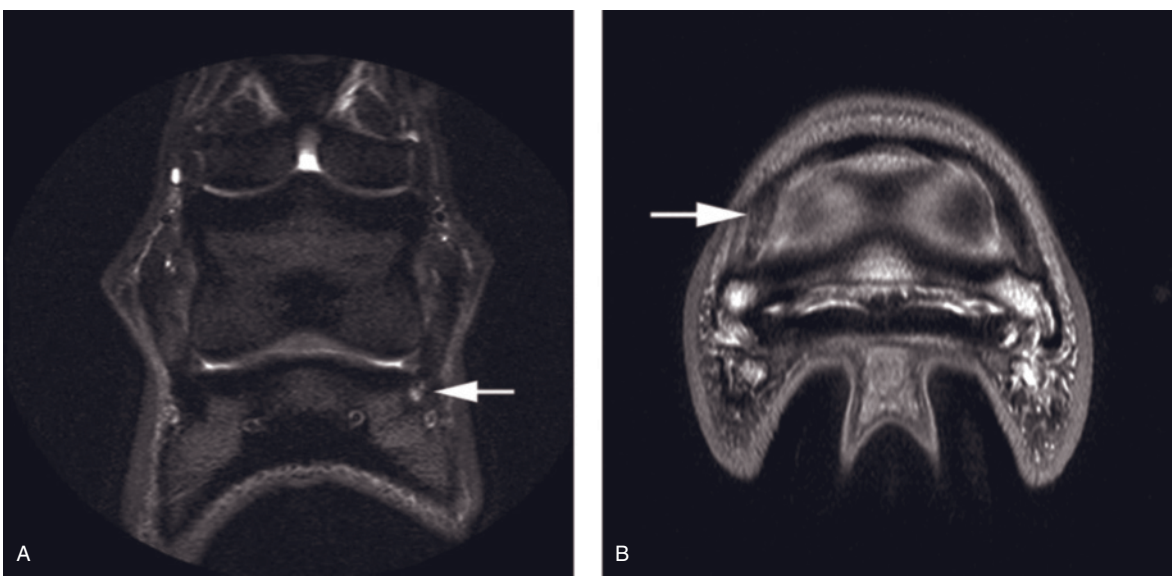



**Figure 5.12.** Frontal STIR (A) and PD axial (B) MR images of the foot of a 12-year-old Quarter horse with abnormalities within the medial collateral ligament of the DIP joint (arrows).

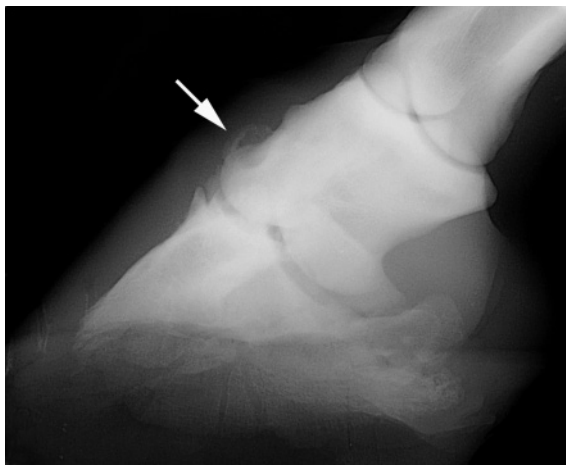


**Figure 5.13.** Bony proliferation on the dorsomedial aspect of P2 on this oblique radiograph (arrow) is suggestive of an injury to the CL of the DIP joint.

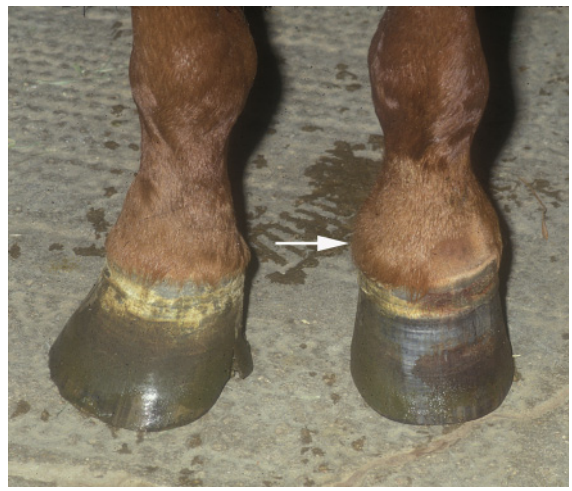


**Figure 5.14.** Effusion within the DIP joint can be seen and palpated as swelling just above the coronary band.

- Horses with multiple soft tissue injuries would most likely have a lower prognosis than those with single isolated lesions, but this has not been documented.

## OSTEOARTHRITIS (OA) OF THE DIP JOINT

### *Etiology*

- Osteoarthritis/synovitis/capsulitis of the DIP joint often is referred to as "low ringbone."
- It may occur as a primary problem or secondary to other injuries within the foot.
- Primary OA can be due to acute or repetitive trauma to the joint comparable to any articulation in the horse. Horses with a broken pastern axis (forward or backward) and other types of hoof imbalances appear particularly prone to this type of trauma.
- Excessive strain of the attachments of the long or common digital extensor tendon to the extensor process may contribute to periostitis and enthesiophyte formation along the dorsal aspect of the joint.
- Secondary OA can occur from other lameness conditions that involve the DIP joint such as navicular disease, navicular bone fractures, articular fractures of the distal phalanx, subchondral cystic lesions (SCL) of the distal phalanx, osteochondral fragmentation within the joint, and desmopathy of the CLs of the DIP joint.

### *Clinical Signs*

- Effusion of the DIP joint usually is present in most horses with OA/synovitis/capsulitis of the DIP joint. Most times significant effusion of the DIP joint can be seen as a slight bulging just above the coronary band (Figure 5.14).
- With chronic or advanced disease the joint capsule may become thickened, resulting in a firm swelling just above the dorsal aspect of the coronary band (Figure 5.15).
- The joint may be painful to flexion and rotation, but this is uncommon unless the OA is advanced or secondary to another problem in the joint.
- The lameness is variable depending on the severity of the disease, and is often worse on hard ground, when circled, and after distal limb or phalangeal flexion.
- The lameness often is greatly improved and sometimes eliminated with a PD nerve block, but anesthesia at the base of the sesamoid bones may be required for complete resolution of the lameness.
- IA anesthesia of the DIP joint is not specific for problems within the joint, but using a small volume of anesthetic (6 mL or less) and observing for a change in lameness very soon after the injection (within 10 minutes) can improve the specificity of the block for the joint. A positive response to an IA block combined with a negative response to navicular bursa anesthesia often incriminates the joint as the primary problem area.

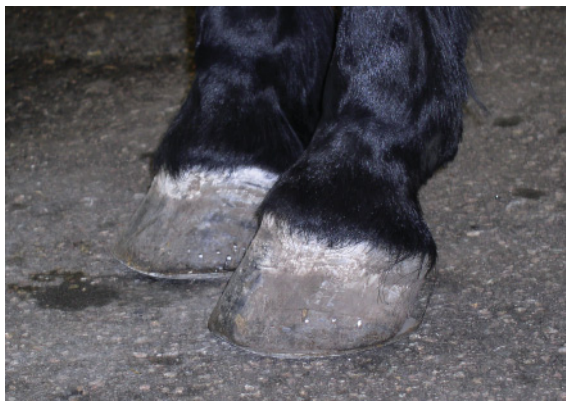


**Figure 5.15.** "Buttress foot" is a term used to describe horses with firm enlargements just proximal to the dorsal hoof wall. Usually this is due to fracture of the extensor process but may also be seen in horses with severe OA of the DIP joint.

### *Diagnosis*

- Radiography usually provides a definitive diagnosis of OA of the DIP joint. Close inspection of the extensor process, palmar/plantar aspect of distal P2, and dorsoproximal aspect of the navicular bone for osteophyte and enesthesiophyte formation is important.
- Horses with synovitis/capsulitis or early OA of the DIP joint may have no radiographic abnormalities. Do not over interpret the radiographic findings because there is much variation in the shape of the extensor process and enthesiophytes may not be associated with lameness.
- MRI is the most comprehensive imaging modality that can detect articular cartilage, subchondral bone, and soft tissue abnormalities of the DIP joint, if present.
- Arthroscopy may used to document articular cartilage or subchondral bone damage, but much of the joint surface of the DIP joint is not visible.

### *Treatment*

- Horses with primary OA/synovitis/capsulitis of the DIP joint are usually treated with a combination of intra-articular medication and corrective shoeing. Shortening the toe and moving the break-over farther palmarly often helps these horses, and using a rim pad may alleviate concussion to the joint.
- Direct medication of the DIP joint usually is more effective than systemic medications. One study reported that three weekly injections of the DIP joint with polysulfated glycosaminoglycan (PSGAG) was more effective in improving lameness associated with the DIP joint than methylprednisolone acetate (MPA) alone. The author often uses a combination of triamcinolone and HA in most horses.
- Treatment of horses with secondary OA of the DIP joint usually focuses on the underlying contributing problem. For instance, extensor process fractures of the distal phalanx should be removed in most cases and other articular distal phalanx fractures should be stabilized with corrective shoeing.
- Systemic joint medications such as IM PSGAG, IV hyaluronan, and nutraceuticals also may benefit these horses.

### *Prognosis*

- Horses with primary synovitis/capsulitis of the DIP joint usually have a very good prognosis to return to performance. However, one study indicated that only 30% of horses responded to treatment.
- The prognosis often is related to the severity of the radiographic abnormalities. Horses with advanced OA often respond less to any form of treatment or the lameness recurs more quickly.
- Horses with secondary OA of the DIP joint have a variable prognosis depending on the underlying problem.
- The development of radiographic signs of OA within the DIP joint does not preclude athletic performance. It is possible that the radiographic abnormalities within the DIP are over interpreted as to their influence on lameness.

## FRACTURES OF THE DISTAL PHALANX (P3, COFFIN BONE)

### *Introduction*

- Fractures of the distal phalanx can occur in any foot but most commonly affect the lateral aspect of the left forelimb and the medial aspect of the right forelimb in racehorses (Type I and II articular "wing" fractures).
- Type I fractures are nonarticular oblique palmar/plantar process (wing) fractures (Figure 5.16).
- Type II fractures are articular oblique palmar or plantar process (wing) fractures (Figure 5.16).
- Type III fractures are sagittal articular fractures that roughly divide the distal phalanx into two separate halves (Figure 5.16).

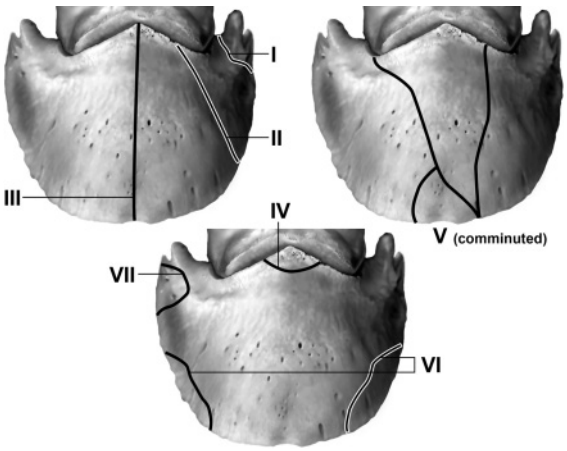



**Figure 5.16.** Classification of P3 fractures in horses. Reprinted with permission from Bertone AL: 1996. Fractures of the Distal Phalanx. *In:* Nixon AJ (ed) *Equine Fracture Repair.* Philadelphia, WB Saunders Co, 146–152.

- Type IV fractures are articular fractures involving the extensor process (Figure 5.16).
- Type V fractures are comminuted articular or nonarticular fractures and can be a variety of configurations (Figure 5.16).
- Type VI fractures are nonarticular solar margin fractures of the distal phalanx (Figure 5.16).
- Type VII fractures are nonarticular fractures of the palmar or plantar process of the distal phalanx in foals. These fractures begin and end at the solar margin and are usually triangular or oblong in shape (Figure 5.16).

## *Etiology*

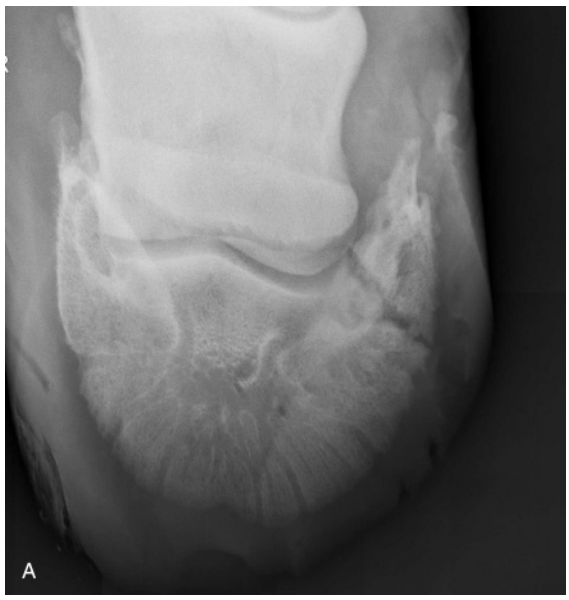
- Single event trauma such as kicking a solid object appears to be the predominant cause of P3 fractures in non-racehorses.
- Repetitive trauma leading the stress-related bone injury is the likely cause of P3 fractures in racehorses.
- Type VI fractures may be a type of pathologic fracture because they can occur concurrently with laminitis and pedal osteitis due to resorption of the apex of P3.
- Type VII fractures in foals are thought to occur from compression, either on the solar or dorsal cortex of the distal phalanx during weight-bearing or from tension forces generated by the DDFT.
- Type IV extensor process fractures may occur due to excessive tension on the common digital extensor tendon resulting in an avulsion fracture, or they may be developmental in origin (osteochondrosis lesion).

## *Clinical Signs*

- There is usually a history of an acute onset of a moderate to severe lameness (grade 4 to 5/5). Exceptions to this are solar margin fractures, type VII fractures in foals, and developmental type IV fractures of the extensor process. These horses are usually less lame.
- An increased digital pulse may be palpable and heat in the affected foot may be appreciated in the acute stage.
- DIP joint effusion often is present if the fracture is articular.
- Hoof tester examination usually reveals pain over the sole region and focal pressure over the fracture site usually induces a marked response. A negative hoof tester response does not rule out the presence of a chronic P3 fracture.
- Perineural anesthesia of the PD nerves or IA anesthesia of the DIP joint may aid in localizing the lameness to the foot region, but is usually only necessary in horses with chronic fractures.
- Horses with large chronic extensor process fractures may have enlargement of the dorsal aspect of the coronary band and abnormal growth of the dorsal hoof wall (it develops a "V" or triangular shape called a "buttress foot").

## *Diagnosis*

- Radiography is used to confirm the diagnosis and document the type and location of the fracture (Figure 5.17). Solar margin fractures are most easily identified on the 60° dorsoproximal-palmarodistal view. Extensor process fractures are usually identified on the lateral view (Figure 5.18).
- Some non-displaced or stress-related fractures in racehorses may not be apparent on the initial radiographs. Radiographs should be repeated in one to two weeks, or nuclear scintigraphy can be used to help identify radiographically occult fractures of P3.
- Computer tomography (CT) can be used to document occult fractures in the palmar/plantar processes or to confirm the fracture configuration in comminuted P3 fractures (Figure 4.79).



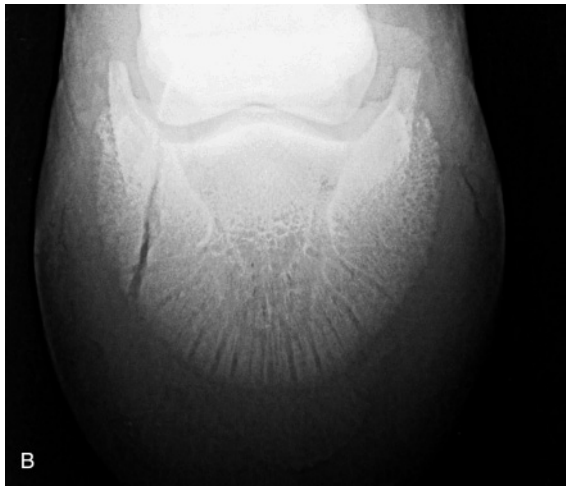




**Figure 5.17.** Two variable sized Type II "wing" fractures of the P3. These are the most common type of P3 fracture.

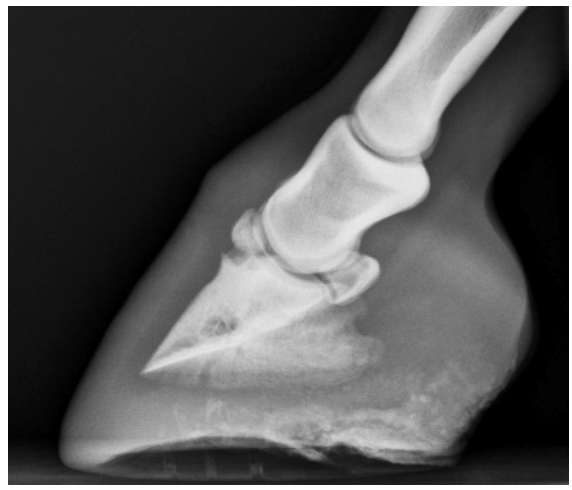


**Figure 5.18.** Type IV P3 fractures involve the extensor process.

### *Treatment (Table 5.1)*

- Type I is best treated with confinement and methods to prevent hoof expansion (shoe or foot cast), but horses also may respond to confinement and rest alone.
- Type II fractures in foals less than six months of age should be treated with stall confinement for at least six to eight weeks. Most adult horses are treated with confinement and methods to restrict hoof expansion (rim shoe or shoe with clips or a foot cast; Figure 5.19). Screw placement can be very difficult with this fracture type and is often only considered in horses with large wing fractures.
- Type III fractures can be treated similarly to Type II fractures. Acute Type III fractures in adult horses are usually the best candidates for surgical repair using lag screw fixation. The primary advantages are less risk of secondary OA developing in the DIP joint and faster healing of the fracture due to surgical compression.
- Type IV fractures are treated with surgical repair with a lag screw for acute fractures or surgical removal of the fracture/fragment in chronic fractures or OCD lesions (Figure 5.20). Acute fractures are rare and chronic fractures usually never heal to the parent bone.
- Type V fractures are best treated with confinement and methods to prevent hoof expansion (shoe or foot cast).
- Type VI, solar margin, fractures are usually treated with corrective shoeing (wide-web shoes, shoes and full pads, or shoes with rim pads) and stall or paddock rest for four to 12 months. Strict immobilization with bar shoes and quarter clips is not necessary.
- Type VII fractures in foals are usually treated satisfactorily with confinement alone for six to eight weeks. Application of restrictive external coaptation (e.g., bar shoe or acrylic) to the hoof is not recommended.
- The majority of horses with P3 fractures are treated with confinement and corrective

**Table 5.1.** Types of distal phalanx fractures.

| Fracture type | Location | Articular | Recommended treatment | Prognosis |
|---|---|---|---|---|
| I | Palmar/plantar process | No | Confinement ± shoeing | Very good to excellent |
| II | Oblique fractures of palmar/plantar process ("wing" fractures) | Yes | Confinement + shoeing (Foot cast instead of shoe) Lag screw repair of large type II fractures | Guarded to good |
| III | Midsagittal fracture | Yes | Confinement + shoeing (Foot cast instead of shoe) Best candidate for lag screw repair | Unpredictable; guarded? |
| IV | Extensor process (variable size) | Yes | Removal in most cases regardless of size: arthroscopy/arthrotomy Lag screw repair: acute cases | Small: excellent Large: good |
| V | Comminuted | Yes or no | Confinement + shoeing (Foot cast instead of shoe) Removal if secondary to infection | Unpredictable Guarded to good? |
| VI | Solar margin | No | Confinement + protective shoeing (wide-web shoes or shoes with full or rim pads) | Very good |
| VII | Palmar/plantar process; begins and ends at solar margin | No | Primarily in foals Confinement alone: no shoeing | Very good to excellent |

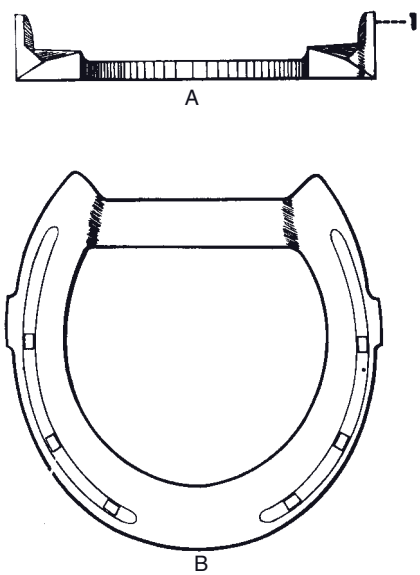



**Figure 5.19.** Full-bar shoe that can be used to treat horses with distal phalanx fractures. (A) Rear view of shoe showing quarter clips. (B) Ground surface view of the shoe showing full bar and quarter clips welded to the shoe.

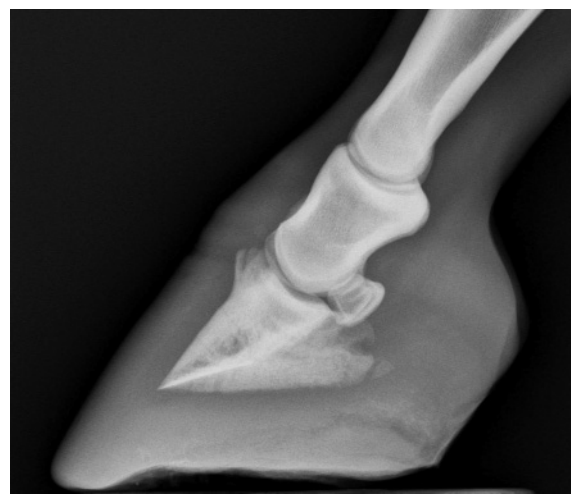


**Figure 5.20.** Lateral radiograph of the horse in Figure 5.18 following arthroscopic removal of an extensor process fracture.

shoeing aimed at immobilizing the fracture and preventing expansion of the hoof wall. However, a foot cast may serve the same purpose as the shoe.
- Types of shoes that may be used include a bar shoe with clips, continuous rim-type shoe, or a contiguous clip shoe. In most cases the foot should remain in one of these shoes for six to eight months and 10 to 12 months of inactivity may be necessary for clinical improvement.
- Many P3 fractures are very slow to heal and may never develop radiographic bone union.
- OA of the DIP joint is a sequel to articular P3 fractures but may not preclude athletic soundness.

### *Prognosis*

- The prognosis usually is very good for all ages of horses for nonarticular P3 fractures (Types I, V, VI and VII).
- Foals with Type VII P3 fractures have an excellent prognosis for return to performance and fracture healing is expected in about eight weeks.
- A 50% to 81% return to soundness has been reported for horses with Type II wing fractures, depending on their occupation.
- The prognosis for small extensor process fractures treated by arthroscopic removal is excellent. Removal of chronic extensor process fragments also results in a good prognosis; as 8/14 cases returned to their intended use in one report.
- It remains unknown if horses with Type III fractures have an improved prognosis with lag screw fixation compared to confinement and corrective shoeing.

## PEDAL OSTEITIS (PO)

### *Etiology*

- Pedal osteitis (PO) is an inflammatory condition of the foot that results in demineralization of the distal phalanx.
- The two recognized classifications of PO are nonseptic and septic.
- Nonseptic PO is typically described as persistent, chronic inflammation of the foot that may be associated with severe or chronic sole bruising due to repeated concussion during exercise on hard surfaces. Other potential causes include persistent corns, laminitis, penetrating wounds, and conformational faults.
- Septic PO refers to bacterial infection within the distal phalanx, usually from deep penetrating wounds or from hematogenous spread in foals. Other causes include chronic severe laminitis, subsolar abscesses (most common), solar margin fractures, deep hoof wall cracks, and avulsion hoof injuries. A sequestrum may develop in the distal phalanx as the osseous infection progresses.

### *Clinical Signs*

- Nonseptic PO most commonly affects the forelimbs and may be unilateral or bilateral. The severity of the lameness is variable but is often worse after exercise, work on hard surfaces, or directly after trimming and shoeing. Focal or diffuse sensitivity of the sole is often found with hoof testers. Perineural anesthesia of the PD nerves usually eliminates the lameness.
- Septic PO occurs most commonly in the forelimbs in adult horses and in the hindlimbs in foals. The severity of lameness in horses with septic PO is usually greater than that seen with nonseptic PO. Increased hoof temperature, prominent digital pulses, and focal sole pain with hoof testers are common in the affected limb. Perineural anesthesia of the PD nerves may not eliminate the lameness in all horses with septic PO.

### *Diagnosis*

- Radiography is the imaging modality of choice.
- The radiographic signs associated with nonseptic PO include:
  1. Demineralization
  2. Widening of the nutrient foramina at the solar margin
  3. Irregular bone formation along the solar margins of the distal phalanx, and type VI fractures (Figures 5.21, 5.22).
- Radiographic signs of septic PO usually are consistent with those of bone infection such as focal osteolysis, decrease in bone density, and sequestration. In foals with septic PO, evidence of localized lysis or focal loss of bone density were observed at the toe (14/22 cases), extensor process (5/22 cases), or the palmar/plantar process (3/22 cases) of the distal phalanx.

### *Treatment*

- Treatment of nonseptic PO is aimed at reducing the inflammation within the distal phalanx,

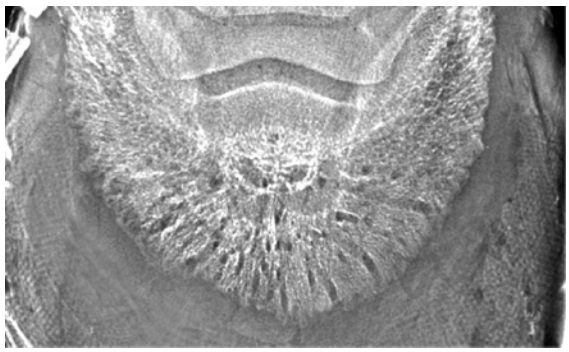


**Figure 5.21.** Horse with pedal osteitis characterized by demineralization of the distal phalanx.

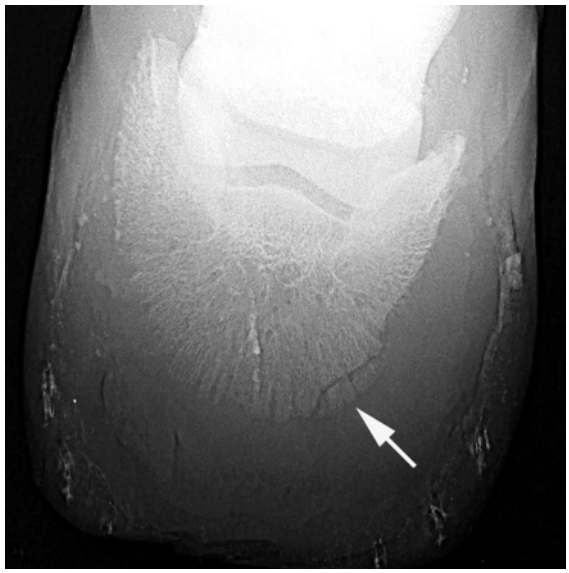


**Figure 5.22.** Type VI fractures are also referred to as solar margin fractures of P3 (arrow) and can be secondary to osteopenia.

minimizing concussion to the foot, and eliminating the inciting cause. A wide-web shoe with a deeply concaved solar surface, shoes with rim pads, or shoes with full pads have been recommended to prevent the injured sole from contacting the ground. Rest and avoiding exercise on hard surfaces are usually indicated until the lameness subsides.

- Treatment of septic PO usually involves systemic and local antimicrobials (regional limb perfusion) and surgical debridement of the infected bone. One study suggested that up to 24% of the distal phalanx could be removed without long-term adverse effects.

### *Prognosis*

- The prognosis for nonseptic PO often is very good if the condition is of a relatively short duration and the exercise environment can be controlled. It is less favorable if the disease is chronic and the horse must continue to compete on hard surfaces, or if the condition is secondary to chronic laminitis.
- The prognosis for horses with septic PO appears very good to excellent. Of 33 cases of septic PO in which follow-up was available, all 33 horses returned to their intended use. In other studies, 7/9 horses returned to their intended use, and 13/18 had a favorable outcome despite a high incidence of sequestra and fractures. Foals with septic PO also have a good prognosis; 86% of the foals survived, 16/22 foals reached racing age, and 11 of these raced.

## SUBCHONDRAL CYSTIC LESIONS OF THE DISTAL PHALANX (P3)

### *Etiology*

- Subchondral cystic lesions (SCLs) of the distal phalanx are uncommon and can affect a wide variety of horse breeds of all ages.
- The cause of most SCLs in the horse is either trauma or developmental such as an osteochondrosis (OC) lesion.
- Most SCLs of P3 communicate with the joint surface, although communication with the adjacent joint space can be variable.

### *Clinical Signs*

- The forelimbs are more frequently affected than the hindlimbs.
- A history of an acute onset of lameness may be present, but more often the lameness is chronic and intermittent. The lameness may subside with rest and recur with exercise, and the severity can be variable.
- There are often no palpable abnormalities (including hoof tester examination) except effusion within the DIP joint.
- A PD nerve block improves the lameness in most cases, but a basi-sesamoid block may be needed for complete resolution of the lameness. Intrasynovial anesthesia of the DIP joint usually eliminates the lameness, especially if the SCL is articular.

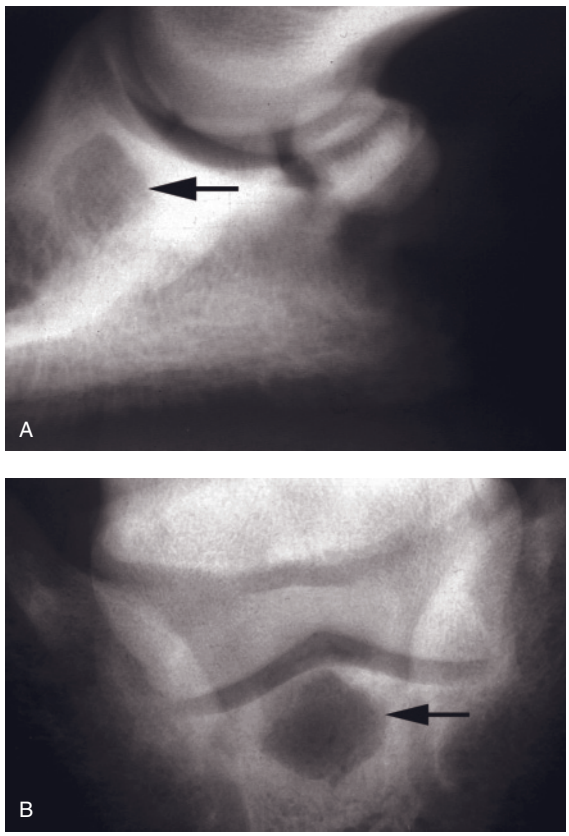



**Figure 5.23.** Lateral (A) and dorsopalmar (B) radiographs of a horse with SCL of P3 (arrows).

### *Diagnosis*

- Radiography usually confirms the diagnosis. The SCLs are variable in size and are usually identified within the body of the distal phalanx (Figure 5.23). In one study, 18/27 SCLs were located centrally in the distal phalanx and communication with the DIP joint was observed in all cases.
- MRI or CT can better define the exact location and the size of the articular opening of the SCL into the joint surface than radiography.

### *Treatment*

- Recommended treatments have included:
  1. Confinement followed by increasing exercise
  2. IA medication of the DIP joint
  3. Transcortical drilling
  4. Extra-articular surgical curettage
  5. Arthroscopic debridement
- Most horses respond only transiently to IA medications and debridement through hoof wall windows has been complicated by recurrent abscessation and lameness.
- A dorsal arthroscopic approach for IA debridement has been described recently and is considered the treatment of choice. However, some SCLs in the distal phalanx may be inaccessible with the arthroscope.

### *Prognosis*

- Variable results have been reported with extra-articular debridement of these lesions, and these techniques are often complicated by infection and continued lameness.
- Ten of 11 (91%) horses treated with arthroscopic debridement of a distal phalanx SCL returned to athletic soundness.
- Arthroscopic debridement of SCLs of the distal phalanx should provide a superior outcome in any age horse compared to extra-articular approaches.

## OSSIFICATION OF THE COLLATERAL CARTILAGE OF THE DISTAL PHALANX (SIDEBONE)

### *Introduction*

- Ossification of the collateral cartilages of the distal phalanx is relativity common in larger breeds such as draft horses, Warmbloods, Finnhorses, and Brazilian jumpers (Figure 5.24).
- The fore feet appear to be more commonly involved than the hind feet, females more than males, and the lateral cartilage often ossifies more than the medial cartilage.
- The clinical significance of the condition remains questionable.

### *Etiology*

- The specific cause(s) of sidebones is/are not clear.
- It is considered partly hereditary in certain horse breeds in Australia, Finland, and Sweden.
- Hoof concussion causing trauma to the cartilage, poor conformation, particularly base narrow, and poor trimming and shoeing have been proposed as inciting causes.
- It has been suggested that prolonged exercise and/or racing may have some preventative influence on ossification of the collateral cartilages.

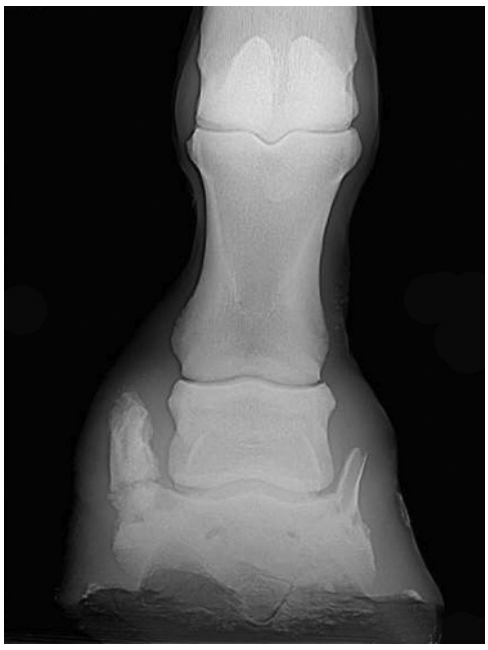


**Figure 5.24.** Standing dorsopalmar radiograph demonstrating a large uniaxial sidebone that was thought to be contributing to lameness.

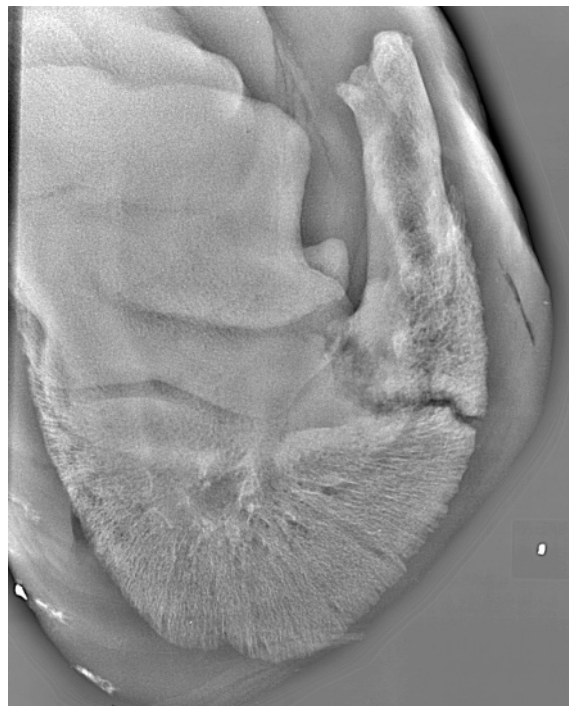


**Figure 5.25.** Type II articular fracture that was associated with a large sidebone of P3.

### *Clinical Signs*

- Lameness due to sidebone is considered rare and the clinical significance of radiographic apparent ossification is questioned.
- Large sidebones have been seen in a few horses associated with Type II distal phalanx fractures by the author, and were thought to contribute to the fracture (Figure 5.25).
- A large sidebone may be visually apparent as an enlargement of the lateral and medial dimensions of the pastern region. Palpation may reveal firmness to the cartilage and pain with digital pressure. If palpable pain is present, the sidebone may be contributing to the lameness or may be associated with a secondary fracture of the distal phalanx.
- Sidebone may accompany other lameness conditions of the palmar heel region (e.g., navicular syndrome) and may be mistaken for the cause.

### *Diagnosis*

- Radiography usually reveals the extent of the ossification of the cartilage or cartilages.
- A fracture in the ossified cartilage can occur but is rare.
- Documenting that sidebone is the cause of lameness can be difficult. Asymmetrical swelling of pastern region, pain on palpation of the collateral cartilage, and improvement of the lameness with a uniaxial PD nerve block would be suggestive of a problem in this region.
- MRI may help determine whether the abnormalities within the cartilage may be contributing to the lameness.

### *Treatment*

- If sidebone is suspected as the cause of lameness, conservative treatment with confinement, topical 1% diclofenac sodium cream (Surpass®), and oral administration of NSAIDs is recommended initially. Any contributing foot problems should be addressed.
- If the lameness persists and sidebone is considered the cause of the lameness, a PD neurectomy can be performed. Surgical removal of a suspected fractured sidebone is not recommended.
- Horses with sidebone and a secondary distal phalanx fracture are treated with corrective shoeing and confinement, similarly to a horse

with a Type II P3 fracture alone (see P3 fractures).

### *Prognosis*

- The prognosis is difficult to predict because this condition is thought to rarely cause lameness.

## SOLE BRUISES, CORNS, AND ABSCESSES

### *Introduction*

- A bruise results from the rupture of blood vessels in the dermis (corium or sensitive tissue) beneath the sole, frog, or hoof wall. With time the hemorrhage spreads into the deep layers of the epidermis and becomes visible as the hoof grows.
- A corn is a bruise that involves the tissues of the sole at the angle formed by the wall and the bar. This site is often referred to as the "seat" of the corn. Corns occur most frequently on the inner angle of the front feet and are rarely found in the hind feet.
- If the bruised site becomes infected a subsolar abscess develops.

### *Etiology*

- Trauma to the sole is the cause of most sole bruising. Horses with flat feet, thin soles, and soft soles appear to be predisposed to sole bruising. Any form of shoeing that concentrates weight-bearing on the sole is likely to cause bruising.
- Corns are usually caused by pressure from horseshoes or when a stone becomes wedged between the shoe and sole. Corns are rare among horses that are not shod. Bending the inside branch of the shoe toward the frog to prevent pulling or stepping off the shoe can result in direct pressure to the sole leading to bruising.

### *Clinical Signs*

- Most sole bruises occur at the toe or quarter regions and corns occur at the angle of the wall and bar.
- The degree of lameness can be variable depending upon the severity and type of the bruise or corn. If the bruise is acute, the hoof may appear warmer and an increased digital pulse may be present. Hoof testers often identify a focal site of pain at the site of the bruise or corn.
- Horses with foot abscesses are typically very lame and often non-weight-bearing. Increased heat is often palpable in the foot and distal limb, and an increased digital pulse is commonly found. Hoof tester pain is typically severe and in some cases digital pressure at the site of the abscess causes a painful response.

### *Diagnosis*

- A tentative diagnosis often can be made based on the history and clinical signs.
- If pain is localized to the foot without obvious external abnormalities, the shoe should be removed and the sole explored.
- Acute sole bruises may not be readily apparent, because the hemorrhage has not migrated far enough distally. Chronic bruises are usually visible as a "stippled" reddened region.
- Sole abscesses may have a small defect in the sole where the abscess is trying to break through. Removing a small area of sole around this defect may reveal purulent material, confirming a subsolar abscess.
- Some subsolar abscesses may be seen radiographically as a gas pocket within the sole. However, many are not visible and the lack of radiographic abnormalities does not rule out an abscess.
- Chronic sole bruising may be associated with nonseptic pedal osteitis.

### *Treatment*

- Many bruises often resolve without treatment if the source of the trauma is removed. The horse should be removed from heavy work and the environment changed so that the horse is not worked on rough ground.
- If the horse must be used, the sole can be protected with a full pad applied under the shoe. The pad should be placed to avoid pressure to the bruised site. Wide-web shoes also may be beneficial to relieve pressure on the sole. Additionally, light paring of the sole overlying the bruise often relieves the pressure and makes the horse more comfortable.
- Drainage is the key to treating suppurative bruises and other subsolar abscesses. Only a small amount of sole overlying the abscess should be removed to permit ventral drainage. The foot can then be soaked in antiseptic solution if desired, and the foot bandaged. Once the abscess has resolved, the sole can be protected with protective boots or shoes until the defect has completely keratinized.

- In cases in which shoeing is contributing to the bruising, removal of the shoe may be all that is necessary. To prevent shoes from causing corns, the heels of the shoe should extend well back on the buttresses and should fit full on the wall at the quarters and heels.

### *Prognosis*

- The prognosis is usually very good for horses suffering from a single traumatic episode and in horses with good foot conformation.
- The prognosis is reduced in horses with poor hoof conformation that are continually worked on hard ground because recurrence is common.
- Horses with routine foot abscesses also have a very good prognosis provided the infection does not involve deeper structures in the foot.
- It should always be remembered that subsolar abscesses can be associated with other conditions of the foot such as keratomas, chronic laminitis, and septic pedal osteitis (Figure 5.26).

## CANKER

### *Introduction*

- Equine canker is described as an infectious process that results in the development of chronic hypertrophy of the horn-producing tissues. It also has been described as a chronic hypertrophic, moist pododermatitis of the epidermal tissues of the foot.

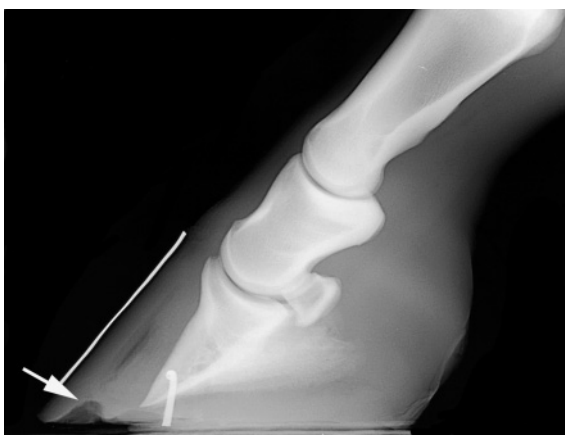


**Figure 5.26.** This horse had what was thought to be a routine abscess at the toe but a lateral radiograph revealed chronic laminitis.

- The infection is thought to cause abnormal keratin production or dyskeratosis, which is seen as filamentous fronds of hypertrophic horn (Figure 5.27).
- The disease is thought to be due to unhygienic environmental conditions and is especially common in draft horses.
- Canker can be misdiagnosed as "thrush," particularly in the early course of the disease. The distinguishing feature of thrush is that primarily loss of frog tissue usually can be differentiated from the proliferative nature of canker.

### *Etiology*

- Affected horses often have a history of being housed on moist pastures year round or in wet, unhygienic conditions. Horses standing in urine-, feces-, or mud-soaked bedding appear to be at risk.
- The causative anaerobic Gram-negative organisms are thought to be *Fusobacterium Necrophorum* and one or more *Bacteroides* spp.

### *Clinical Signs*

- Lameness usually is not present in early stages of the disease because the superficial epidermis is primarily involved.

**Figure 5.27.** Small, pale, demarcated growth along the caudal aspect of the frog that could be consistent with early canker.

- Early stages of canker may present as a focal area of granulation tissue in the frog that bleeds easily when abraded. Without treatment, it may spread to the other structures of the hoof.
- Examination of the foot usually reveals a fetid odor, and the frog, which may appear intact, has a ragged proliferative filamentous appearance. The proliferative frog may have numerous small finger-like papillae of soft off-white material that have a cauliflower-like appearance. The epidermal tissue of the frog is usually friable, will bleed easily when abraded, and may be extremely painful when touched (Figure 5.27).

### *Diagnosis*

- Often a presumptive diagnosis can be made based on the physical findings of a moist exudative pododermatitis with characteristic hypertrophic filamentous fronds involving the frog and surrounding tissue.
- It can be confirmed with a biopsy, but this is seldom performed by most clinicians.
- Cultures are rarely performed because a mixed population of bacteria is often found on the epidermis of the frog.

### *Treatment*

- Multiple treatment protocols have been described, none of which is consistently effective.
- Treatment principles include:
  1. Early recognition of the problem
  2. Thorough debridement of the lesion
  3. Methodical topical treatment
  4. Keeping the wound clean and dry until the defect begins to cornify
- Topical treatments include chloramphenicol, metronidazole powder, 2% metronidazole ointment, a mixture of ketoconazole, rifampin and DMSO, and a mixture of 10% benzoyl peroxide in acetone and metronidazole powder. The latter topical treatment was reported to successfully treat 54 cases of canker with minimal recurrence.
- Topical medications usually are applied directly to the debrided area; direct contact of the medication to the defect is important for success.
- Keeping the wound clean and dry with bandages or a treatment plate (Figure 5.28) and maintaining the horse in a dry environment are critical aspects of aftercare.

**Figure 5.28.** Treatment plate that can be very beneficial in treating canker. Keeping the foot clean and dry is a very important aspect of treatment.

- The duration of treatment may be several weeks to months, depending on the stage of the disease.

### *Prognosis*

- The prognosis is favorable for complete resolution of the problem if treatment is instituted early in the course of the disease.
- Advanced cases of canker that invade the sole, bars, and hoof wall, and those that involve multiple limbs, remain very difficult to treat.

## THRUSH

### *Etiology*

- Thrush is a degenerative condition of the frog involving the central and lateral sulci; it is characterized by the presence of black necrotic exudate and a foul odor.
- Contributing factors are wet, unhygienic stable conditions, especially when horses stand in urine- and manure-soiled bedding, neglect of daily foot care, and lack of exercise.
- Inadequate or improper trimming and shoeing, which promote long contracted heels and deep sulci, also contributes to the risk of infection.
- Although no specific organism has been identified as the cause, *Fusobacterium necrophorum* is commonly isolated.

### *Clinical Signs*

- The hindlimbs are most frequently involved.
- There is usually an increased amount of moisture on the bottom of the foot and a black, odiferous discharge in the sulci of the frog.

The affected sulci of the frog are often deeper than normal and the frog may be undermined and detached from the underlying tissue.
- Lameness is usually present in severe cases that involve the corium, and swelling of the distal limb may be seen.

### *Diagnosis*

- The diagnosis is usually based the presence of black, odiferous discharge in the sulci of the frog together with the loss of the frog.
- Thrush should be differentiated from canker; both conditions can occur in similar types of horses.

### *Treatment*

- Early cases usually respond to debridement of the diseased tissue and topical application of an astringent with or without foot bandages.
- Astringents that can be used include copper sulfate, 2% iodine alone or mixed with phenol, methiolate, and 10% formalin. These treatments should be repeated until the infection is controlled.
- The horse should be kept in a dry, clean stall or in a dry yard. Repeated trimming of the frog may be required before the infection is controlled.
- Severe cases of thrush are treated in a similar manner as above except debridement of the diseased undermined tissue is more extensive.
- Prevention is superior to treatment and is most important for horses confined to stalls for prolonged periods. Proper foot care is critical because horses with overgrown hooves are more susceptible to the disease.

### *Prognosis*

- The prognosis is good if the disease is diagnosed early before the foot has suffered extensive damage.
- The prognosis is less favorable if the infection is extensive and involves the corium.

## WHITE LINE DISEASE

### *Introduction*

- White line disease has been described as a keratolytic process on the solar surface of the hoof. It is characterized by progressive separation of the inner zone of the hoof wall.
- White line disease differs from laminitis because it does not involve the sensitive tissue beneath the hoof wall. The separation occurs in the non-pigmented horn between the stratum medium and stratum internum.
- It usually begins at the solar surface of the hoof and most frequently affects the toe region. The disease progresses with varying heights and configurations proximally toward the coronary band but never involves the coronary band (Figure 5.29).
- It has numerous other names such as seedy-toe, yeast infection, hoof-wall disease, environmentally induced separations, onychomycosis, and Candida.

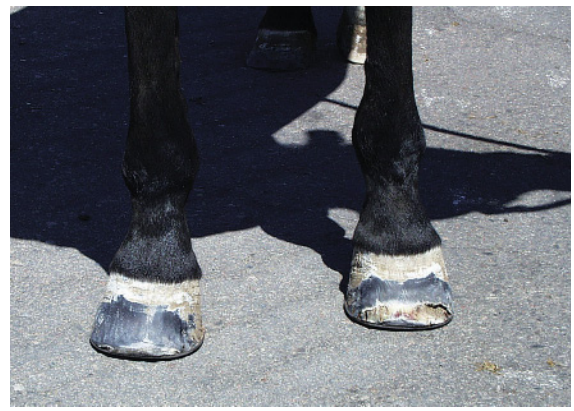


**Figure 5.29.** Flaky, chalky material beneath the dorsal hoof wall is often characteristic of white line disease.

### *Etiology*

- Proposed causes include mechanical stress on the hoof wall associated with long toes and poor hoof conformation, environmental conditions such as excessive moisture or dryness that affects the inner hoof-wall attachment, toxicity associated with selenium, and infection of the white line with bacteria and/or fungi.
- Pathogens usually isolated include a mixed flora of bacteria and *Pseudoallsheria*, *Scopulariopsis,* and *Aspergillus* fungi. However, it is currently debatable whether bacteria and fungi found with white line disease are the primary cause or simply secondary opportunists.

### *Clinical Signs*

- Horses with white line disease will have variable to no lameness, and the condition is often only found during routine trimming.
- In the early stages, the only noticeable change may be a small powdery area located just

dorsal to the hoof wall/sole junction. Other early signs may include sole pain with hoof testers, occasional heat, and increasingly flat soles.
- With time the hoof separation enlarges and the hoof wall growth may slow and be of poor consistency. Exploration of the inner hoof wall often reveals a separation that is filled with white/gray powdery horn material and debris (Figure 5.29).
- The hoof defect may progress up the dorsal hoof wall. Severe undermining of the hoof wall at the toe region can resemble chronic laminitis but the hoof separation is within the hoof wall and does not involve the sensitive laminae.

### *Diagnosis*

- The diagnosis is usually made on the basis of clinical findings. The sole/wall junction will usually be wider than normal and have a chalky texture. Further examination dorsal to the white line will reveal a concavity that contains white/gray powdery horn material and occasionally black serous drainage.
- Radiographs are always recommended to rule out the possibility of other bony abnormalities such as chronic laminitis or pedal osteitis. A gas line along the dorsal hoof wall may be seen with both white line disease and chronic laminitis.
- Other diagnostics that can be performed but are typically unrewarding include bacterial or fungal cultures and biopsies of the hoof wall.

### *Treatment*

- Therapy is directed toward protecting and unloading the damaged section of foot with therapeutic shoeing combined with removing the hoof capsule over the affected area. The type of therapeutic shoe and its method of attachment are dictated by the extent of the damaged hoof wall.
- Topical disinfectants/astringents are often applied after hoof wall resection.
- Systemic medical treatment is considered unnecessary in most cases and is of no value without resection of the damaged hoof wall.
- In mild to moderate cases, local debridement and regular shoeing to protect the sole are often all that is required. A full-support-bar shoe is recommended to protect and unload the damaged section of the foot if extensive removal of the hoof wall is required.
- Repeat debridement of the lesions may be required and the shoeing interval should be four weeks or less. The feet should be kept as dry as possible and the hoof wall defects should be covered with a light bandage to keep them clean.
- The duration of treatment depends on the amount of wall removed, but most horses can return to work when the surface of the defect has cornified.

### *Prognosis*

- Most horses with white line disease have a very good prognosis with local debridement and corrective shoeing.
- There is no known medical treatment and the condition is unlikely to improve unless the affected, undermined hoof wall is removed.

## PENETRATING INJURIES OF THE FOOT

### *Etiology*

- Penetrating injuries of the foot are usually caused by the horse stepping on (bottom of the foot) or contacting (coronary band, heel bulbs, and pastern region) a sharp object (Figure 5.30).
- Those that penetrate the central third of the frog, the coronary band, and the heel bulbs are at greatest risk to involve deeper vital structures (Figure 5.31).

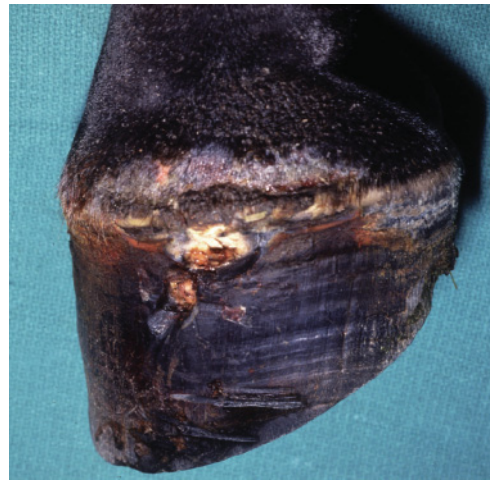


**Figure 5.30.** This horse had a draining tract at the coronary band but no abnormalities could be found on the solar surface of the foot. Exploration of the tract revealed a small piece of wood.

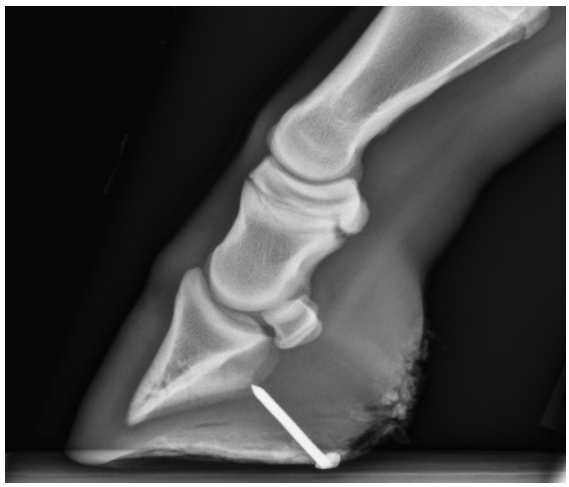


**Figure 5.31.** Taking radiographs of the foot prior to removing the foreign body can help identify the direction and depth of the puncture.

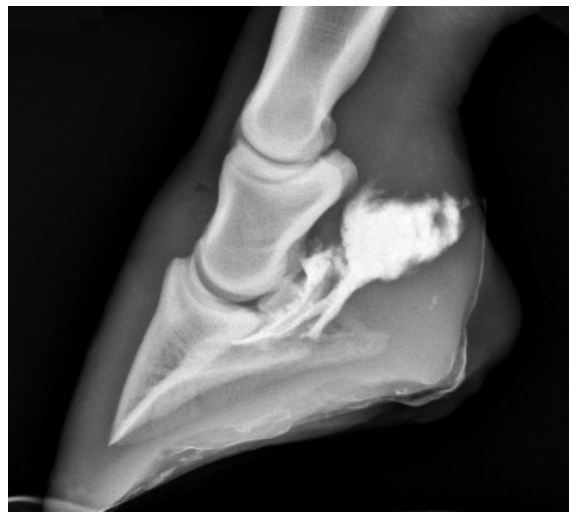


**Figure 5.32.** Contrast radiography can be helpful to document synovial involvement in horses with chronic penetrating injuries of the frog and sole. The puncture in this horse had completely healed but the horse developed an abscess just above the heel bulb. Contrast injected into the abscess communicated with the navicular bursa.

- Injuries elsewhere on the bottom of the foot will most likely only damage the digital cushion or the solar surface of the distal phalanx and are usually less problematic.

### *Clinical Signs*

- The clinical signs vary depending on the injury's depth (superficial vs. deep), location (sole vs. coronary band), and duration (acute vs. chronic).
- Deep injuries that completely penetrate through the hoof wall and contact a bone, tendon, or synovial cavity typically cause severe and acute lameness.
- Increased heat and a prominent digital pulse usually can be palpated.
- If a foreign body such as a nail is present in the bottom of the foot, it is ideal to take a radiograph to determine the exact depth and direction of the nail's path before removing it (Figure 5.31).
- If a wound is not obvious, careful application of hoof testers may help identify focal pain which may indicate the site of penetration.
- Wounds that penetrate the frog can be particularly difficult to locate because the softer and more elastic tissues of the frog tend to collapse and fill in the tract. Probing of the tract can help identify both the depth and direction of the injury. A radiograph can be taken with the probe placed into the tract to further verify its location.
- Perineural anesthesia usually is not needed to localize the site of lameness but is very beneficial to facilitate close examination of the injury site and removal of the frog or sole if needed.
- A penetrating wound of the coronary band can be overlooked if the hair is long or if local swelling and wound drainage are not present.
- Heat, pain, and swelling of one heel bulb are often seen with migration of a subsolar abscess.
- Effusion of the digital tendon sheath or DIP joint may suggest infectious arthritis.

### *Diagnosis*

- Additional diagnostics that can be performed to confirm the location and depth of a penetrating injury include distention of a synovial cavity with saline to detect leakage from the wound, plain radiographs, radiographs with a metallic probe inserted in the wound, contrast radiography (fistulogram), or ultrasound (Figure 5.32).
- Synovial fluid analysis usually can be used to confirm the diagnosis of infectious synovitis.

An increased white blood cell count (greater than 30,000) with neutrophilia and increased protein (greater than 4 gm/dl) are highly suggestive of a septic process.

### *Treatment*

- Treatment of superficial penetrating wounds that do not involve vital structures (bone, tendon or synovial cavities) is aimed at providing adequate drainage, removing infected and necrotic tissue, and protecting the site from further contamination. An antiseptic dressing is applied and the foot is protected to minimize further contamination.
- Penetrating injuries that involve bone, tendon, or synovial cavities require more aggressive treatment depending on the deeper structure that is involved. Typical treatments include both systemic (IV) and local (IV regional perfusion and intrasynovial) antimicrobials; NSAIDs; local debridement of the wound; and lavage, endoscopy, or arthroscopy if a synovial cavity is involved. Involvement of the navicular bursa is best treated with endoscopy or lavage and local debridement of the defect in the frog.
- Wounds that penetrate the distal phalanx should be enlarged and the distal phalanx curetted if possible. Soaking the foot to lavage deep wounds of the foot is generally not recommended.

### *Prognosis*

- Horses with penetrating injuries that do not involve bone, tendon, or a synovial cavity typically do very well. Horses with deep penetrating injuries outside the frog or frog sulci also do well.
- Horses with septic osteitis of the distal phalanx also have a good prognosis following debridement.
- Horses with injuries that involve the navicular bursa and DIP joint represent the most difficult challenge to return to performance but can do well with prompt, aggressive treatment.

## KERATOMA

### *Introduction*

- A keratoma is a non-neoplastic condition of the hoof that is characterized by keratin containing tissue growing between the hoof wall and the distal phalanx.
- The growth usually begins near the coronary band, but it may extend to the solar surface anywhere along the white line.
- A visible deviation of the coronary band and/or hoof wall is often present, and the most commonly affected areas of the foot are the toe and quarter.
- Keratomas have been observed in horses ranging from two to 20 years of age and should be differentiated from other growths that can occur in the hoof such as squamous cell carcinoma, canker, and melanoma.

### *Etiology*

- Trauma and chronic irritation in the form of sole abscesses or direct hoof injuries are the cause in the majority of cases.
- Keratomas can develop without a history of previous injury and the initiating cause often cannot be determined.
- Lameness and the radiographic changes are thought to arise from the growth of the keratoma and the subsequent pressure that it applies to the sensitive lamina and distal phalanx.

### *Clinical Signs*

- A history of a slow onset of intermittent lameness is common. Moderate to severe lameness is commonly observed at presentation.
- The lameness is often seen before the distortion at the coronary band and hoof wall becomes obvious. The coronary band and hoof wall may or may not be abnormally shaped and close examination of the foot may be required to identify any abnormality.
- A bulge in the hoof wall and a deviation in the white line toward the center of the foot may be seen.
- In some cases a fistulous tract may develop in the sole or hoof wall, mimicking a subsolar abscess.
- Hoof tester examination often elicits a painful response when pressure is applied over the lesion
- A PD nerve block often improves the lameness, but a basi-sesamoid or abaxial sesamoid block may be required to completely eliminate the lameness.

### *Diagnosis*

- A definitive diagnosis of keratoma is usually made based on the characteristic radiographic

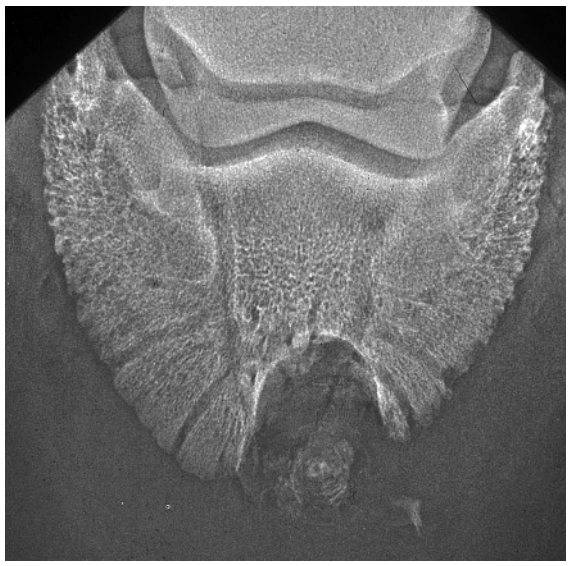


**Figure 5.33.** Dorsopalmar radiograph of P3 demonstrating a smooth margined lytic defect within the bone that is characteristic of a keratoma.

features. A discrete semicircular defect in the distal phalanx is often seen (Figure 5.33). The radiographic signs of a keratoma can usually be differentiated from lysis due to infection because of the smooth borders and lack of a sclerotic margin.
- Ultrasonographic imaging of a keratoma has been reported as a hypoechoic, well-delineated soft tissue mass under the hoof wall.

### *Treatment*

- Treatment involves complete surgical removal of the abnormal growth (Figure 5.34). Incomplete removal of the keratoma is thought to result in recurrence of the growth.
- If hoof stabilization is necessary, a bar shoe with large clips on either side of the defect will prevent independent movement of the hoof wall.
- The goal of surgery is to remove as little hoof wall as possible to prevent complications and to shorten the healing time.

### *Prognosis*

- The prognosis is generally very good for return to performance if the abnormal tissue is completely removed. In one study, 25/26 horses returned to their previous exercise level after surgery.
- In one study, 42% of horses treated without surgery (12 horses) returned to performance compared to 83% that were treated with surgery (23 horses).
- Adequate stabilization of the hoof defect and complete removal of the lesion are important for a successful outcome.

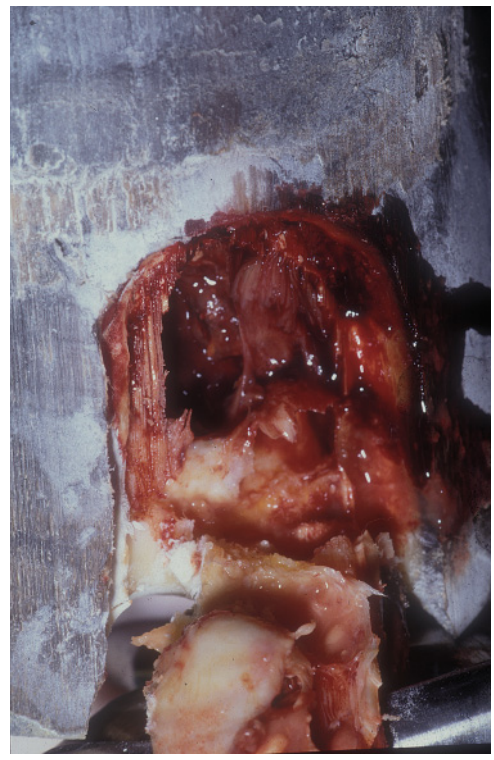


**Figure 5.34.** Hoof wall removal directly over the keratoma demonstrated in Figure 5.33. The keratoma can be seen beneath the hoof wall

## FOOT IMBALANCES

### *Introduction*

- Foot imbalances can be broadly classified as dorsopalmar (DP) or medial-lateral (ML) imbalances.
- Sheared heels and quarters is a type of ML hoof imbalance that occurs between the heel bulbs and hoof capsule with a disproportionate use of one heel and/or quarter (Figure 5.35).

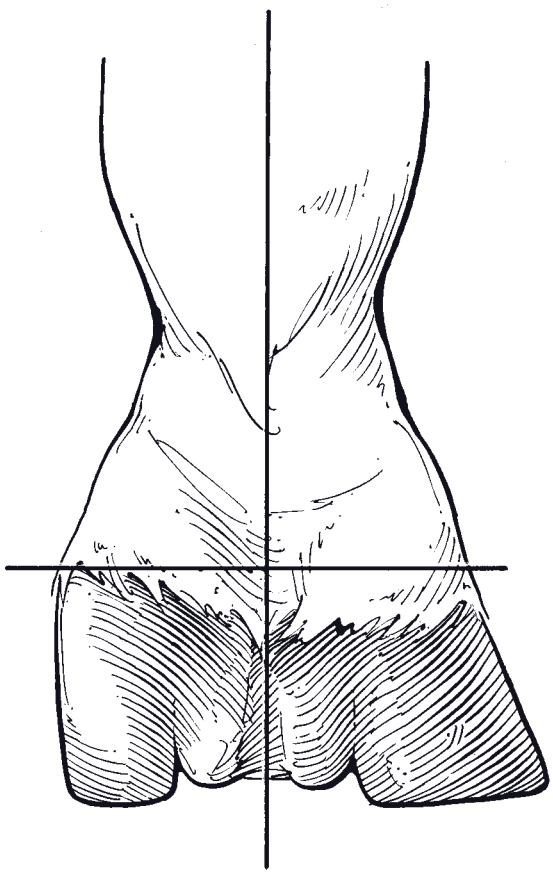


**Figure 5.35.** Sheared heels. Notice the left heel bulb is higher than the right. The hoof is straighter on the affected side (left side) while the hoof wall associated with the lower heel (right side) is flared.

- Hoof imbalances can be a primary cause of lameness or secondary to other conditions in the foot such as navicular disease/syndrome, laminitis, flexural deformity of the DIP joint, hoof cracks, etc. The degree of lameness is usually proportional to the duration and degree of the foot imbalance.
- Contracted heels usually occur secondarily to another primary lameness condition in the foot.

### *Etiology*

- Improper trimming and shoeing is often a common cause. Leaving the toe long for extended periods can contribute to DP imbalances (long-toe, low-heel conformation).

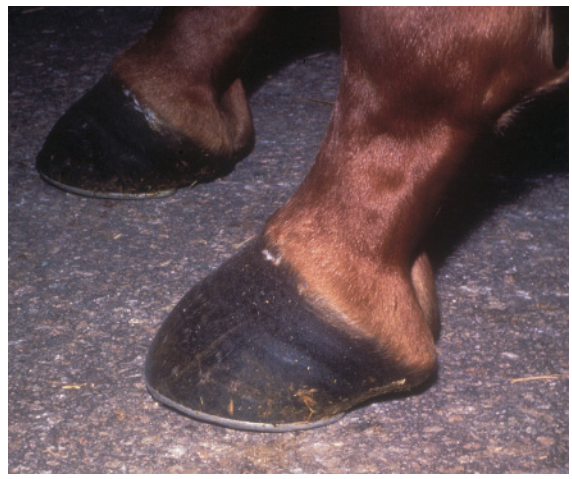


**Figure 5.36.** Broken back hoof pastern axis that is often seen in horses with navicular disease/syndrome.

- Asymmetrical trimming (one heel longer than the other) can contribute to ML imbalances and sheared heels. A disproportionate amount of force is applied to the longer heel during weight-bearing, creating shearing forces between the heel bulbs and quarters.
- Conformational defects in the upper limb (carpal of fetlock varus and valgus) often contribute to abnormal hoof wear that is manifested as ML imbalances.
- Chronic lameness conditions can lead to less heel expansion from reduced weight-bearing and an upright foot conformation.
- Hereditary factors most likely play a role, since some horses often have a long toe, low heel conformation regardless of the circumstances

### *Clinical Signs*

- Most DP imbalances have a long-toe, low-heel foot conformation and a broken back hoof-pastern axis (Figures 3.1, 5.1, 5.36).
- ML imbalances usually have one heel longer than the other; the longer hoof wall may be flared outward and the shorter hoof wall is excessively straight (Figures 3.1, 5.35). The foot usually lands on the longer side first and then rolls to the opposite side of the hoof.
- With sheared heels, the heel bulb and/or quarter on the affected side are visually higher, the hoof wall is straighter, and there is an abnormal flare to the hoof wall on the opposite unaffected side (Figure 5.35). The hoof

wall on the affected side may be rolled under in very severe chronic cases.
- The lameness can be variable and depends on the severity of the hoof imbalance and whether it is primary or secondary.
- Pain across the heels and frog is often found with hoof testers regardless of the type of imbalance; it must be determined whether it is primary or secondary. A PD nerve block usually eliminates the lameness.

### *Diagnosis*

- Primary hoof imbalances must be differentiated from the multitude of other lameness conditions that can affect the foot. Most hoof imbalances are secondary to some other problem in the foot.
- The diagnosis of a hoof imbalance is often made based on the physical examination findings and the lack of another definitive cause of the lameness.
- Standing lateral and horizontal dorsopalmar/plantar radiographs of the feet can be useful to document the severity of the DP and ML imbalances (Figure 5.37).
- Complete radiographic examination of the foot and other imaging such as MRI may be needed to rule out other potential causes of the lameness.

### *Treatment*

- Treatment is directed at correcting the hoof imbalances with corrective trimming and/or shoeing, and treating the primary lameness condition if present.
- Mild cases usually respond to trimming alone. The foot should be trimmed to correct both ML and DP imbalances if possible. Often this only requires shortening the toe and trimming the heels level with the widest part of the frog.
- In severe cases of sheared heels, the affected side is trimmed from the heel through the quarter to create a space when the shoe is applied (Figure 5.38). The weight of the horse and time will permit the heel to drop down into correct alignment. A full-bar shoe is usually recommended because of the instability between the heels.
- Horses with severe DP imbalances (negative angle of P3) often require shoeing with some form of heel elevation. The reader is referred to Chapter 12 of *Adams and Stashak's Lameness in Horses, Sixth Edition*, for further information.

### *Prognosis*

- The prognosis is considered very good for mildly affected horses and those without a primary lameness condition.

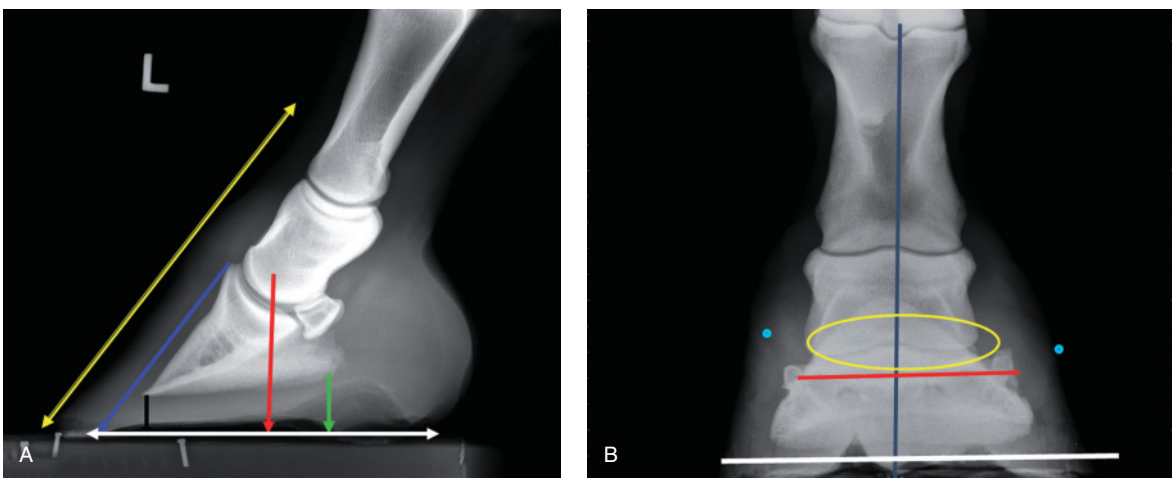



**Figure 5.37.** Lateral radiograph (A) used to illustrate hoof-pastern axis (yellow), center of articulation (red), sole depth (black), palmar angle of distal phalanx (green), break-over (blue), and placement of the shoe (white). DP radiograph (B) used to illustrate the axial alignment of the digit (dark blue), position of the foramina in the distal phalanx relative to the ground (red), alignment of the joint space (yellow), and position of coronary band at heel bulbs (light blue). Courtesy of Steve O'Grady.

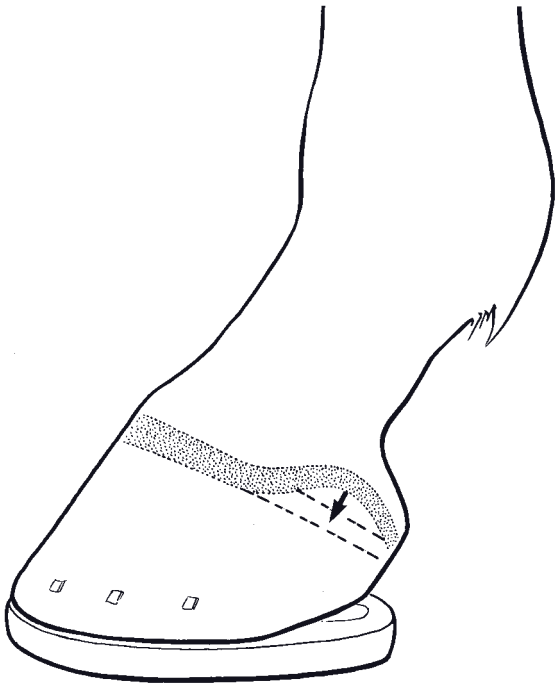


**Figure 5.38.** Corrective trimming and shoeing of a horse affected with sheared heels. The affected side is trimmed from the heel through the quarter to create a space between the hoof wall and the full-bar shoe. The stippled area indicates the level of the coronary band. The arrow is pointing to where the coronary band should be.

- Horses with severe hoof imbalances and secondary hoof wall deformities usually require several shoe resets to correct the imbalance.
- The prognosis of horses with secondary hoof imbalances depends on the success of treating the primary lameness problem.
- Horses with a negative palmar/plantar angle of P3 that fail to grow heel are particularly problematic and often require continued corrective shoeing.

## CLUB FOOT

### *Introduction*

- A club foot is defined as an upright conformation of the foot associated with a flexural deformity of the DIP joint (palmar movement of P3 in relation to P2).
- A club foot can also develop without affecting the DIP joint and is often seen as a secondary hoof imbalance associated with chronic lameness.
- The broken forward hoof-pastern axis or flexural deformity is created by some degree of shortening of the musculotendinous unit (DDFT and associated muscle bellies), causing the DIP joint to be drawn into a flexed position.

### *Etiology*

- Flexural deformity of the DIP joint in young horses is considered a developmental abnormality of unknown cause. Failure of tendons and ligaments to develop at the same rate as bone and a discrepancy between bone growth and the capacity for lengthening of the check ligament are proposed theories as to why it occurs.
- Pain secondary to a primary lameness may cause chronic unweighting of the foot, leading to foot contracture, high heels, and the development of a club foot.
- Lack of exercise has been suggested to prevent proper stretching of the tendons and ligaments, contributing to limb contracture.
- Severe trauma to the DDFT or its associated muscles is also known to cause tendon contracture due to fibrous tissue deposition.

### *Clinical Signs*

- A club foot (hoof angle greater than 60°) is nearly always seen in the forelimbs and lameness may or may not be present. In severe cases the dorsal hoof wall will be vertical to the ground or directed in a palmar direction with the heels completely unweighted.
- Most young horses with developmental club feet should not be lame. The presence of lameness often suggests that the club foot may be secondary to some other musculoskeletal problem in the limb (Figure 5.39).
- Hoof abnormalities associated with club foot conformation are recessed frogs, thin flat soles, poor hoof wall consistency, toe cracks and abscesses, hoof wall separations, and white line disease.
- Poor performance and injuries associated with a high hoof angle are thought to include DIP joint inflammation due to abnormal loading of the joint, sole bruising, especially at the toe from toe first landing, and increased strain on the suspensory ligaments of the navicular bone.

### *Diagnosis*

- A tentative diagnosis is based on the visual examination of an upright hoof conformation.
- Pain on hoof testers applied across the frog and heels may or may not be present.

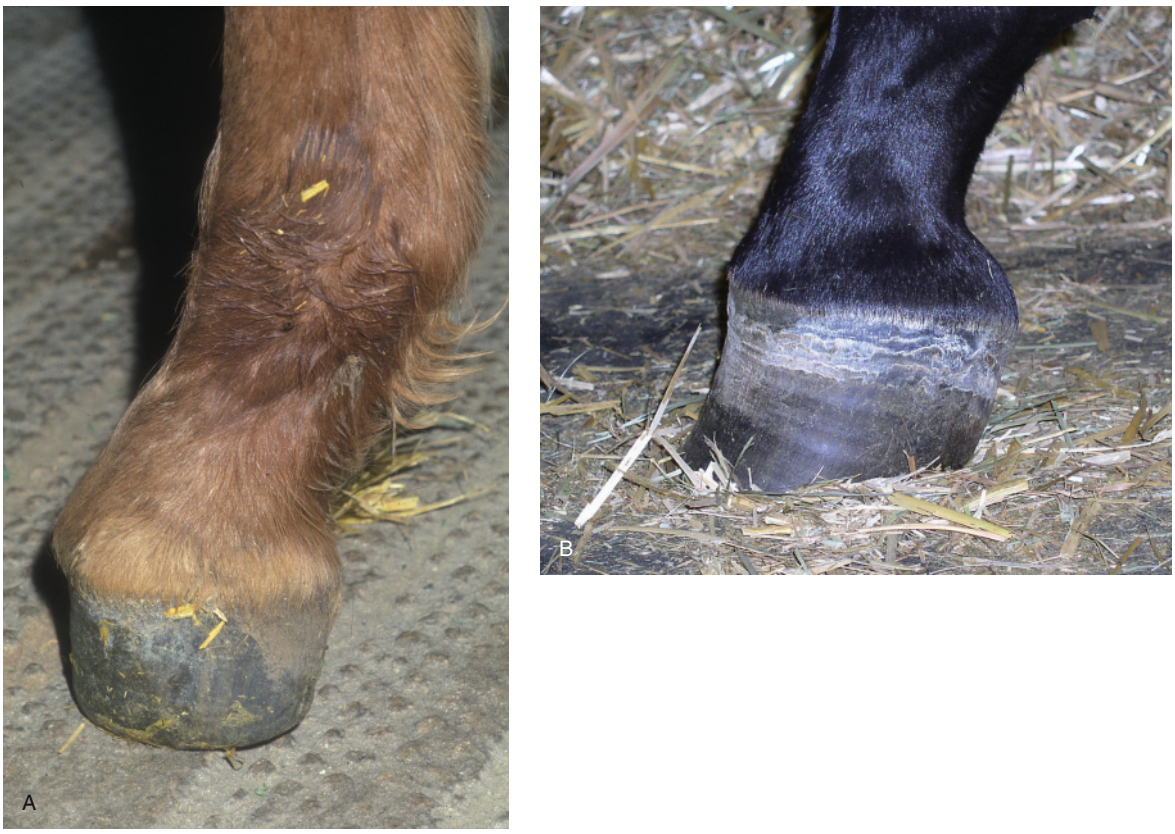



**Figure 5.39.** Weanling quarter horses with typical club foot appearances. The dorsal hoof wall was vertical to the ground (A) with a concavity of the hoof wall (B). The deformities were corrected in both with an inferior check ligament desmotomy.

- A lateral radiograph of the foot is necessary to document whether a flexural deformity of the DIP joint is present.
- Diagnostic nerve blocks and other imaging may be required to document other musculoskeletal abnormalities if a concurrent lameness is present.

### *Treatment*

- High hoof angles with no or mild phalangeal malalignment can generally be improved by gradually lowering the heels in a tapered fashion from the apex of the frog to the heels (Figure 5.40). This increases the ground surface of the foot and attempts to re-establish weight-bearing on the entire solar surface of the foot. The goal is to load the heels, compensate for the shortening of the DDFT, and improve the hoof-pastern axis when possible.

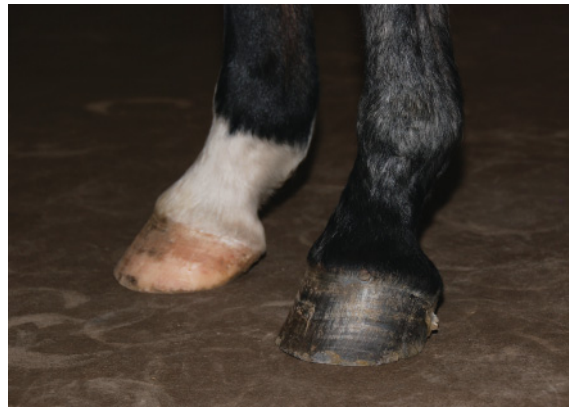


**Figure 5.40.** This young Warmblood had a mild club foot on the left forelimb that did not have phalangeal malalignment. Note the mismatched front feet.

- Farriery for a high hoof angle with a moderate flexural deformity of the DIP joint is less successful. The heels are lowered but heel elevation is added with a pad or wedged shoe to compensate for the shortening of the tendon unit. Several shoeings are often required to correct the problem.
- Horses with the dorsal hoof wall oriented beyond vertical with severe phalangeal malalignment or those that cannot touch the heels to the ground nearly always require surgery (inferior check ligament desmotomy or deep digital flexor tenotomy; Figure 5.39).
- Young horses usually respond better to surgery than older horses, regardless of the severity.

### *Prognosis*

- Horses with mild club feet usually respond well to corrective trimming and shoeing.
- The prognosis for horses with a secondary club foot depends on resolving the primary musculoskeletal problem, but generally these are more problematic.
- Foals with a club foot treated at a younger age (less than six to eight months) with an inferior check ligament desmotomy have an improved prognosis for normal hoof conformation and athletic performance.
- Foals treated surgically also have an improved prognosis for racing over those treated conservatively.

## TOE CRACKS, QUARTER CRACKS, HEEL CRACKS (SAND CRACKS)

### *Introduction*

- Hoof wall cracks represent a focal wall failure, and as such, they can occur anywhere on the hoof wall.
- Hoof wall cracks are generally described by their location (toe, quarter, heel, or bar), length (partial or full), depth (superficial or deep), and the presence or absence of hemorrhage or infection (Figures 5.41, 5.42)
- Quarter cracks and heel cracks are usually the most severe because they often involve the sensitive laminae.

### *Etiology*

- Excessive hoof growth from lack of trimming may cause a lever-arm effect on the hoof wall, contributing to cracks.
- Previous injuries to the coronary band often cause a weak and deformed hoof wall that grows distally.

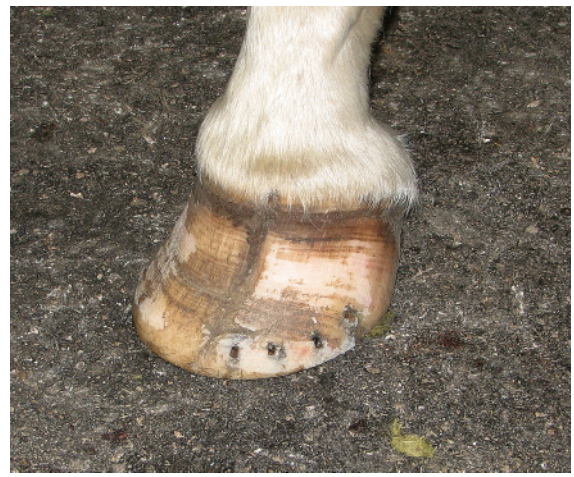


**Figure 5.41.** A partial-thickness toe crack that developed in a horse with a concavity of the dorsal hoof wall.

**Figure 5.42.** A full-thickness quarter crack just after debridement down to the sensitive laminae.

- Drying of the hoof and thin walls contribute to weakening of the wall and cracking.
- Chronic laminitis, white line disease, and club foot conformation may contribute to central toe cracks.
- Heel and quarter cracks are frequently associated with underrun heels and long toes. Placing the shoe too far forward also contributes to quarter cracks.
- Secondary infection can develop in any full-thickness crack from environmental bacteria.

### *Clinical Signs*

- The presence of the defect in the hoof wall is usually obvious. However, not all hoof cracks

are clinically significant and contribute to lameness.
- Lameness may not be present with superficial cracks, but is usually obvious with deep cracks because they "pinch" the sensitive tissues beneath the hoof wall.
- If secondary infection is present, lameness may be severe and purulent exudate is often seen within the hoof crack.
- The hoof wall around the crack is usually very sensitive to hoof testers with infected hoof cracks and in those that are contributing to lameness.
- Perineural anesthesia can be used to determine if the hoof crack is contributing to the lameness.

### *Diagnosis*

- The diagnosis is based on the presence of the crack, which is easily identified, and is classified according to its location and depth. Blood or purulent exudate is only present with full-thickness hoof cracks.
- Most clinically significant hoof cracks are painful to hoof testers.
- Radiographs of the feet should be taken with central toe cracks to determine the presence of an underlying cause.

### *Treatment*

- The type of treatment often depends on the location and depth of the crack. Initial treatment of superficial cracks is often to apply the necessary farriery to change the forces on the section of the foot with the defect. Any abnormal hoof conformation should be evaluated and corrected.
- Any horse with a full-thickness quarter crack should be placed in a bar shoe if possible. These shoes effectively increase the ground surface of the foot, provide palmar/plantar support, and decrease the independent vertical movement at the bulbs of the heels. The foot is trimmed and the affected wall below the crack is unloaded. No nails should be placed palmar/plantar to the defect in the foot (Figure 5.43).
- If infection is present, the crack should be opened, debrided, and bandaged with a suitable disinfectant. The infection must be resolved before any type of composite repair is considered (at least 48 hours). Unstable and infected quarter cracks may need to be removed completely to prevent lameness and continued infection.
- If the horse must continue in work, a repair should be performed to stabilize the crack along with the appropriate farriery.
- Methods of repair include placing stainless steel wire, umbilical tape, or metal or brass bands (for toe cracks) across the crack, often combined with acrylic (Figures 5.44, 5.45). The area of the crack should be "floated" to prevent weight-bearing.

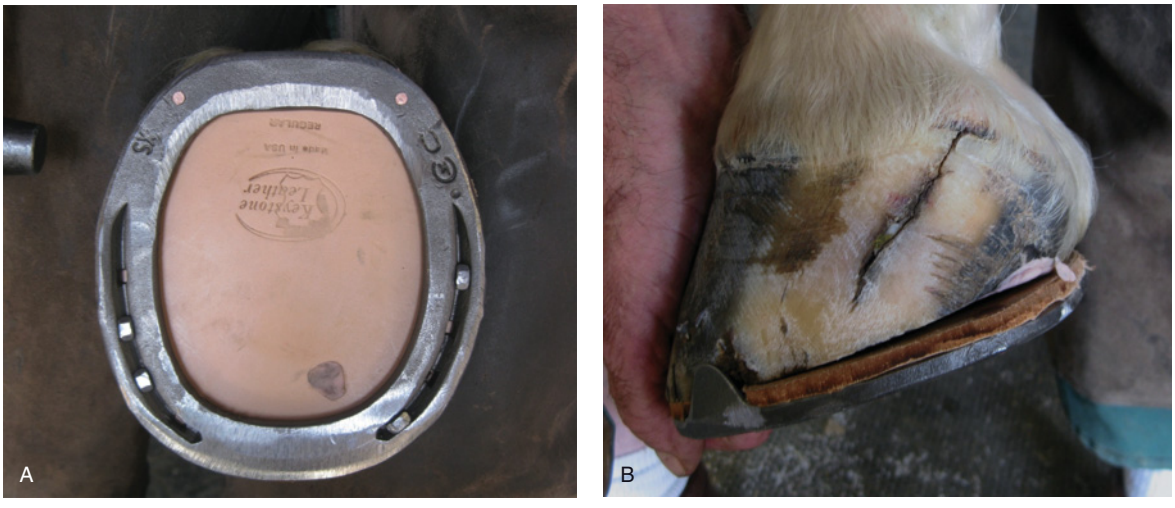



**Figure 5.43.** Straight-bar shoe (A) with the medial heel unloaded (B). Note the quarter crack which is often present with a sheared heel (B). Courtesy of Steve O'Grady.

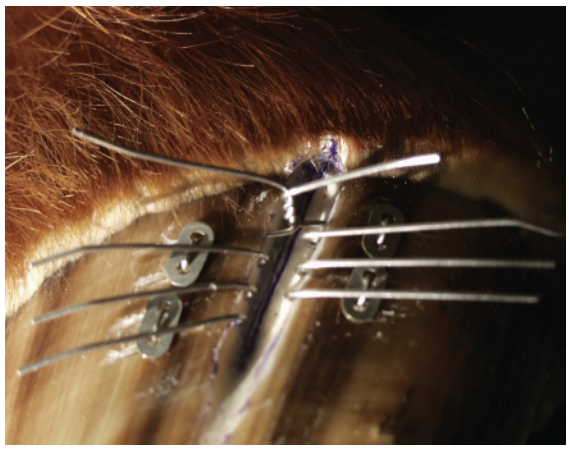


**Figure 5.44.** Placement of wires for a quarter crack repair. Courtesy of Steve O'Grady.

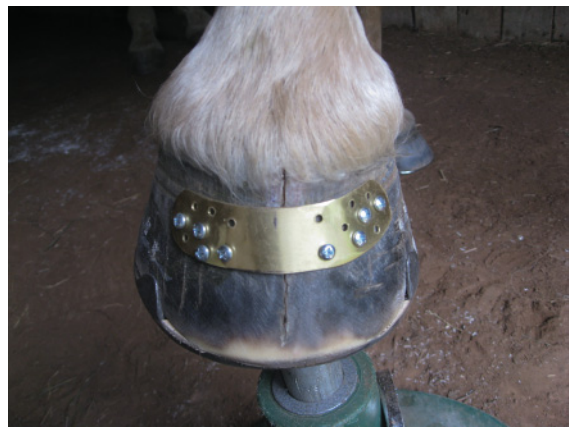


**Figure 5.45.** Brass band attached to dorsal hoof wall to stabilize a dorsal hoof wall defect. Note that the contour of the band matches the coronet. Courtesy of Steve O'Grady.

### *Prognosis*

- Superficial cracks often do not contribute to lameness and can be successfully managed with farriery alone.
- Full-thickness quarter cracks and horizontal cracks can be problematic, especially if infection is present. Often these can cause continued lameness until the crack grows out completely.
- Factors contributing to the hoof cracks must be addressed or they will likely continue.

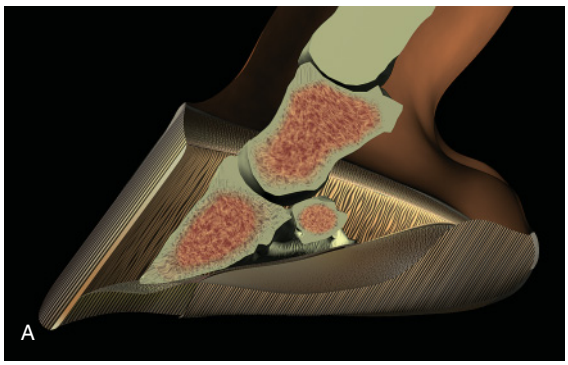
## LAMINITIS

### *Introduction*

- Equine laminitis is a failure of the interdigitation between the dermal and epidermal laminae of the digit that results in displacement of the distal phalanx within the hoof capsule.
- Displacement of the distal phalanx can take place as a symmetrical distal displacement or "sinking" of the phalanx, an asymmetric distal displacement of the phalanx (either medial or lateral), or as a rotation of the distal phalanx away from the dorsal hoof wall (Figures 5.46 to 5.48). Both distal displacement and rotation can occur in the same horse.
- Laminitis is usually a sequelae to four different clinical entities:
  1. Diseases associated with sepsis/endotoxemia
  2. Excessive weight placed on a limb due to injury to the opposite limb
  3. Cushing's disease in the older horse
  4. Equine metabolic syndrome (EMS) including pasture-associated laminitis
- Laminitis is often classified into three different stages: developmental (before clinical signs), acute (presence of clinical signs but no movement of the distal phalanx), and chronic (movement of the distal phalanx has occurred).

### *Etiology*

- In diseases associated with sepsis/endotoxemia, bacterial toxins and even proteins from injured tissue work synergistically with endotoxin to induce systemic inflammation and organ/tissue injury. It is likely that endotoxin plays a role in laminitis, but other circulating mediators and/or toxins are necessary to result in laminar failure.
- Venoconstriction is thought to occur in the digital microvasculature, which increases capillary pressure leading to laminar edema, increased arteriovenous shunting, and possibly capillary collapse due to the inability of edematous laminar tissue to expand against the constraints of the inelastic hoof wall.
- Coagulopathies within the digital vasculature are suspected because platelets play an important role in vascular pathology, exacerbating vascular disturbances and inflammatory injury via the release of inflammatory and vasoactive mediators.
- A severe inflammatory response is thought to occur within the laminae, contributing to



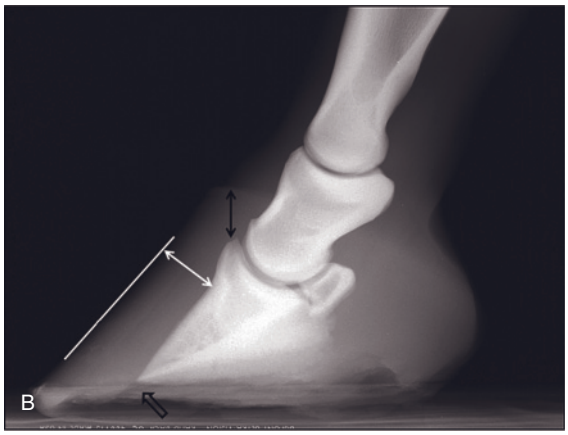
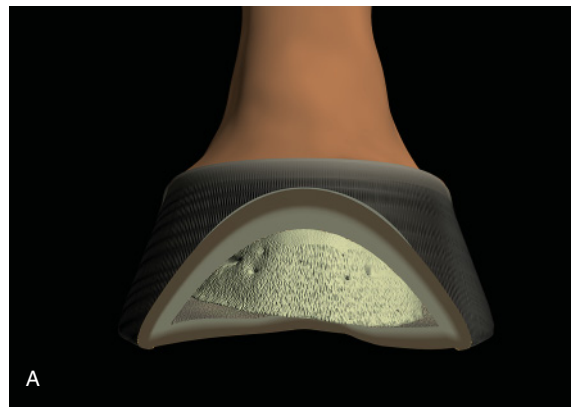




**Figure 5.46.** In symmetrical distal displacement, the distal phalanx descends within the hoof capsule (A). Therefore, the distal phalanx retains its alignment with the more proximal phalanges and the hoof capsule (B), but the distance between the parietal surface of the distal phalanx and the hoof wall increases (white arrow) and the distance between the proximal extensor process and the proximal border of hoof wall (black double arrow) and the distance between the distal phalanx and the sole and ground decrease (open arrow). Courtesy of James Belknap and Andy Parks.



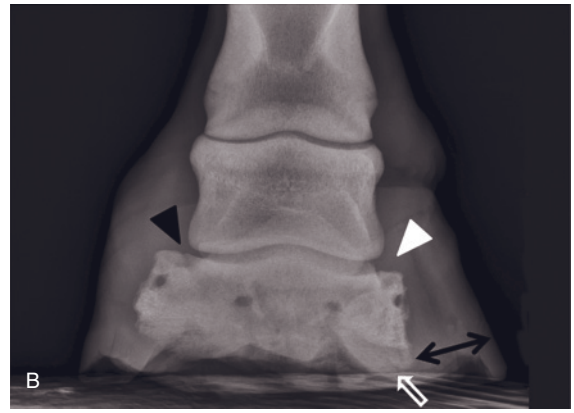



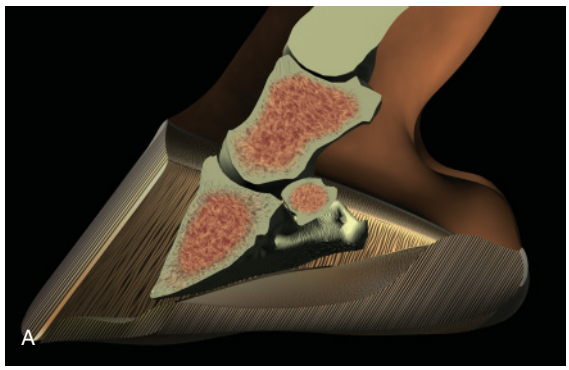

**Figure 5.47.** In medial or lateral asymmetrical displacement (A), one side of the distal phalanx descends (white open arrow in [B]). The distance between the wall and the distal phalanx increases (black double arrow), and the distance between the distal phalanx and the sole and ground decreases on the affected side (white open arrow). Additionally, the DIP joint becomes asymmetrical when viewed on a dorsopalmar radiograph; the joint space is increased on the affected side (white arrowhead) and decreased on the unaffected side (black arrowhead). Courtesy of James Belknap and Andy Parks.

basal epithelial cell injury, dysregulation of hemidesmosomes, and dysadherence of the epithelial cells from the underlying basement membrane.

- The loss of adhesion of the epidermal laminae to the underlying dermal laminae permits the normal forces acting on the hoof wall to essentially "tear" the remaining laminae, causing movement of the distal phalanx.
- The severity of the laminar insult most likely correlates with how fast and how much the distal phalanx moves within the hoof wall.
- Predisposing factors include diseases causing sepsis/toxemia (grain overload, retained placenta, colitis, etc.), equine metabolic syndrome (insulin resistance), Cushing's disease, excessive weight-bearing (support limb laminitis), exercise on hard surfaces, and the use of corticosteroids.
- The reader is referred to Chapter 5 in *Adams and Stashak's Lameness in Horses, Sixth Edition*, for further information.

## *Clinical Signs*

- Laminitis is seen most commonly in both front feet but all four feet, both hind feet, or only a



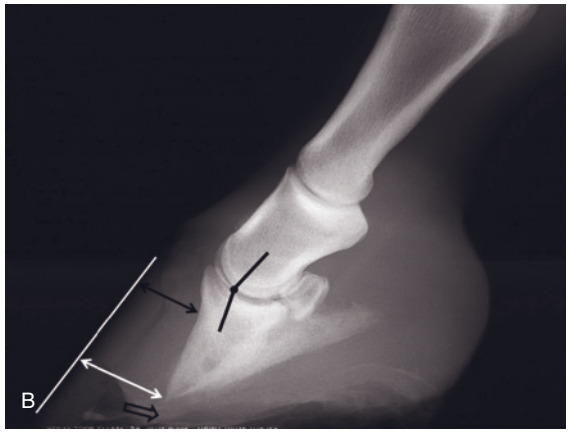




**Figure 5.48.** (A and B) In horses with early chronic laminitis, the surface of the hoof wall is unchanged, but the distal phalanx rotates about the DIP joint (the joint flexes, black lines in B); consequently, the normal alignment of the distal phalanx with the other phalanges is changed (phalangeal rotation). Additionally, the distance between the dorsal distal parietal surface of the distal phalanx and hoof capsule increases (white double arrow) while it remains close to normal proximally (black double arrow); (i.e., there is divergence of the surfaces [capsular rotation]). Also, the distance between the dorsal margin of the distal phalanx and the sole and ground is decreased (open arrow). Courtesy of James Belknap and Andy Parks.

single foot (with support limb laminitis) may be involved

- The onset of clinical signs commonly occurs 24 to 72 hours following the onset of a septic disease process.
- The onset of disease in EMS is variable, but commonly occurs in the spring during consumption of lush pasture.
- The lameness can vary from being barely detectable to complete recumbency.

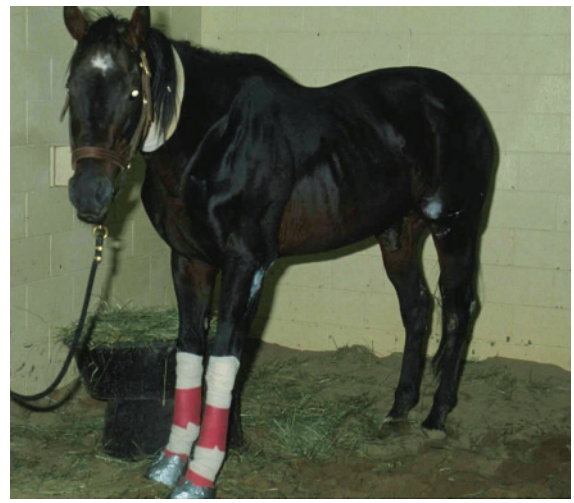


**Figure 5.49.** Classic stance of a horse with laminitis with the forelimbs placed abnormally far forward and weight shifted toward the hindlimbs.

- Signs of acute laminitis include lameness, an increase in the temperature of one or more hooves, increased digital pulses, and elicitation of a painful withdrawal response to hoof-testers over the toe.
- The characteristic stance of a laminitic horse with both fore feet affected is placement of the fore feet well in front of the normal position and anterior placement of the hind feet in order to shift more weight to the hindlimbs (Figure 5.49).
- Horses with chronic laminitis may have different degrees of hoof capsule deformation and lameness. These include a dorsal concavity to the hoof wall, abnormal growth rings which are more widely spaced in the heel than the toe, and a flat or convexity to the sole dorsal to the apex of the frog (Figure 3.5). The white line may be wider than normal and show evidence of prior bruising/hemorrhage.
- In cases of distal displacement of the third phalanx, there may be a palpable (and sometimes visible) groove at the junction of the skin of the pastern and the coronary band.

### *Diagnosis*

- The diagnosis of acute laminitis is often made based on the history, the characteristic stance, and the digital exam (pulse, heat, and hoof testers).
- Horses with acute laminitis may not improve with a PD block but usually respond to an

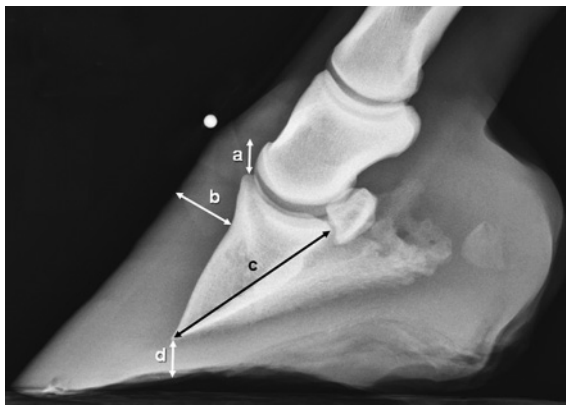



**Figure 5.50.** For assessment of symmetrical distal displacement of the distal phalanx, lateral radiographs of the distal limb can be used to measure: from the proximal extensor process to the proximal aspect of the hoof wall (immediately distal to coronary band [a], the distance from the dorsal parietal surface of the distal phalanx to the dorsal surface of the hoof capsule [b], the ratio of [b] to the length of the palmar cortex of the distal phalanx [b/c]), and the distance from the dorsodistal tip of the distal phalanx to the ground surface of the sole (d). Courtesy of James Belknap.

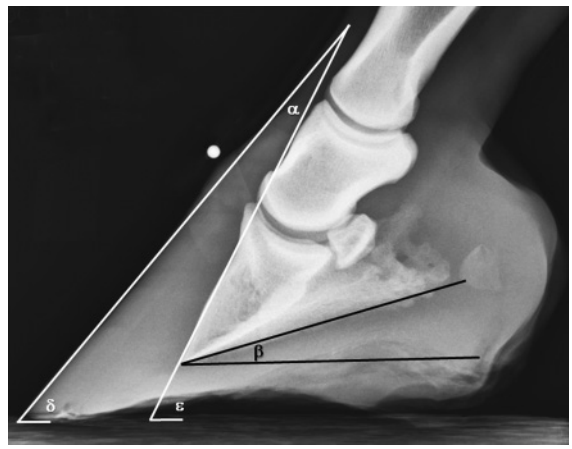



**Figure 5.51.** For assessment of rotation of the distal phalanx, the clinician can assess the degree of capsular rotation (angle α) at the intersection of the dorsal capsular and dorsal phalangeal lines, or can measure the difference between the dorsal angles δ and ε. The relationship of the solar margin of the distal phalanx to the ground surface of the foot can be assessed by measuring angle β. Courtesy of James Belknap.

abaxial sesamoid block. In some severely painful cases, the lameness may not block out entirely with local perineural anesthesia.
- Horses with chronic laminitis often improve with a PD block because a significant part of the lameness is often due to sole pain.
- Radiographs may or may not be useful in the acute stage but are usually diagnostic in horses with chronic laminitis. Lateral and horizontal beam dorsopalmar/plantar views are recommended.
- Types of movement of the distal phalanx that should be assessed using radiography include capsular vs. phalangeal rotation, symmetrical distal displacement, and asymmetrical or uniaxial distal displacement of the distal phalanx (Figures 5.50, 5.51). The reader is referred to Chapter 5 in *Adams and Stashak's Lameness in Horses, Sixth Edition*, for further information).

### *Treatment*

- The initial goal of treatment of acute laminitis is to stabilize the digit in the short term regardless of the degree of displacement.
- Concurrent treatment of the inciting cause in horses with sepsis/toxemia-induced laminitis is important.
- Anti-inflammatory therapy is considered mandatory in acute laminitis and may include the use of NSAIDs, dimethyl sulfoxide (DMSO), pentoxyfylline, and local cryotherapy of the digits.
- Anticoagulant therapies such as low-molecular-weight heparin or aspirin may be used.
- Analgesic therapy using a constant rate infusion of a combination of ketamine, morphine, lidocaine, detomidine, and acepromazine (Pentafusion) can be used if the forelimbs are affected, or an epidural if the hind feet are involved.
- The two main objectives of hoof care are to redistribute the force of weight-bearing away from the wall and decrease the extensor moment about the DIP joint (Figure 5.52).
- Methods to redistribute the force of weight-bearing away from the wall include removing the shoes; placing the horse in sand, shavings, or peat; and applying Lilly pads, silicone putty, or commercial pad systems such as the Soft-Ride boots (Soft-Ride, Inc., Vermillion, OH) to the affected feet.
- Methods to decrease the extensor moment about the DIP joint include elevating the heels and/or moving the break-over more palmar/plantar to usually be shortening the toe (Figure 5.52).
- The goals of treating horses with chronic laminitis include maintaining stability of the distal

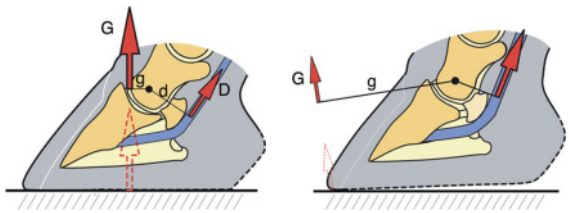



**Figure 5.52.** (A) At rest, the foot is stable with respect to the ground. The ground reaction force is approximately vertical and positioned approximately in the center of the foot, slightly in front of the center of rotation of the distal interphalangeal joint. The product of the magnitude of the GRF (G, large red arrow) and the length of its moment arm (g) is the extensor moment, which is opposed by the flexor moment, which is the product of the force in the deep digital flexor tendon (D, small red arrow) multiplied by the length of its moment arm (d). (B) At break-over, the position of the foot is dynamic and the magnitude of the ground reaction force (G, small red arrow) is decreased as the horse moves off the leg, but the length of the moment arm (g) is increased because the GRF is positioned at the toe. To cause the foot to move from the stable position at rest to the dynamic state, the flexor moment exceeds the extensor moment. Courtesy of Andy Parks.

**Figure 5.53.** This horse has medial asymmetrical distal displacement, evidenced by disparate growth of the medial and lateral walls. A wooden shoe has been positioned to act as a lateral extension to increase weight-bearing by the healthier lateral side of the foot to decrease pain from compression of the sole and tension in the laminae medially. Courtesy of Andy Parks.

phalanx, controlling pain, and restoring the relationship between the hoof capsule and the distal phalanx.

- The mainstay of treatment is usually hoof care (trimming with or without shoeing), and will vary depending on whether there is rotation or displacement of the distal phalanx.
- Several shoe types have been used in horses with rotation including regular keg shoes, egg-bar shoes, reverse shoes, heart-bar shoes, four-point rail shoes, and wooden shoes/clogs (Figure 5.53).
- There is no perceived benefit to elevating the heels in horses with distal displacement and the wooden shoe/clog may be the best shoeing choice to treat distal displacement in the forelimbs.
- Coronary grooving is being used in some cases to encourage dorsal hoof wall growth (Figure 5.54).
- A DDFT tenotomy, usually at the level of the mid-metacarpus, can be used to help restore alignment of the distal phalanx in horses with severe rotation that have not responded to corrective shoeing (Figure 5.55).
- Sole abscesses, pedal osteitis, and solar margin fractures are sequelae of chronic laminitis.

**Figure 5.54.** Grooving of the proximal dorsal hoof wall immediately distal to the coronary band is used to encourage dorsal hoof wall growth and mechanically dissociate new hoof wall growth from the older distal wall. Courtesy of Andy Parks.

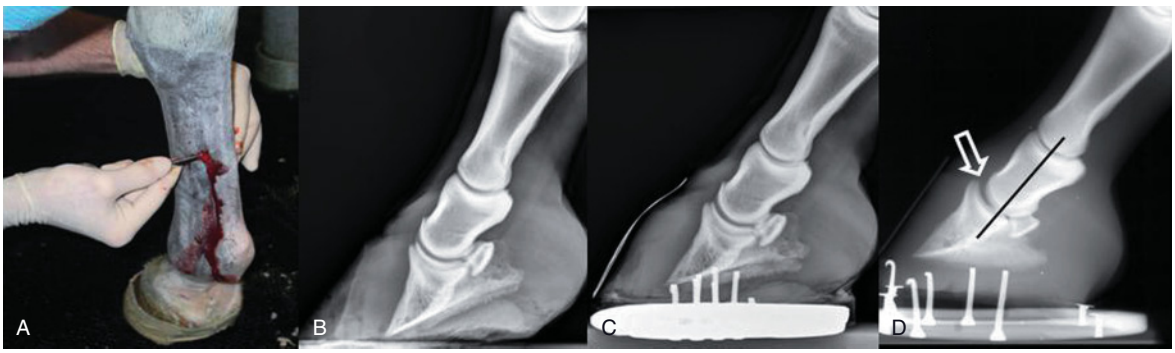



**Figure 5.55.** Tenotomy of the DDFT is most commonly performed while standing at the mid-cannon region using a guarded bistoury (A). The tenotomy allows realignment of the rotated distal phalanx (B) with the ground surface (note realignment of the foot in [C] after six weeks). Subluxation of the DIP can occur following the procedure (D), characterized by dorsal displacement of the extensor process of the distal phalanx away from middle phalanx (arrow), and by caudal displacement of the distal articular surface of the middle phalanx so that a line bisecting the middle and proximal phalanges does not bisect the middle of the articular surface of the distal phalanx (black line). Courtesy of James Belknap.

### *Prognosis*

- The three most important factors that affect the prognosis are the severity of the original pathology, the type of displacement, and the severity of the clinical signs.
- Horses with rotation are considered to have a more favorable prognosis than those with distal displacement. The prognosis following displacement is always considered guarded to poor.
- Capsular rotation greater than 11.5° and a coronet-to-extensor-process distance of greater than 15.2 mm ("founder" distance) have been associated with poor outcomes.
- The thickness of the sole, and the angle that the solar margin of the distal phalanx subtends with the ground, may help assess prognosis; thinner soles and greater angles of the solar margin are anecdotally associated with a reduced prognosis.

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**Revised from "Lameness in the Extremities, The Foot" in *Adams and Stashak's Lameness in Horses, Sixth Edition,* by Gary M. Baxter, James K. Belknap, and Andrew Parks.**

6

CHAPTER

# Common Conditions of the Forelimb

## OA OF THE PROXIMAL INTERPHALANGEAL (PIP) JOINT

### *Introduction*

- The term "high ringbone" is often used synonymously with OA of the PIP joint.
- Older horses are at greater risk and the forelimbs are more frequently affected than the hindlimbs.
- The development of secondary OA from P2 fractures (particularly plantar eminence fractures) or osteochondrosis (OC) occurs more commonly in the hindlimbs than the forelimbs.

### *Etiology*

- Chronic overuse or repetitive trauma of the PIP joint and surrounding structures is the most common cause.
- Inherent conformational traits (upright pasterns) and the type of work (Western performance) the horse performs also may contribute to problems in this joint.
- Single-event, high-energy trauma that does not cause a fracture or joint luxation also can damage the articular cartilage and subchondral bone, predisposing to OA.
- OA may develop secondary to OC, unrecognized palmar/plantar eminence fractures of P2, traumatic blows or lacerations, septic arthritis, and selective weight-bearing in young horses.

### *Clinical Signs*

- Focal or diffuse enlargement of the pastern region may be evident visually or with palpation (Figure 6.1).
- Palpable heat and pain with firm digital pressure may be appreciated, depending on the duration.
- Usually there is pain on flexion and rotation of the pastern region.
- Obvious enlargement of the pastern and/or varus deformity of the phalanges may be present in horses with advanced disease.
- Horses with mild disease may have no visual or palpable abnormalities.
- Lameness is often variable (2 to 4/5) depending on the severity and duration of the disease, and is usually worsened when the affected limb is on the inside of the circle.
- The lameness should not improve with a very low PD nerve block, but may improve if the

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

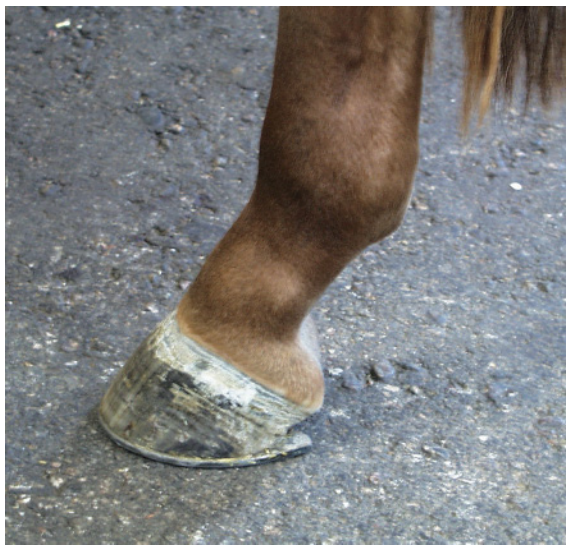
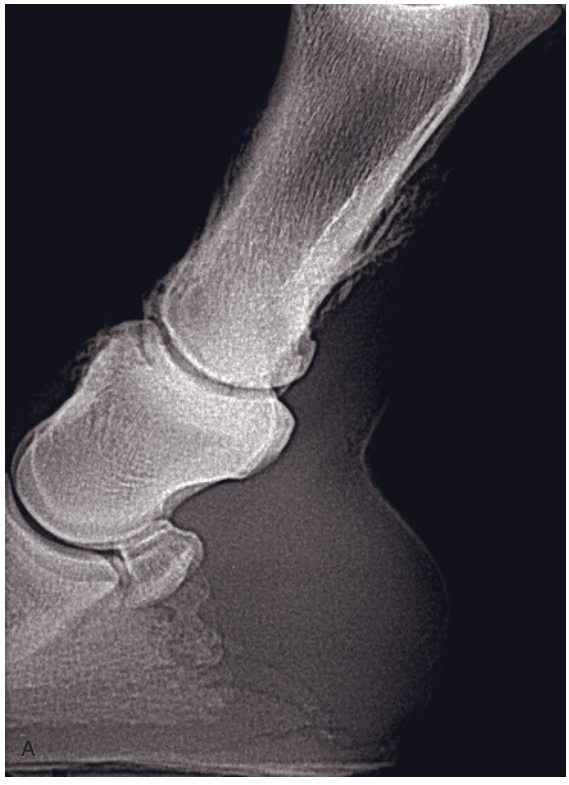


**Figure 6.1.** Typical enlargement of the pastern that may be visible in horses with OA of the PIP joint. This was a young horse with osteochondrosis of the hindlimb PIP joint.

block is performed in the mid-pastern region or if a large volume of anesthetic is used.
- Most horses improve with a basisesamoid or abaxial sesamoid nerve block, or with intra-articular (IA) anesthesia.

### *Diagnosis*

- A tentative diagnosis is often made based on physical examination findings and responses to local anesthesia.
- Radiography is usually used to confirm the diagnosis. Radiographic abnormalities may include (Figure 6.2):
  1. Joint space narrowing or collapse
  2. Osteophyte formation
  3. Subchondral bone sclerosis and/or lysis
  4. Periosteal/periarticular bony proliferation
  5. Subchondral cystic lesions
- Infectious arthritis also can cause severe periosteal/periarticular bony proliferation, osteophyte formation, and subchondral lysis on radiographs.



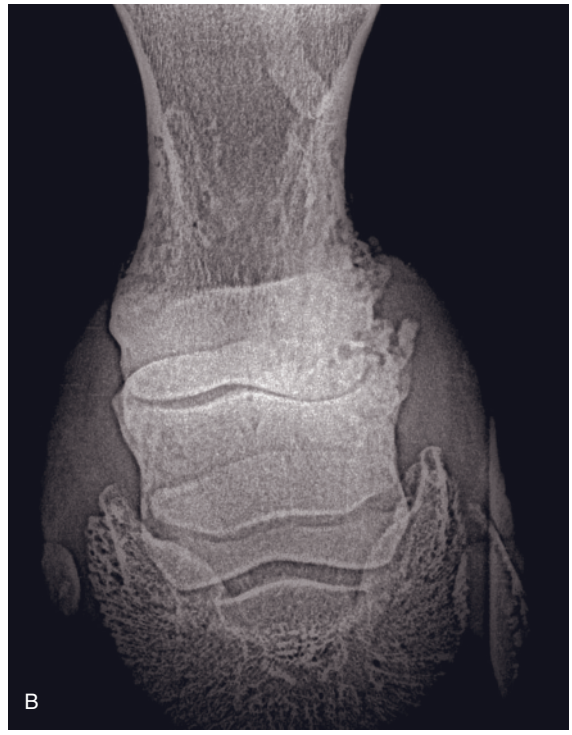




**Figure 6.2.** Lateral(A) and dorsopalmar (B) radiographs of the pastern region with a marked periosteal reaction, joint space collapse, and subchondral lysis consistent with advanced OA within the joint. This horse was lame at the walk and underwent arthrodesis of the joint.

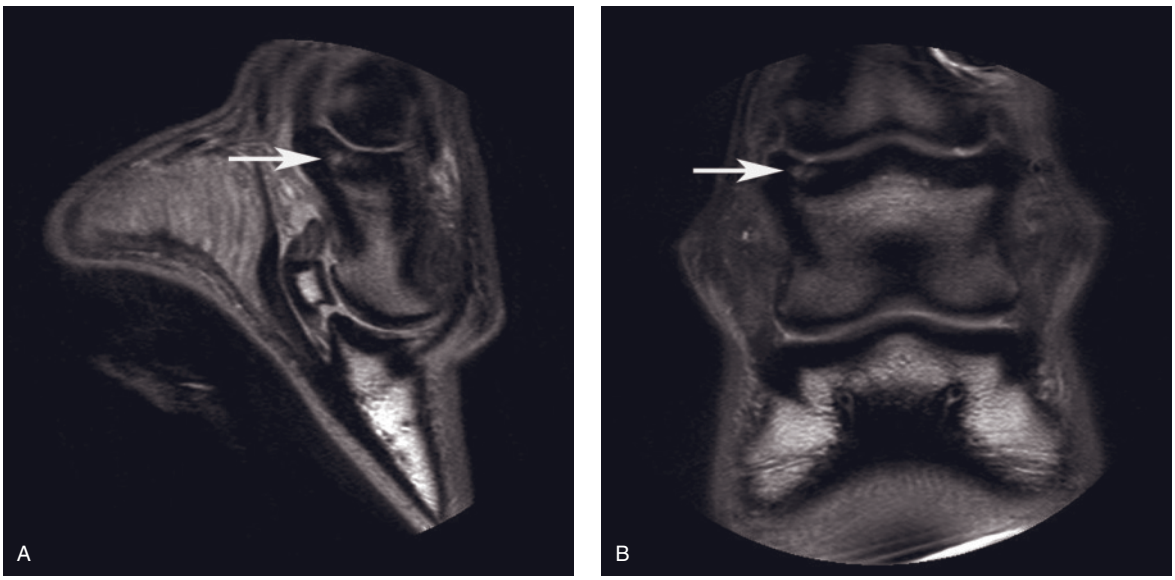



**Figure 6.3.** Lateral (A) and dorsopalmar (B) proton density (pd) MRI images of a horse with lameness isolated to the pastern region that revealed subchondral bone disease/SCL (arrows) of the palmar aspect of P2.

- Radiographs of the contralateral joint should be considered in horses with OC or older horses with advanced OA because these conditions are often bilateral.
- Radiographic abnormalities may be limited in horses with only mild disease or those with acute traumatic injuries. Repeat radiographs in three to four weeks may reveal abnormalities suggestive of OA.
- MRI of the pastern region may reveal subchondral bone and joint abnormalities (bone bruising/edema or bone marrow lesions) that are not apparent on radiographs (Figure 6.3).

### *Treatment*

- Treatment is variable and is often made on a case-by-case basis. For example, horses with severe lameness and advanced radiographic abnormalities usually are not good candidates for nonsurgical treatments because their effectiveness is often short-lived. In contrast, horses with a single traumatic injury to the PIP joint may respond well to rest and develop minimal radiographic abnormalities (Figure 6.3).
- Conservative management of PIP joint OA may involve periods of rest, systemic and/or IA anti-inflammatory therapy, oral or systemic disease modifying drugs, trimming and shoeing, and a change in the horse's career.
- In acute cases, rest from exercise is important to prevent further trauma, reduce inflammation, and permit healing to occur. Confinement and rest is much less effective in horses with chronic OA.
- Treatment may include intermittent use of NSAIDs such as phenylbutazone, flunixin meglumine, or firocoxib. NSAIDs can be given prior to, during the days of performance, and for one to two days after performance to permit some horses to perform relatively pain free for prolonged periods.
- Oral NSAIDs are often combined with IA medication of the PIP joint in horses with chronic OA to reduce the signs of lameness and improve the effectiveness of both treatments.
- Other nonsurgical treatments may include IA autologous serum or polysulfated glycosaminoglycans (PSGAGs), IM PSGAGs, IV hyaluronan (HA), oral nutraceutical supplements, and corrective shoeing if the hoof-pastern axis is abnormal.
- Surgical treatment for OA of the PIP joint consists of arthrodesis; natural ankylosis of the joint may occur, but it is often a long, painful process. The current recommended technique is a single dorsal midline plate with two additional transarticular 5.5-mm screws (Figure 6.4). This technique is thought to improve the comfort level of horses in the immediate post-operative period, and requires casting for only two to three weeks post-operatively.

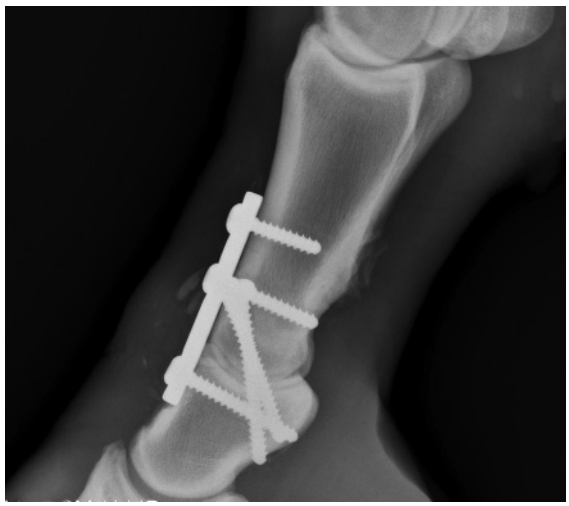


**Figure 6.4.** Lateral radiograph of the pastern following placement of a three-hole plate with two transarticular 5.5-mm screws for arthrodesis of the PIP joint.

### *Prognosis*

- Most horses with mild to moderate OA can be managed with some combination of non-surgical treatments.
- The prognosis for horses following PIP joint arthrodesis is less predictable in the forelimb than in the hindlimb.
- Approximately 85% to 95% of horses with hindlimb and 70% to 85% of horses with forelimb lameness should return to their intended use, and 85% will return to athletic soundness following an arthrodesis.
- Complications that may prevent horses from becoming athletically sound include implant infection, excessive bony proliferation that impinges on the DIP joint, exostosis of the extensor process of the distal phalanx, and soft tissue "irritation" associated with the implants.
- Horses with chronic infectious arthritis treated by PIP joint arthrodesis have a worse prognosis than those treated for non-septic conditions.

## OSTEOCHONDROSIS (OC) OF THE PIP JOINT

### *Introduction*

- OC of the PIP joint is identified less commonly than other joints in the horse. However, both osteochondral fragmentation and subchondral cystic lesions (SCLs) can occur.
- Osteochondral fragments tend to occur dorsally (usually from the distal aspect of P1) or palmarly/plantarly (midline from the eminences of P2 (Figure 6.5).
- SCLs usually involve the distal aspect of P1 but occur rarely in the proximal aspect of P2 (Figure 6.6).
- Malformation of the condyles of the distal aspect of P1 without fragmentation also has been recognized by the author, and may represent another form of OC that leads to early OA within the PIP joint.

### *Etiology*

- OC of the PIP joint is assumed to be due to similar factors that cause the condition in other locations.
- Traumatic fragmentation and subchondral bone damage leading to SCLs can occur within the PIP joint, and it may be difficult to differentiate between developmental and traumatic causes.

### *Clinical Signs*

- Developmental lesions tend to occur in young horses and may cause variable signs of lameness.
- Physical examination findings are similar to other problems within the PIP joint and may include no abnormalities, enlargement of the pastern, pain with flexion and manipulation of the pastern region, and positive response to flexion tests.
- Horses with SCLs of the distal aspect of P1 tend to be more lame than horses with osteochondral fragmentation; these are more common in the hindlimb than the forelimbs.

### *Diagnosis*

- The diagnosis of OC is confirmed with radiographs. Osteochondral fragmentation usually can be seen on both lateral and dorsopalmar/plantar views (Figure 6.5), whereas SCLs are often only visible on the dorsopalmar/plantar radiographic projection (Figure 6.6).
- Some lesions, particularly osteochondral fragmentation, may be incidental findings.
- Most SCLs that involve the distal condyle of P1 are clinically significant and are often accompanied by radiographic changes consistent with OA.
- Radiography of the opposite PIP joint should be performed because OC lesions can be bilateral.

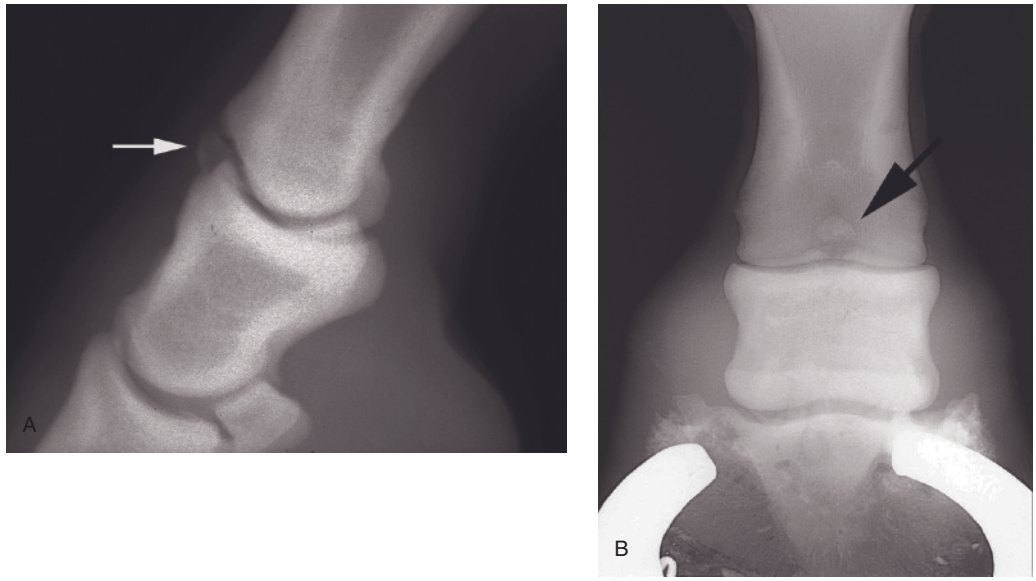



**Figure 6.5.** (A) Lateral radiograph of a horse with a dorsal OC fragment (arrow) and (B) dorsopalmar radiograph of another horse with fragmentation in the palmar aspect of the PIP joint (arrow). Both fragments were removed with arthroscopy.

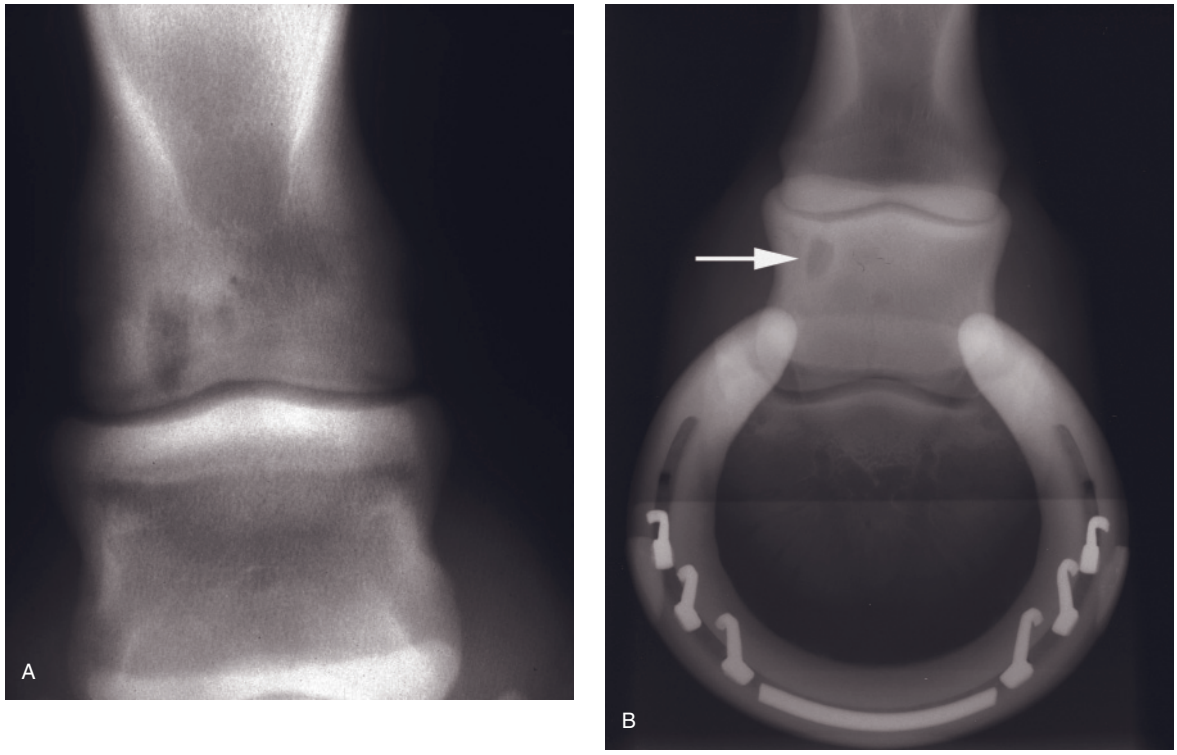



**Figure 6.6.** Dorsoplantar radiographs of two different young horses demonstrating SCLs of the distal medial condyle of P1(A) and the proximal aspect of P2 (arrow; B). The SCL in (B) was not considered to be the cause of the lameness in that horse.

### *Treatment*

- Arthroscopic removal is the treatment of choice for osteochondral fragments within the PIP joint that are causing clinical problems Both the dorsal and palmar/plantar pouches of the PIP joint are accessible with the arthroscope; however, the surgery can be difficult (Figure 6.5).
- SCLs of distal P1 are often clinically significant and can be managed conservatively or surgically depending on the severity of lameness. Conservative management with NSAIDs and IA medication usually resolves the lameness; however, recurrence is common.
- Most horses with SCLs and secondary joint OA are best treated surgically with arthrodesis of the joint.

### *Prognosis*

- Horses with OC fragmentation within the PIP joint have a good prognosis after arthroscopic removal. Some fragments may be incidental findings and horses can perform without removal.
- Horses with SCLs have a worse prognosis than those with fragmentation and often require arthrodesis of the joint. The prognosis is similar to that of any horse with a PIP joint arthrodesis.

## LUXATION/SUBLUXATION OF THE PIP JOINT

### *Introduction*

- Luxation of the PIP joint is uncommon and can occur in a medial/lateral or palmar/plantar direction.
- Medial/lateral luxation involves the collateral ligaments of the PIP joint and may be open or closed.
- Palmar/plantar luxation is associated with complete tearing of the straight DSL, branches of the SDFT, or a combination of these injuries (Figure 6.7).
- Subluxations of the PIP joint usually occur in a dorsal direction and are most common in young horses secondary to flexural deformities and other developmental orthopedic diseases (Figure 6.8).

### *Etiology*

- Lateral/medial luxations are usually caused by severe trauma to the joint capsule and collateral ligament (e.g., distal limb caught in something and the horse struggles and/or falls) or lacerations that transect the collateral ligament.

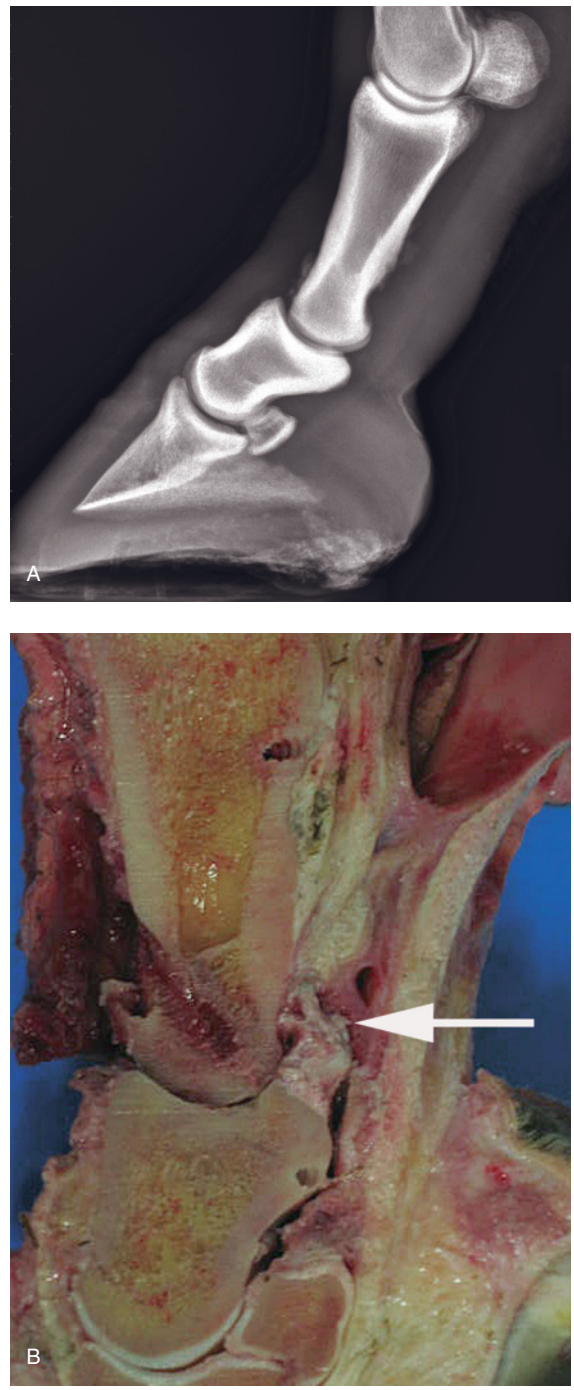



**Figure 6.7.** Lateral radiographic view (A) and postmortem specimen (B) from a horse that had complete plantar luxation of the PIP joint. Excessive dropping of the fetlock can be seen in (A) and rupture of the straight DSL can be seen in B (arrow).

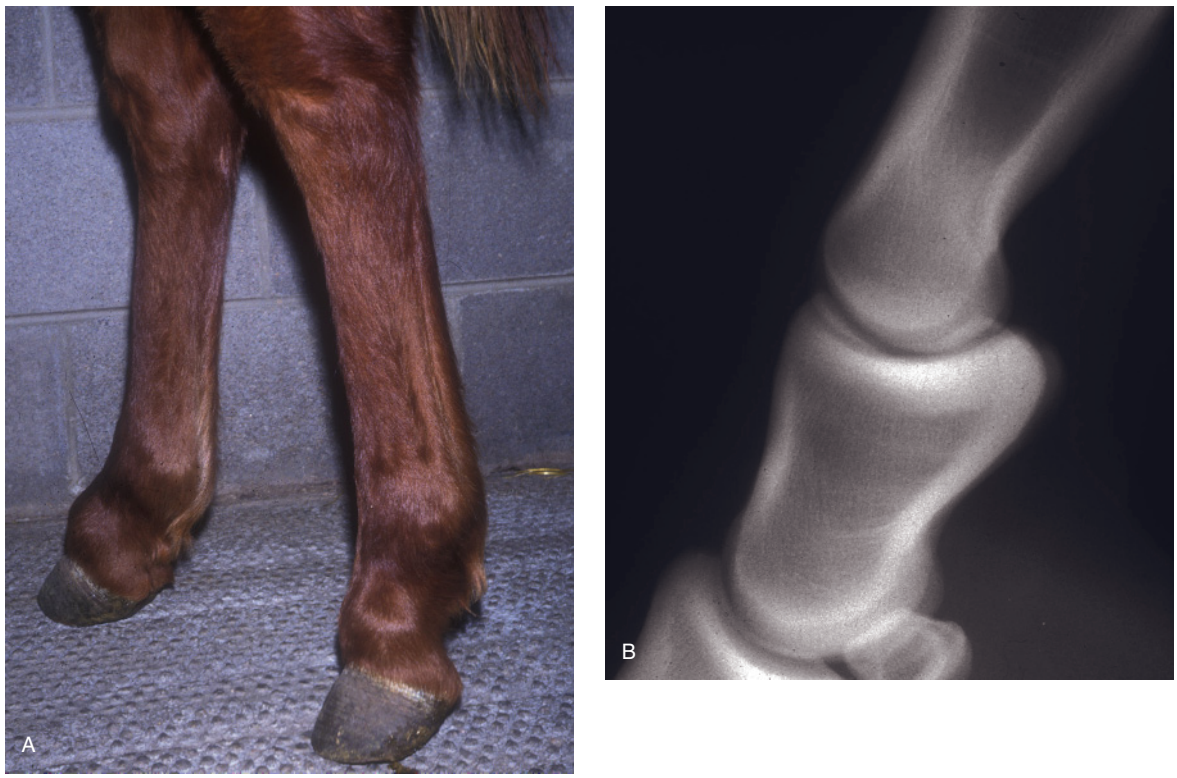



**Figure 6.8.** Visual (A) and radiographic (B) lateral views of a young horse with bilateral dorsal subluxation of the PIP joints. Dorsal swellings over both rear pastern joints can be seen in (A) and dorsal subluxation of P1 in relation to P2 within the pastern joint is present in (B).

- Palmar/plantar luxation generally occurs from acute trauma resulting in overextension of the PIP joint and tearing of the joint capsule, straight DSL, and the insertion of the SDFT.
- Palmar/plantar subluxation also can be seen in foals and weanlings that have jumped from heights and in foals with flexor tendon laxity that overexert themselves during free exercise.
- Dorsal subluxations are thought to be secondary to flexural deformities or limb contracture, and may be seen in foals/weanlings that are rapidly growing with an upright conformation (Figure 6.8).

### *Clinical Signs*

- Horses with medial/lateral luxation are often non-weight-bearing or lame at the walk, have swelling of the pastern associated with the ligament injury, and a limb deformity may be present. Instability and pain may be identified with rotation or medial/lateral movement of the phalanges.
- Horses with palmar/plantar luxation are also often very lame in the acute stage. The lameness may subside over time but they usually remain lame at the walk. The dorsal surface of the pastern will appear concave (dished out) rather than straight or convex, as would occur with dorsal subluxation. In chronic cases, excessive hyperextension of the pastern and sinking of the fetlock is noted when the horse walks (Figure 6.7).
- Dorsal subluxation occurs primarily in the hindlimbs in young horses and lameness is usually absent or mild. This type of subluxation is often dynamic in nature; it is seen when the limb is unweighted and usually resolves during full weight-bearing. With chronicity, an obvious swelling over the dorsal aspect of the pastern region similar to high ringbone becomes evident.

### *Diagnosis*

- A tentative diagnosis usually can be made from the history and physical examination of the horse.

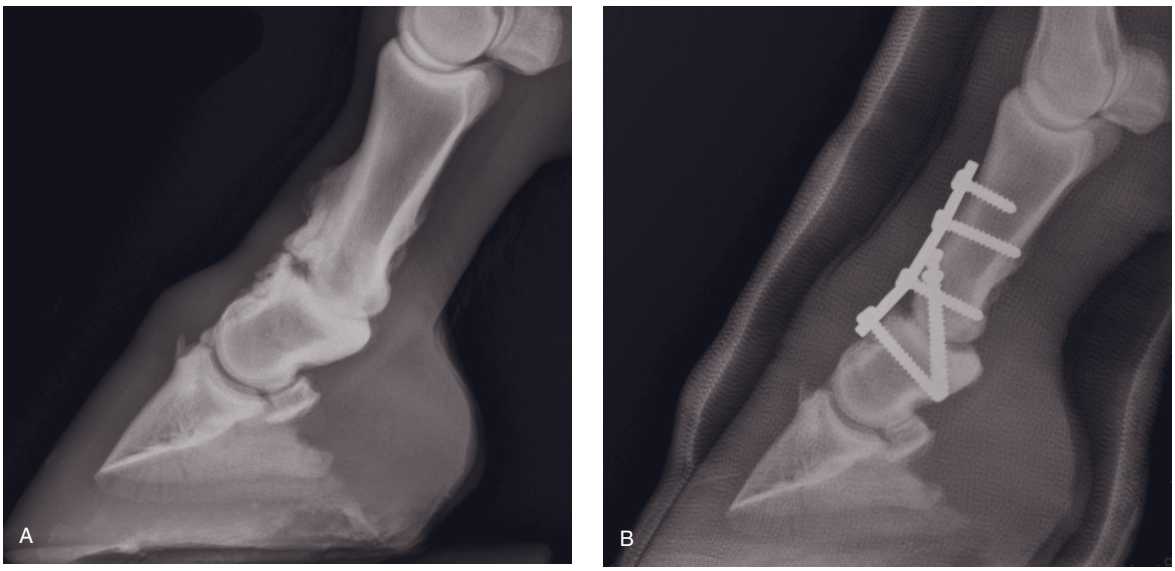



**Figure 6.9.** Lateral radiographs before (A) and after arthrodesis (B) of a horse with chronic palmar luxation of the PIP joint with secondary OA.

- Radiographs should be taken to confirm the diagnosis and to identify concurrent abnormalities such as fractures or OA. Stress films may be needed to confirm medial/lateral subluxation because the phalanges often remain in correct anatomic alignment unless pulled medially or laterally.
- Dorsal and palmar/plantar subluxations/luxations are usually obvious on standing lateral-to-medial views of the pastern (Figures 6.7, 6.8).

### *Treatment*

- The treatment of choice for medial/lateral and palmar/plantar luxations of the PIP joint is arthrodesis of the joint (Figure 6.9). Medial/lateral luxations of the PIP joint do not respond well to casting alone and often develop secondary OA and persistent lameness.
- Conservative treatment of palmar/plantar luxations is usually unsuccessful. Chronic palmar/plantar luxations develop fibrosis of the PIP joint in an abnormal position, making surgical re-alignment difficult (Figure 6.9).
- Horses with intermittent dorsal subluxation with no apparent lameness may be treated conservatively with anti-inflammatory medication and a controlled exercise program. Horses with intermittent dorsal subluxation of the pelvic limb associated with excessive tension of the DDFT have been treated successfully with transection of the medial head of the DDFT.
- Dorsal subluxations also can be treated with arthrodesis if they fail to respond to other methods of treatment. Horses with dorsal luxation with secondary OA are best treated with arthrodesis.

### *Prognosis*

- Although there are few reports on long-term follow-up, the prognosis appears to be fair to good for horses with luxations/subluxations treated early by arthrodesis where good reduction and stabilization of the PIP joint was achieved.
- Three cases of bilateral acquired pelvic limb intermittent dorsal subluxation treated by tendonectomy of the medial head of the DDFT responded favorably to the treatment and the subluxation resolved between one to seven days post-operatively.

## FRACTURES OF THE MIDDLE PHALANX (P2)

### *Introduction*

- A variety of fracture types involving P2 have been reported, including osteochondral (chip) fractures, palmar/plantar eminence fractures, axial fractures, and comminuted fractures.

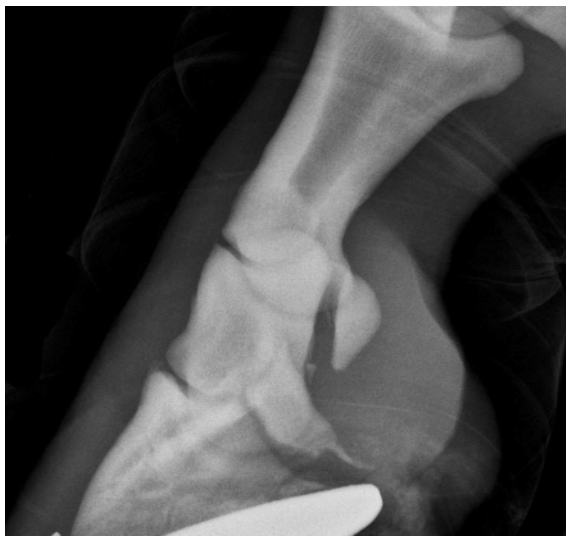


**Figure 6.10.** Oblique radiograph of the pastern region demonstrating a medial plantar eminence fracture of P2. This horse presented for a hindlimb lameness of two weeks duration.

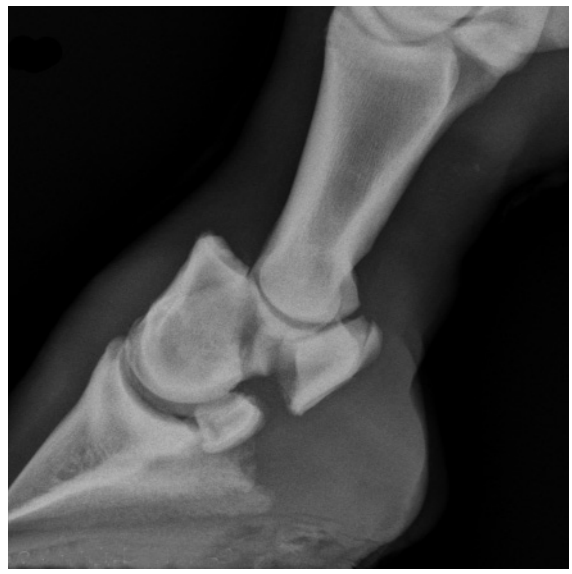


**Figure 6.11.** Lateral radiograph of the pastern demonstrating biaxial eminence fractures of P2. Internal fixation is recommended for these types of fractures to prevent palmar/plantar luxation of P1.

- Osteochondral fractures and axial fractures are rare, whereas eminence and comminuted fractures occur commonly.
- Fractures of P2 occur most commonly in the hindlimbs of middle-aged Western performance horses used for cutting, roping, barrel racing, pole bending, and reining.
- However, these fractures may occur in any horse of any breed during lunging, after kicks or falls, or from any form of single-event trauma.
- Thoroughbred and Standardbred racehorses and hunter/jumper horses appear to be at increased risk for osteochondral fractures; they can be located dorsally (most common) or on the palmar/plantar aspect.
- Palmar/plantar eminence fractures can be uniaxial (involving one eminence; Figure 6.10) or biaxial (involving both eminences; Figure 6.11).
- Comminuted fractures are the most common fracture and always involve the PIP joint (uniarticular) but often extend distally into the DIP joint (biarticular; Figure 6.12).

## *Etiology*

- Osteochondral fractures may occur from use, direct trauma to the bone (e.g., penetrating wound), or avulsion of soft tissue attachments (palmar/plantar aspect), or they may be associated with osteochondrosis.
- Palmar/plantar eminence fractures may occur from compression and rotation that is associated with sudden stops and short turns. They also may occur during PIP joint overextension which results in excessive tension of the SDFT and straight DSL, causing an avulsion of the eminence(s).
- Comminuted fractures are thought to result from a combination of compression and torsion (twisting) forces that occur with sudden stops, starts, and short turns. Most comminuted P2 fractures are thought to occur as a single-event injury, but a history of lameness in the affected limb may precede the fracture in some horses. Horses turned out for exercise after long-term confinement also have been reported to be at risk for comminuted P2 fractures.

## *Clinical Signs*

- Lameness associated with P2 fractures that do not disrupt the weight-bearing capabilities of P2 (osteochondral fragments, single eminence and simple axial fractures) can be variable. Some horses may have a history of an acute

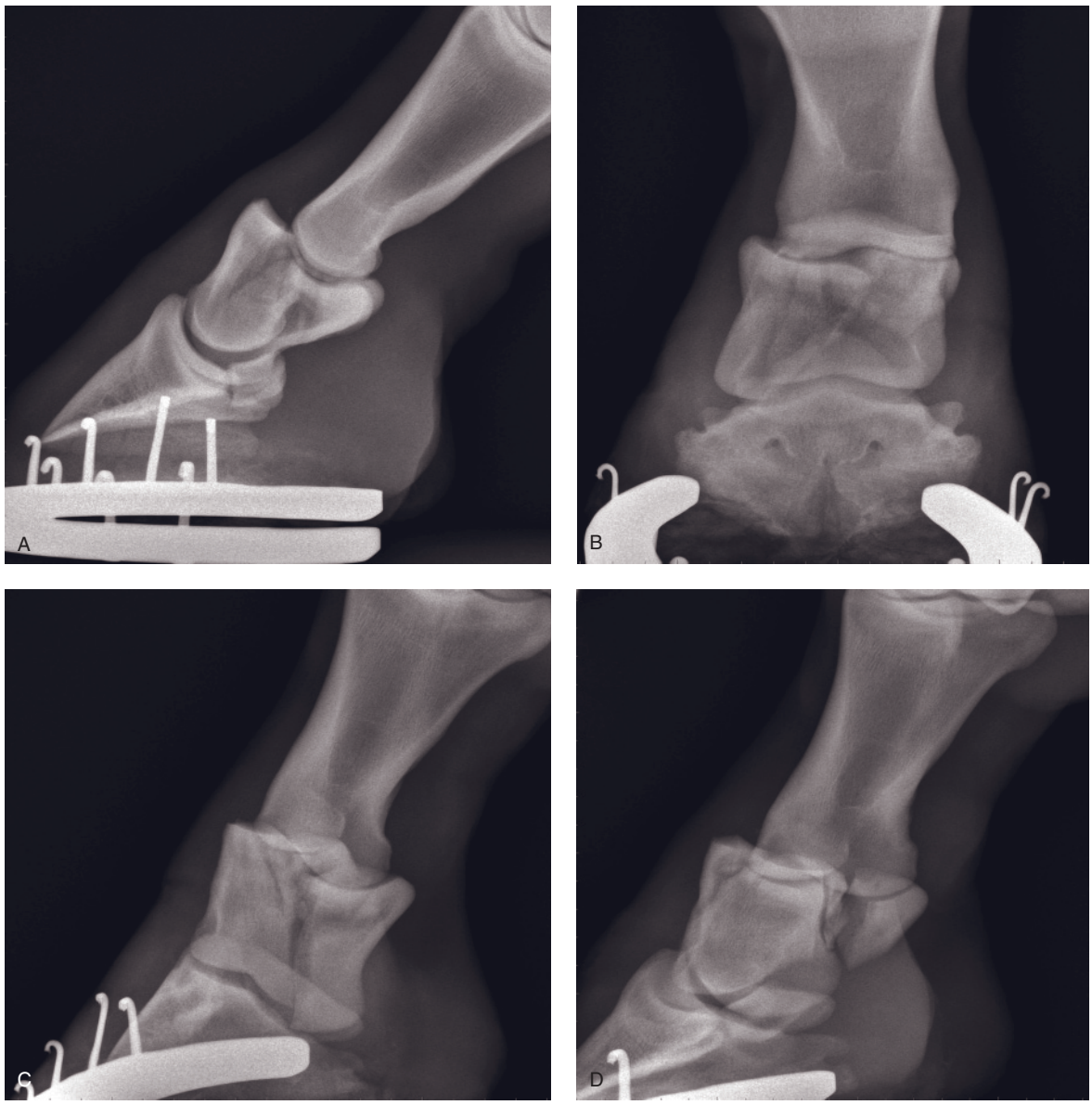



**Figure 6.12.** Lateral (A), dorsoplantar (B), and two oblique (C and D) radiographic projections of a horse with a comminuted P2 fracture. Multiple fracture lines are commonly seen and most comminuted fractures involve both the PIP and the DIP joint surfaces.

onset of lameness, whereas others may present for a chronic forelimb or hindlimb lameness.

- In most horses, exercise increases the severity of the lameness. Swelling of the pastern is not a reliable finding, but fetlock/phalangeal flexion and rotation of the pastern region often elicit a painful response. Circling at a trot usually exacerbates the lameness.
- Diagnostic anesthesia with a basisesamoid nerve block or IA anesthesia is often required to localize the lameness to the PIP joint region. However, diagnostic anesthesia is contraindicated with other types of P2 fractures because of the risk of fracture displacement.
- Horses with comminuted or biaxial P2 eminence fractures often have a history of acute

onset of severe lameness. Horses are usually very lame, painful to manipulation of the pastern, and crepitus may be felt. The pastern also may appear to be "unstable" during manipulation, and swelling may be present just above the coronary band in horses with comminuted fractures (due to effusion of the DIP joint).

### *Diagnosis*

- A definitive diagnosis requires a complete radiographic examination. Osteochondral fractures usually are easily diagnosed with the routine radiographic views. Additional views may be necessary with comminuted fractures so that the fracture location and configuration can be accurately assessed. The fracture configuration has considerable bearing on the treatment method selected as well as the prognosis for future soundness (Figures 6.10 to 6.12).
- Computer tomography (CT) of comminuted P2 fractures has been shown to be helpful in defining the fracture configuration in comminuted fractures (Figure 4.77). The use of CT can be especially important if there is doubt about whether a comminuted P2 fracture can be repaired and the extent of involvement of the DIP joint.

### *Treatment*

#### Osteochondral Fractures

- Fracture fragments associated with the PIP joint that contribute to lameness are best removed with arthroscopy. Removal with an arthrotomy is acceptable but less desirable.

#### Eminence Fractures

- Uniaxial or biaxial eminence fractures of P2 that involve the PIP joint are best treated by arthrodesis of the PIP joint.
- A single, narrow, dorsally applied three-to four-hole plate with two transarticular screws is recommended for horses with uniaxial eminence fractures, and two three-to four-hole dorsally applied plates are recommended for biaxial eminence fractures (Figure 6.13).
- For adult horses. 4.5-mm DCP, LC-DCP, or LCP plates may be used, whereas 3.5-mm plates may be suitable for foals.
- Although casting (half-limb fiberglass cast) alone has been reported as an acceptable method of treatment, it should be reserved for cases in which pasture or breeding soundness is desired and economic constraints dictate the approach.
- Single eminence fractures rarely heal back to the parent bone with casting, and secondary OA of the PIP joint leads to chronic lameness
- If internal fixation of biaxial P2 fractures is not elected, then transfixation pin-casts or another type of external fixator is recommended over casting alone to maintain phalangeal alignment.

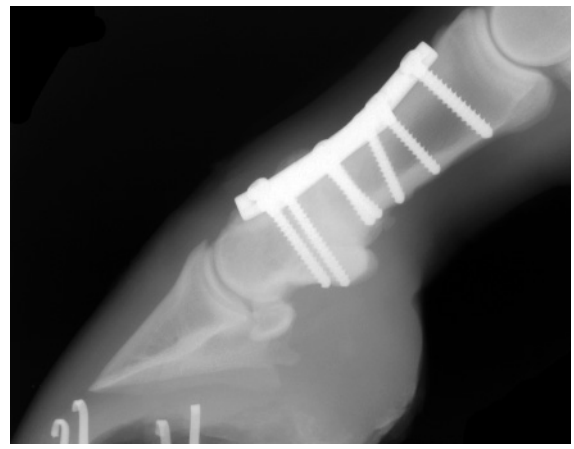


**Figure 6.13.** Lateral radiograph one year after the biaxial eminence fracture of P2 depicted in Figure 6.11 was repaired with two plates.

#### Comminuted P2 Fractures

- In general, comminuted P2 fractures should be repaired with some type of internal fixation if at all possible. Currently, the use of two narrow DCP or LCP plates is usually preferred (Figure 6.14).
- Horses with an intact strut of bone spanning from the PIP to the DIP joint are ideal candidates for internal fixation. Horses that do not have an intact bony strut yet have large enough bony fragments for screw fixation also often benefit from internal fixation.
- Horses with highly comminuted P2 fractures (so-called "bag of ice") that do not have fracture fragments large enough to engage screws are best treated with transfixation pin-casts or another type of external fixator.
- Casting alone can also be used for these highly comminuted fractures but fracture collapse is not uncommon.

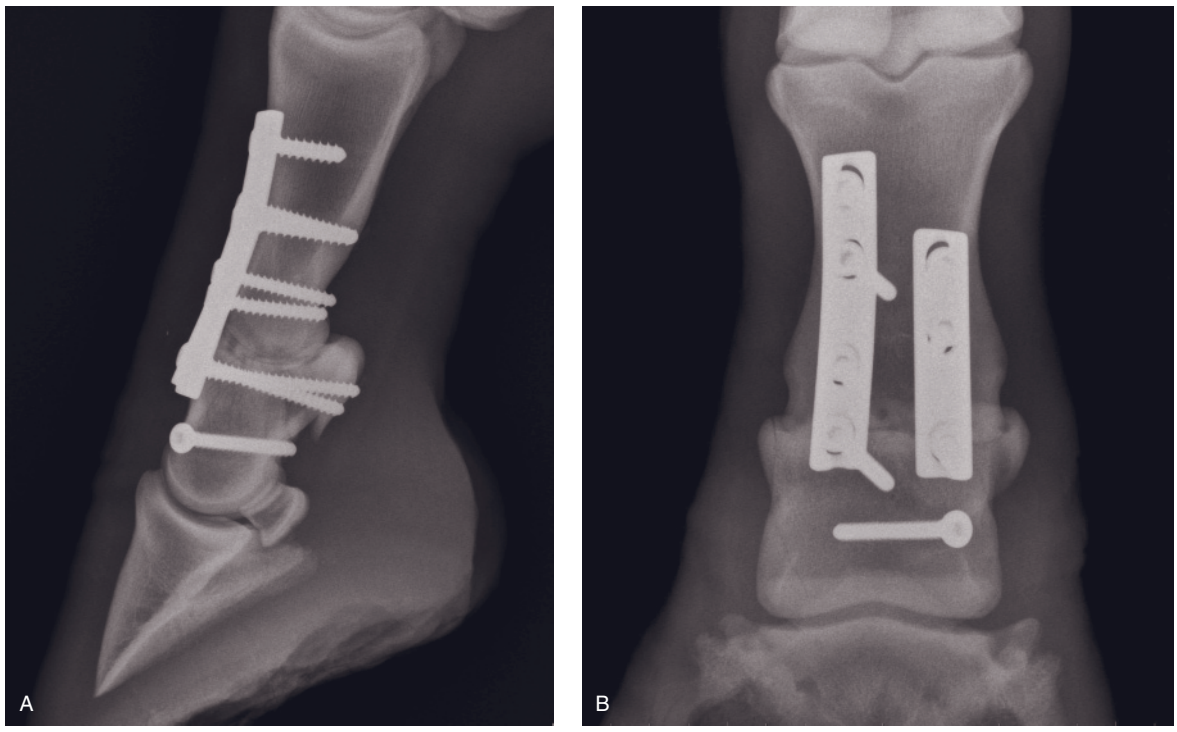



**Figure 6.14.** Lateral (A) and dorsoplantar (B) radiographs of the repair of the fracture depicted in Figure 6.12.

### *Prognosis*

- The prognosis for horses with osteochondral fractures treated by arthrotomy or arthroscopy appears to be very good for return to full serviceability.
- The prognosis for uniaxial or biaxial palmar/plantar eminence fractures treated by arthrodesis also is very good for return to performance and should be considered similar to horses treated for OA of the joint.
- Horses with comminuted fractures that only involve the PIP joint usually have a good prognosis for return to athletic performance provided they are treated with internal fixation.
- The prognosis for horses with biarticular comminuted P2 fractures is often dictated by the amount of fracture displacement at the DIP joint and the ability to reduce this displacement at the time of surgery. Internal fixation with bone plates increases both survival and return to athletic function. In general, horses with biarticular comminuted P2 fractures should be considered to have a 40% to 50% chance of returning to performance after internal fixation.

## FRACTURES OF THE PROXIMAL PHALANX (P1)

### *Introduction*

- Fractures of P1 occur frequently and can be broadly categorized into noncomminuted and comminuted fractures.
- Fracture configurations (excluding osteochondral fragmentation) range from small fissures that enter the MCP/MTP joint to highly comminuted biarticular fractures ("bag of ice").
- Stress or fatigue-type fractures that may not be identified on routine radiographs also may occur in performance horses.
- Noncomminuted P1 fractures have been classified into several types:
  1. Midsagittal or sagittal fractures: Exist primarily in the sagittal plane and begin at the MCP/MTP joint
     a. Short (extend less than 30 mm in length distally)
     b. Long (extend more than 30 mm in length distally; Figure 6.15)
     c. Complete (exit the lateral cortex or are biarticular)

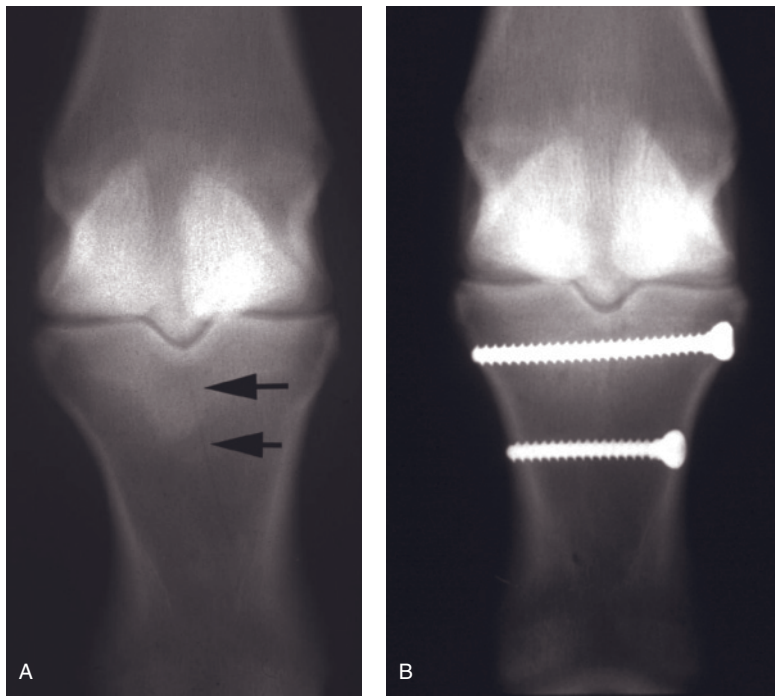



**Figure 6.15.** Dorsopalmar radiographs of a long sagittal P1 fracture before (left) and after (right) lag screw fixation. This is the most common type of P1 fracture.

2. Dorsal frontal fractures: Begin at the MCP/MTP joint in the frontal plane and extend to the dorsal cortex or distally toward the PIP joint; can be incomplete or complete
3. Distal joint fractures: Involve the PIP joint
4. Palmar/plantar eminence fractures: Involve the MTP/MCP joint (Figure 6.16)
5. Physeal fractures: Usually Salter-Harris Type 2
6. Oblique or transverse diaphyseal fractures

- Comminuted P1 fractures can range from fairly simple three-piece fractures to the "bag of ice" type injury (Figure 6.17). For treatment purposes, they can be divided into fractures that have an intact cortex (strut) of P1 from the proximal to distal joint surfaces (moderately comminuted) and fractures that do not have an intact bone strut (severely comminuted).
- Less common types of P1 fractures include proximal medial collateral ligament avulsion fractures (Figure 6.18), dorsal nonarticular fractures, and stress or fatigue fractures.
- Sagittal P1 fractures occur most commonly and are primarily seen in racing Thoroughbreds and Standardbreds.
- Comminuted P1 fractures also occur commonly in racing Thoroughbreds and Standardbreds but may also occur in any horse at pasture or at exercise from a single traumatic event.

### *Etiology*

- A combination of longitudinal compression in conjunction with asynchronous lateral-to-medial rotation of P1, or twisting of P1 in relation to the MC/MT, may be the cause. During normal weight-bearing, the convex sagittal ridge of the distal end of the MC/MT fits into the concave groove in the proximal surface of P1. If this alignment is not perfect, the convex sagittal ridge may act as a wedge to create the fracture.
- In most cases, a combination of axial weight-bearing and torsional forces usually contribute to P1 fractures.
- Stress or fatigue type fractures also occur in P1 and may precede the development of a radiographic apparent sagittal P1 fracture or may predispose to a comminuted fracture. Stress fractures tend to occur most commonly

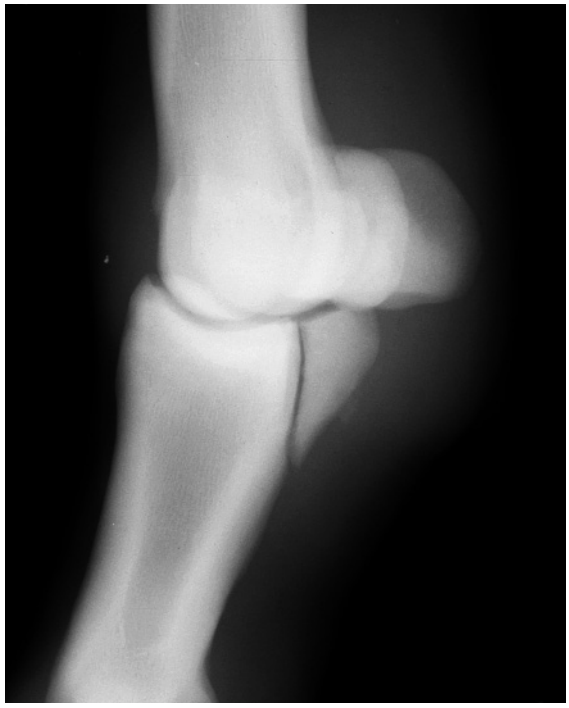


**Figure 6.16.** Lateral radiograph of the fetlock demonstrating a fracture of the palmar eminence of P1. This fracture was an acute injury and was repaired by lag screw fixation. Smaller, chronic fractures are more common in this location and are often removed if they are problematic.

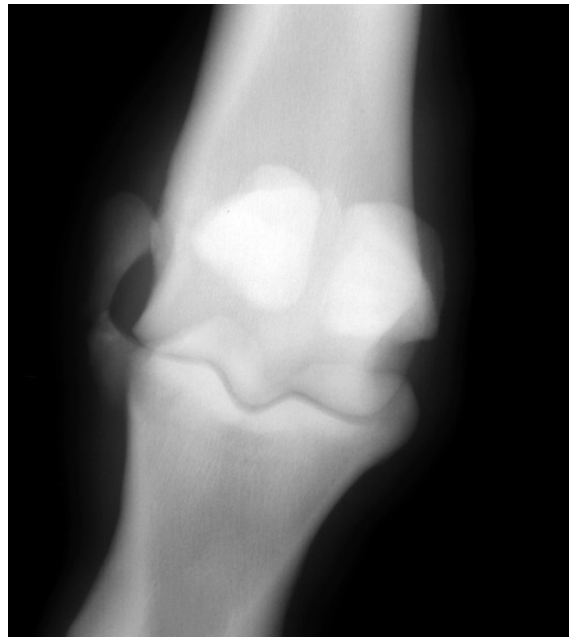


**Figure 6.18.** Oblique radiograph of the fetlock demonstrating avulsion fractures of the medial collateral ligament from the proximal aspect of P1. An open approach to the fetlock was used to reduce the fractures and repair with lag screws.

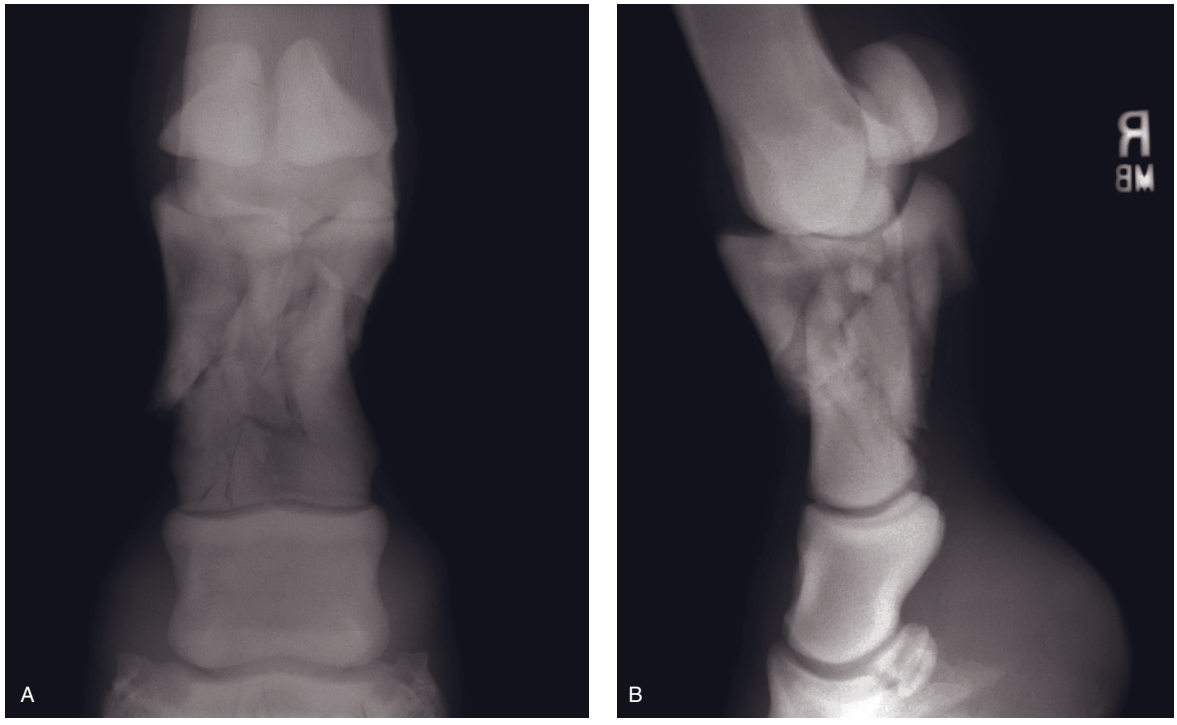



**Figure 6.17.** Lateral (A) and dorsopalmar(B) radiographs of a horse with a severely comminuted fracture of P1. Internal fixation is not possible with these types of fractures and transfixation pin-casts or external fixators are usually used to attempt salvage of these horses.

in the midsagittal groove at the proximal aspect of the bone, the same area where most P1 fractures originate.

### *Clinical Signs*

- The clinical signs associated with P1 fractures are variable and depend on the fracture type and degree of fracture propagation.
- Horses with incomplete sagittal fractures may demonstrate moderate pain and lameness initially, but it may be short in duration. However, fetlock effusion is usually present and a painful response is often elicited with flexion and rotation of the phalanges.
- Horses with complete sagittal fractures are usually quite lame (grade 3 to 4/5) and fetlock effusion and swelling of the pastern region is usually apparent.
- Horses with comminuted fractures are usually non-weight-bearing and may show signs of physical distress such as sweating. The pastern region is often obviously swollen, and crepitus and instability are palpable.
- Perineural anesthesia is usually unnecessary to make the diagnosis but some horses with short sagittal P1 fractures may present for a routine lameness evaluation. Perineural anesthesia is contraindicated if any type of P1 fracture is suspected because it will increase the risk of fracture propagation.

### *Diagnosis*

- Radiographs are required to characterize the type of P1 fracture and dictate the appropriate treatment. The radiographic examination should include at least four views, but additional views at varying angles may be necessary to accurately document the fracture configuration.
- Midsagittal fractures are often readily apparent on the DP view but some short, incomplete fractures may be difficult to see radiographically.
- In horses with comminuted P1 fractures, the presence or absence of an intact bony strut that spans from the MCP/MTP to the PIP joints is an important radiographic feature.
- Similar to comminuted P2 fractures, CT can be very beneficial to more accurately assess the degree of comminution of P1 fractures and to aid in preoperative planning for surgery (Figure 4.80).

### *Treatment*

#### Noncomminuted P1 Fractures

- The decision on how to treat horses with noncomminuted fractures usually depends on the fracture type, fracture location and length, degree of displacement, and intended use of the horse.
- Horses with displaced fractures that are not treated with internal fixation have a reduced chance of returning to performance.
- Most noncomminuted P1 fractures are best treated with internal fixation using lag screws placed through stab incisions (Figure 6.15).
- Short, incomplete sagittal fractures can be treated without surgery but most are treated with lag screw fixation. Fracture propagation is a risk of treating short, incomplete sagittal fractures conservatively, and a recent study found an improved prognosis in non-racehorses with short incomplete saggital fractures that were treated with lag screws.
- Horses with long (greater than 30 mm) sagittal incomplete fractures that are to be used for racing should be treated with lag screw fixation placed through stab incisions followed by external coaptation.
- Complete sagittal fractures that extend distally from the MCP/MTP joint to involve the PIP joint or that exit the lateral cortex are best treated by internal fixation and coaptation. These fractures are often displaced and generally can be better reduced with open approaches to P1 followed by lag screw stabilization. A distal limb cast is usually recommended after surgery for 2 to 4 weeks, depending on the security of the fixation.
- Bandaging and/or external coaptation has been used alone in cases in which breeding soundness is the objective or if there are economic constraints. These horses may develop considerable exostosis at the fracture site and secondary OA of the MCP/MTP joint.
- Dorsal frontal incomplete or complete nondisplaced P1 fractures can be treated by rest and bandaging or by internal fixation using lag screws, depending on the fracture size. Arthroscopic examination of the fetlock joint should be considered to visualize the dorsal articular margin and debride damaged cartilage if needed.

#### Comminuted Fractures

- The objective for treatment of most horses with comminuted P1 fractures is usually to

preserve the horse for breeding purposes or pasture soundness. Even horses with only moderately comminuted P1 fractures repaired surgically rarely return to racing.

- The goals of surgery are usually to restore the articular congruity of the joint(s) involved and to stabilize the fracture to maintain bone length (Figure 6.19).
- Methods for treatment include:
  1. External coaptation alone
  2. External skeletal fixation alone (transfixation pin-casts or Nunamaker skeletal fixator)
  3. Lag screw fixation through stab incisions with or without external skeletal transfixation
  4. Open reduction with lag screws and external coaptation
  5. Open reduction with plates and screws and external coaptation
  6. Open reduction combined with transfixation pin-casts
- Internal fixation is usually recommended in horses with moderately comminuted P1 fractures (those with an intact strut of P1 that extends from the proximal to distal joint surfaces) that permit fracture realignment (Figure 6.19). These horses have a much greater chance of surviving than do those without an intact strut of bone.
- External skeletal fixation is usually the treatment of choice to repair severely comminuted P1 fractures that lack an intact bony strut (Figure 6.17), and for fractures that are open or have a severely compromised blood supply. Either transfixation pin-casts or the Nunamaker external fixator may be used in these horses.
- Internal fixation with either screws alone or plates and screws may be combined with a transfixation pin-cast in some horses. The transfixation pin-cast is used to protect the implants from potential failure in horses that may not have an intact strut of bone.
- Casting alone can be used to treat some horses with comminuted P1 fractures, but it is less than optimal. Case selection is important and the fracture should be minimally comminuted and be relatively stable to prevent axial collapse of the fracture.

## *Prognosis*

### Noncomminuted Fractures

- These horses generally have a very good prognosis for long-term survival and many return

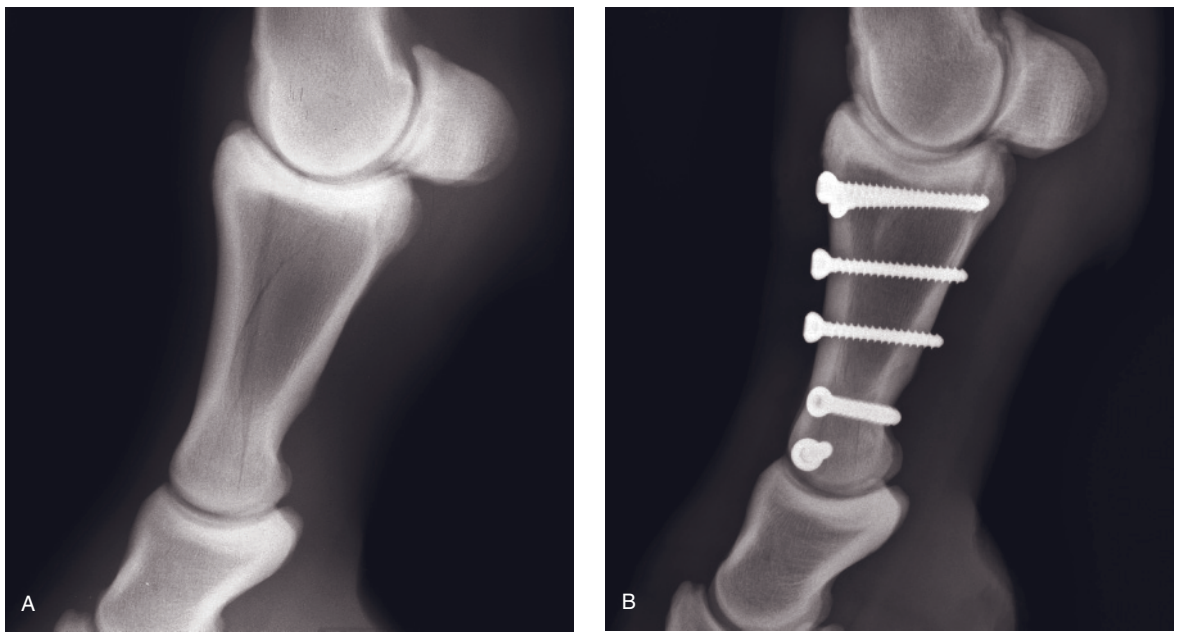



**Figure 6.19.** Lateral radiographs of a nondisplaced, moderately comminuted P1 fracture before (A) and after repair (B) with multiple lag screws placed through stab incisions.

to performance, although often at a reduced level.
- In one study in racehorses, a significantly lower percentage of horses returned to racing following repair of complete sagittal fractures that extended into the PIP joint (46%) than following repair of short, incomplete sagittal fractures (71%), long, incomplete sagittal fractures (66%), or complete sagittal fractures that extended to the lateral cortex (71%).
- In another study in Standardbred racehorses, 89% of the horses returned to racing, but at significantly decreased performance levels.

### Comminuted Fractures

- Horses with open or closed severely comminuted fractures that do not permit reconstruction of the fragments remain difficult to treat and have only a fair prognosis for survival, regardless of the treatment approach used.
- Moderately comminuted P1 fractures (those with an intact boney strut) usually can be repaired with internal fixation; a 92% successful outcome has been reported.

## DESMITIS OF THE DISTAL SESAMOIDEAN LIGAMENTS (DSLs)

### *Introduction*

- There are three DSLs: straight (superficial), paired oblique (middle), and paired cruciate (deep) (Figure 1.10). All of the ligaments originate from the base of the proximal sesamoid bones and intersesamoidean ligament. See Chapter 1 for further information regarding the anatomy of the DSLs.
- Desmitis of the oblique, straight, and cruciate DSLs occurs in all types of performance horses; injury to the oblique DSL is most common.
- Horses that jump (e.g., event horses, show jumpers, field and show hunters, steeplechasers, and timber race horses) and race appear to be particularly prone to these injuries.
- The medial branch of the oblique DSL is more commonly injured than the lateral branch and these injuries are thought to more common in the forelimb than the hindlimb.
- Horses with a valgus or varus limb conformation or long, sloping pasterns may be at increased risk for injury.

### *Etiology*

- The DSLs are an important part of the suspensory apparatus which provides resistance to extension of the MCP/MTP joint during the stance phase. Hyperextension of the MCP/MCT joint can result in supraphysiologic strains in the suspensory apparatus, which may lead to failure of the DSLs.
- The straight DSL would most likely be injured during hyperextension, but injury to this ligament is less common than to the oblique DSL.
- Injuries to the oblique DSLs usually occur unilaterally, probably as a result of asymmetric loading caused by abnormal conformation, lateral/medial foot imbalances, a misstep or poor footing.

### *Clinical Signs*

- In acute cases (less than three weeks duration), mild swelling of the palmar/plantar surface of the pastern region may be present as a result of digital flexor tendon sheath (DFTS) effusion. Heat and pain with digital pressure also may be palpable.
- Horses with chronic injuries often present for a routine lameness evaluation and the location of the lameness must be localized with perineural anesthesia.
- The lameness is usually mild to moderate in severity, positive to fetlock or phalangeal flexion, and worsened when the affected limb is on the inside of the circle.
- Palpation of the DSLs is best performed with the foot held off the ground and the MCP/MTP joint flexed so the flexor tendons are relaxed. Careful digital palpation dorsal to the flexor tendons midway between the heel bulbs and the proximal sesamoid bones may reveal firm swellings and/or pain.
- Swelling of a DSL must be differentiated from swelling of the medial or lateral branch of the SDFT, which is also located in the mid-pastern region.
- Perineural anesthesia of the PD nerve at the base of the sesamoid bone (basisesamoid block) should improve the lameness in most cases. However, an abaxial sesamoid or low four-point block may be necessary if the ligament injury is located proximally in the pastern.

### *Diagnosis*

- Diagnostics that may help document an abnormality to one of the DSLs include radiography, ultrasound, and MRI.

- Radiographic abnormalities that may suggest a previous or concurrent injury of a DSL include: enthesiophyte formation on P1 or the sesamoid bones, avulsion fractures/fragments, and dystrophic mineralization within one of the DSLs.
- Bone fragments also have been observed on the nonarticular proximal extremity of P1 and at the base of the sesamoid bones that may involve the oblique, cruciate, or short DSLs.
- Sonographic evidence of acute desmitis is manifested by a diffuse increase in ligament size, fiber disruption, discrete core lesions, and peri-ligamentous fluid surrounding the affected ligament.
- Although ultrasound can be useful to diagnose problems in the DSLs, lack of ultrasound abnormalities does not rule out a problem.
- Currently, MRI is the best diagnostic tool to make a definitive diagnosis (Figures 4.75, 6.20).

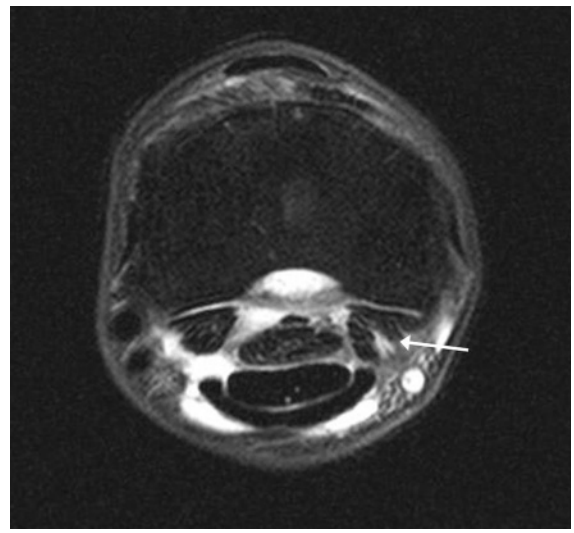


**Figure 6.20.** Axial proton density MRI image showing high signal intensity (arrow) in the lateral branch of the oblique DSL in the pastern region.

### *Treatment*

- Injuries to the DSLs are treated very similarly to other soft tissue problems such as tendinitis.
- In acute cases, confinement, cold therapy, pressure/support wraps, and administration of NSAIDs are recommended. Cold therapy in the form of an ice water slurry applied for 30 minutes twice a day for the first 48 hours after the acute injury appears to be most beneficial.
- A six-month rest and rehabilitation program is currently recommended. This usually involves a short period of stall confinement depending on the severity of the injury (three to six weeks), followed by increasing periods of hand-walking and controlled exercise. Clinical evaluation should be performed at four to six weeks, and if the horse has improved, controlled exercise can be increased.
- Adjunctive treatments that may be used in addition to the rehabilitation protocol include extracorporeal shockwave, ligament splitting, intrasynovial treatment of the DFTS, and intralesional treatment of the damaged ligament with stem cells or platelet-rich plasma (PRP).
- Nonarticular basal sesamoid fragments that may be associated with DSL avulsion injuries can be removed using a "keyhole" surgical approach through the DFTS.

### *Prognosis*

- The prognosis for horses with DSL injuries to return to performance has historically been guarded because of the high probability of re-injury.
- More recent studies have reported that 66% to 90% of horses with DSL injuries or avulsion fractures of the proximal sesamoid bones returned to performance following treatment.
- Recurrence of DSL desmitis is always a possibility, similar to other soft tissue injuries.

## SDFT AND DDFT INJURIES IN THE PASTERN

### *Introduction*

- In general, injuries to the SDFT in the pastern region occur most frequently in the forelimb and injuries to the DDFT within the DFTS occur most frequently in the hindlimbs. (Injuries to the DDFT that are associated with navicular syndrome are covered in the Foot section).
- Injuries involving the SDFT most commonly involve the branches of the SDFT located outside the DFTS. The SDFT branches at the level of the MCP/MTP joint and the medial

and lateral branches insert on the palmar/plantar eminences of P2.
- Injuries to the DDFT within the pastern are nearly always within the DFTS, often cause effusion of the sheath, and may contribute to chronic tenosynovitis of the DFTS (Figure 6.21).

### *Etiology*

- Injuries to the SDFT in the forelimbs are usually associated with hyperextension of the MCP joint resulting in non-physiologic stretching and overload of the SDFT.
- The cause of DDFT injuries within the DFTS is unknown but hyperextension of the MCP/MTP joint and overstretching of the tendon is also likely.

### *Clinical Signs*

#### SDFT Branch Injuries

- Lameness usually occurs at the onset of injury with focal heat, swelling, and sensitivity noted on palpation. However, careful palpation and comparison of the medial to lateral branches is important to detect differences in size, heat, and pain as these injuries can be easily missed.
- The medial SDFT branch appears to be more frequently injured than the lateral branch.

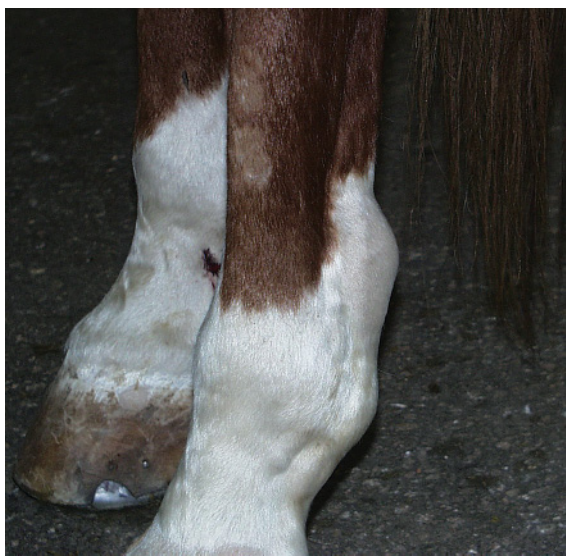


**Figure 6.21.** This horse was lame in the left hindlimb and had severe effusion of the digital flexor tendon sheath. Lesions of the DDFT within the tendon sheath appear to be more common in the hindlimbs than the forelimbs.

#### DDFT

- Deep digital flexor tendinitis occurs in a variety of sport horses and typically presents as an acute-onset, unilateral, moderate to severe forelimb or hindlimb lameness that is persistent.
- Distension of the DFTS often occurs in conjunction with the injury and many horses present with chronic tenosynovitis of the DFTS of undetermined cause.
- Lameness is often worse on a soft surface and generally improves with perineural anesthesia above the proximal sesamoid bones or with intrasynovial anesthesia of the DFTS.

### *Diagnosis*

- Ultrasonography is currently the most commonly used method to diagnose branch lesions of the SDFT and abnormalities within the DDFT.
- Core lesions, followed by diffuse injury to the affected SDFT branch. are the most common lesions identified but the absence of swelling may result in false-negative results.
- A DDFT lesion may involve one or both lobes and is typically characterized by enlargement and alteration of the tendon with or without a hypoechoic region (Figure 6.22).
- Dystrophic mineralization may be seen with chronic injuries.
- The sensitivity of ultrasound appears to be worse for lesions of the DDFT than the SDFT. Because of these limitations, an MRI examination is thought to be superior to an ultrasound examination to characterize the location, type, and severity of damage to both the SDFT and DDFT within the pastern.
- Tenoscopy of the DFTS is also a useful diagnostic tool to document lesions of both SDFT and DDFT that may not be visible with ultrasound.

### *Treatment*

- Branch lesions of the SDFT are usually treated with a controlled rehabilitation program similar to any bowed tendon injury.
- If the SDFT lesion is within the DFTS or if DFTS effusion is present, tenoscopy is often

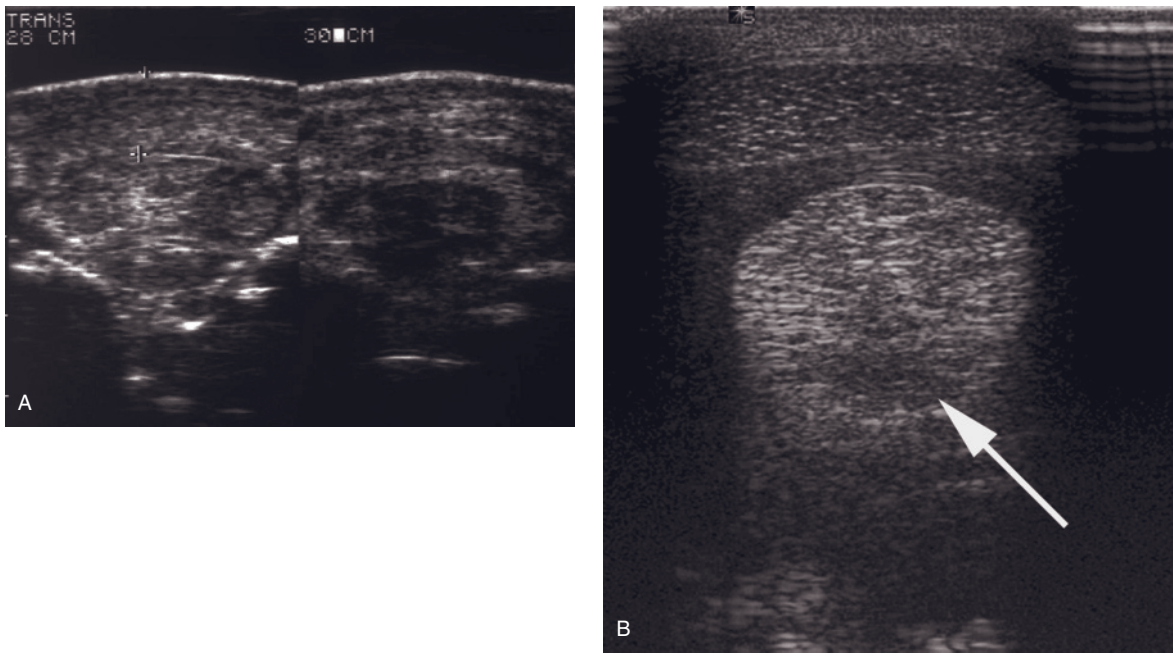



**Figure 6.22.** Ultrasound images demonstrating abnormalities of the DDFT within the tendon sheath at the level of the sesamoid bones (A) and above the sesamoid bones (arrow) (B) in two different horses.

helpful to further diagnose the specific problem and to debride the damaged tendon.
- Lesions of the DDFT at the level of the pastern are often within the DFTS and tenoscopy is often beneficial.
- Additional treatment options include medication of the DFTS and intralesional injection of PRP or stem cells directly into the damaged tendon. See Chapter 9 for more detail on these treatments.

### *Prognosis*

- In general, soft tissue injures of the SDFT and DDFT in the pastern region can be difficult to diagnose and affected horses have a reasonable chance of returning to their intended use.
- Injuries to the SDFT and DDFT in the pastern region are prone to recurrence.
- Horses with SDFT branch injuries are thought to have a poorer prognosis to return to racing than those with SDFT injuries in the metacarpal region, with more frequent recurrence.
- Lesions of the DDFT within the DFTS can contribute to chronic tenosynovitis. In one retrospective study, 68% of treated horses were sound and 54% returned to preoperative level of performance.

## OSTEOCHONDRAL (CHIP) FRACTURES OF PROXIMAL P1

### *Introduction*

- Dorsal and palmar/plantar chip fractures of P1 are relatively common in racehorses.
- Most fractures of this type involve the dorsal eminences just medial (most common) or lateral to midline.
- Fractures of the lateral or medial palmar/plantar eminences occur less frequently than dorsal fractures.

### *Etiology*

- Excessive concussion and overextension of the joint are thought to contribute to both dorsal and palmar/plantar fractures.
- Overextension compresses the dorsal aspect of P1 against the MCIII/MTIII and contributes to

avulsion fractures of the palmar/plantar aspect of P1.
- Limb fatigue may contribute to fetlock overextension as is often noted at the end of races when the back of the fetlock nearly contacts the ground.

### *Clinical Signs*

- Effusion of the fetlock joint is commonly palpable and heat may be detected in acute injuries.
- Lameness can be variable but usually increases after exercise, and a workout or a race may cause the horse to be markedly lame.
- Passive flexion of the affected fetlock often elicits pain and a fetlock flexion test usually exacerbates the lameness.
- With chronicity, there may be fibrous enlargement of the dorsal aspect of the fetlock joint.
- Intrasynovial anesthesia is the most specific method to document that the fetlock is the cause of the lameness.

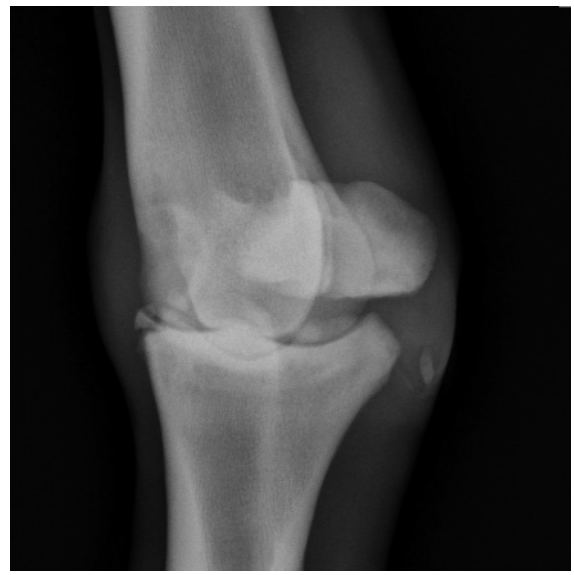


**Figure 6.23.** Slightly oblique radiograph of the fetlock demonstrating multiple osteochondral fractures of the proximal dorsal aspect of P1.

### *Diagnosis*

- A definitive diagnosis is usually made with radiographs. Both dorsal and palmar/plantar fractures are seen on the lateral view but oblique views are necessary to determine the exact location (medial vs. lateral; Figures 6.23, 6.24).
- Radiography of the contralateral fetlock is recommended because many of these fractures can be bilateral.
- Ultrasonography also may be used to diagnose chip fractures of the dorsal aspect of P1 and concurrent proliferative synovitis of the synovial pad if present.

### *Treatment*

- Arthroscopic removal of chip fractures is the treatment of choice for both dorsal and palmar/plantar fragments.
- Lesions commonly seen in association with P1 chip fracture include proliferative synovitis of the dorsal metacarpal synovial pad and cartilage erosion of the MCIII condyle.

### *Prognosis*

- The prognosis is usually good to excellent (approximately 80%) for horses to return to athletic soundness after arthroscopic removal of dorsal or palmar/plantar chip fractures.
- Factors that lower the prognosis include extreme large size of the fragment, chronicity, the degree of synovitis/capsulitis, and the amount of OA present.

## FRACTURES OF THE PROXIMAL SESAMOID BONES

### *Introduction*

- Fractures of the proximal sesamoid bones are common injuries in racing Thoroughbreds, Standardbreds, and Quarter horses.
- Types of sesamoid fractures include apical, abaxial (articular and nonarticular), apical/abaxial, midbody, basilar (articular and nonarticular), sagittal, and comminuted (Figure 6.25).
- The forelimbs are most frequently affected in the Thoroughbred and Quarter horse, whereas the hindlimbs are more frequently affected in the Standardbred (left hindlimb).
- Apical fractures are by far the most common and are usually articular and involve less than one-third of the bone (Figure 6.26).

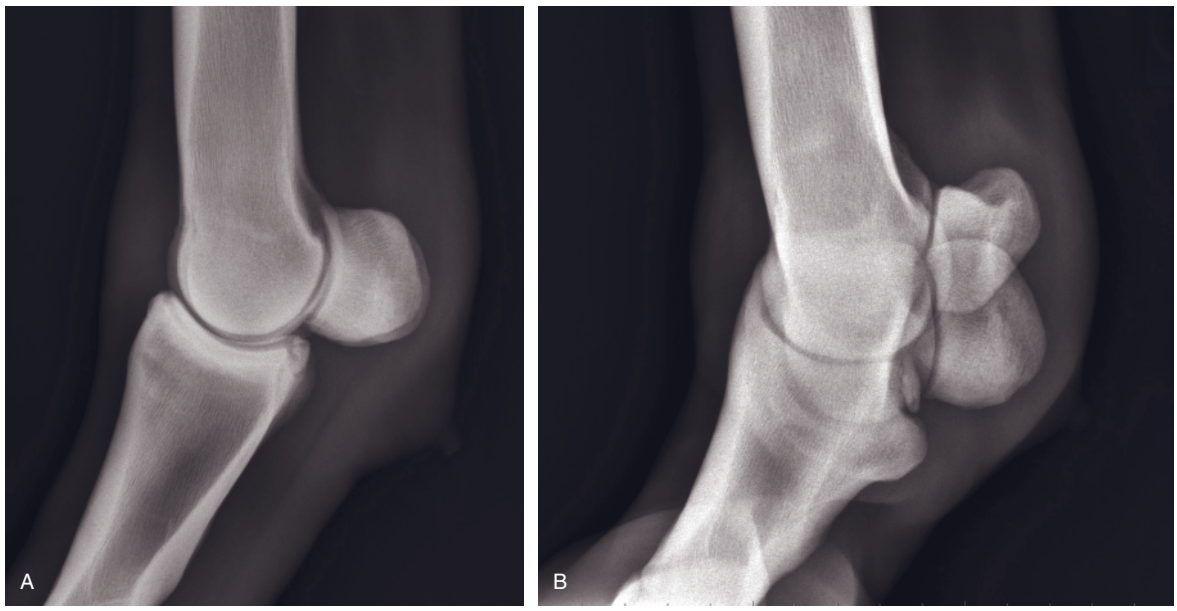



**Figure 6.24.** Lateral (A) and oblique (B) radiographs demonstrating a typical palmar/plantar osteochondral fragment of the first phalanx.

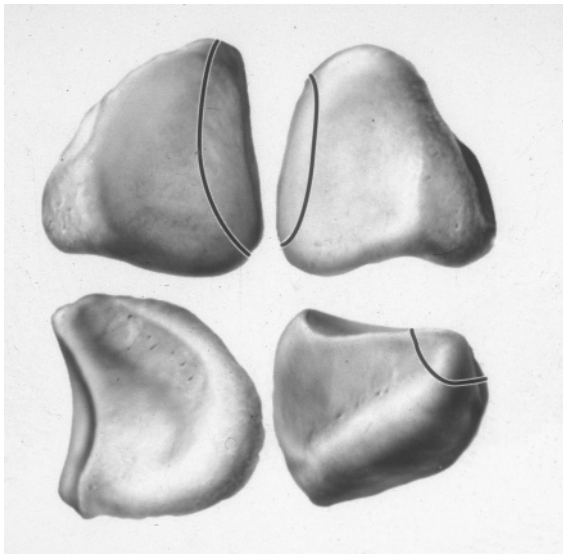


**Figure 6.25.** Articular abaxial sesamoid fractures. Reprinted with permission from Bertone AL: 1996. Fractures of the Proximal Sesamoid Bones. *In:* Nixon A. (ed) *Equine Fracture Repair*. Philadelphia, WB Saunders Company, 16:163–171.

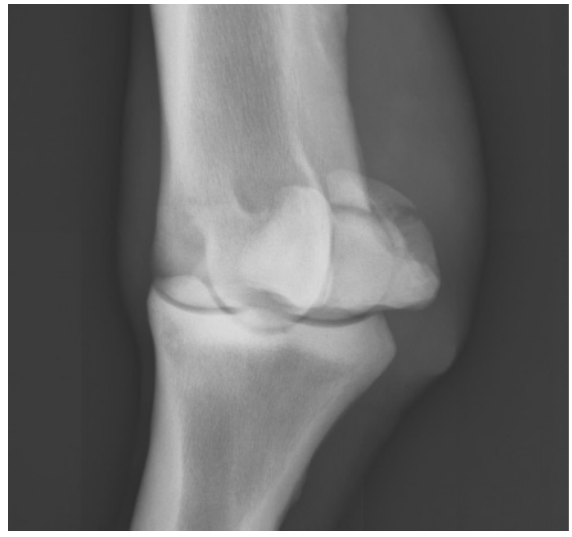


**Figure 6.26.** Oblique radiograph of a Quarter horse mare with a large apical sesamoid fracture.

- Basilar and abaxial fractures are uncommon and can be articular or nonarticular (Figure 6.27). Abaxial fractures require an oblique tangential radiograph to document if they are articular (Figure 6.28).
- The midbody transverse fracture divides the bone into equal portions and invariably enters the fetlock joint. Biaxial midbody fractures cause loss of the suspensory support apparatus, resulting in a breakdown injury.

### *Etiology*

- The cause of proximal sesamoid bone fractures is excessive tensile forces on the bone. Fetlock hyperextension (from muscle fatigue) maximally loads the sesamoid bones. When the sesamoid bone can no longer withstand the distraction forces applied to it by the suspensory ligament and distal sesamoidean ligaments, the bone fails.
- Other factors such as poor conditioning, improper trimming and shoeing, training and racing schedules, and poor conformation also may create additional stresses on the bone.
- Foals that have been confined to a box stall for several days and then turned out for free exercise with the dam appear to be at risk for sesamoid fractures (Figure 6.29).
- Direct trauma to the sesamoid bone such as with interference also may result in a fracture.

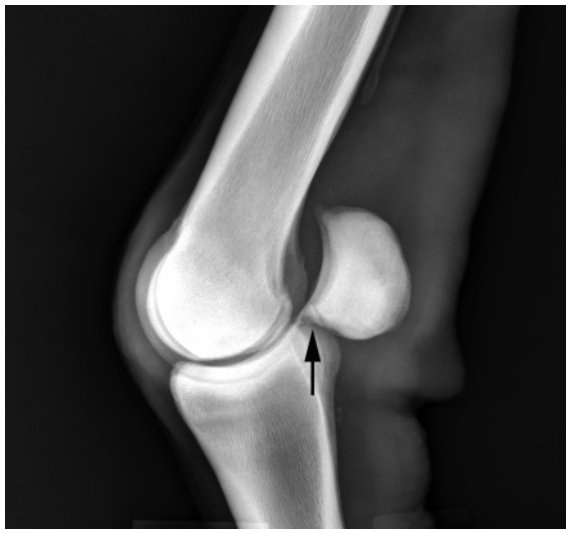


**Figure 6.27.** Flexed lateral radiograph demonstrating a small articular basilar fracture in a Thoroughbred racehorse. This fracture extended across the entire base of the medial sesamoid bone.

### *Clinical Signs*

- Horses with different types of sesamoid fractures may have similar clinical signs. Lameness is usually very pronounced in acute stages.
- Palpable fetlock effusion and heat, pain on direct palpation of the sesamoid bone, and pain with passive flexion of the fetlock are typical findings.
- The horse may be reluctant to bear weight on the limb and will not permit the fetlock to descend to a normal position during weight-bearing.
- Desmitis of the suspensory ligament and distal sesamoidean ligaments may occur concurrently with fractured sesamoids.

### *Diagnosis*

- The diagnosis is confirmed with a complete radiographic examination of the affected fetlock.
- The skyline projection of the abaxial surface of the sesamoid bone is usually needed to document if an abaxial fracture is articular (Figure 6.28).

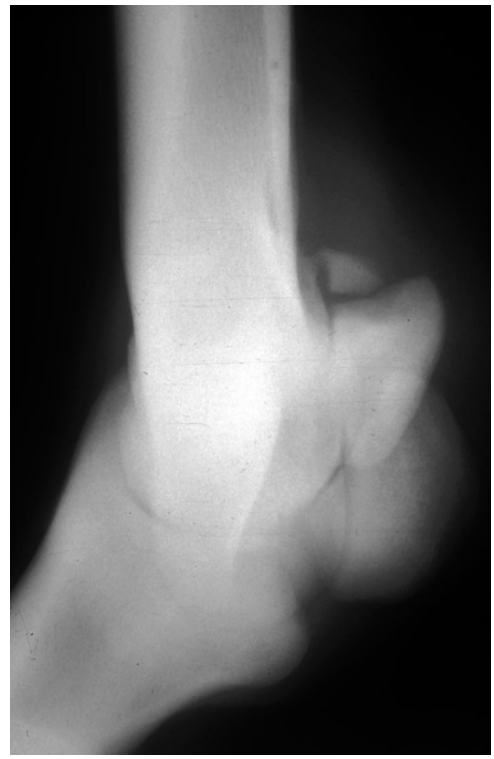
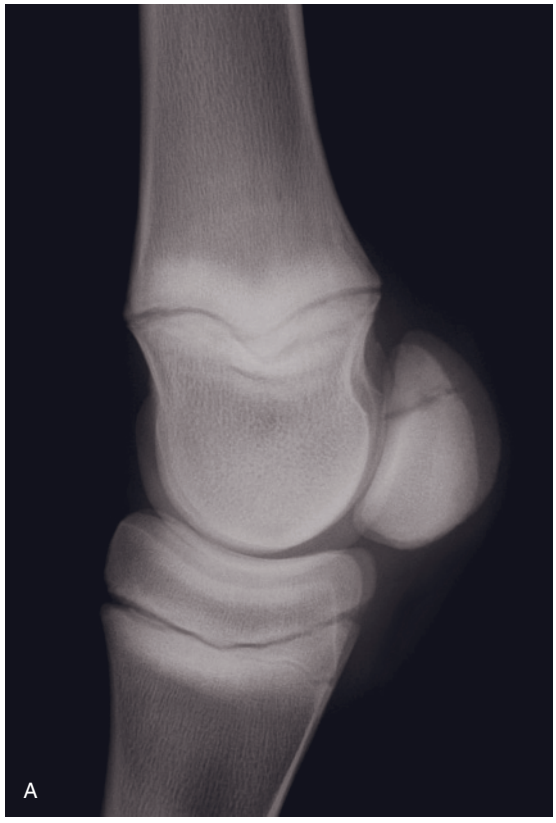


**Figure 6.28.** Skyline radiograph of the sesamoid bone that was performed to document the articular involvement of an abaxial sesamoid fracture.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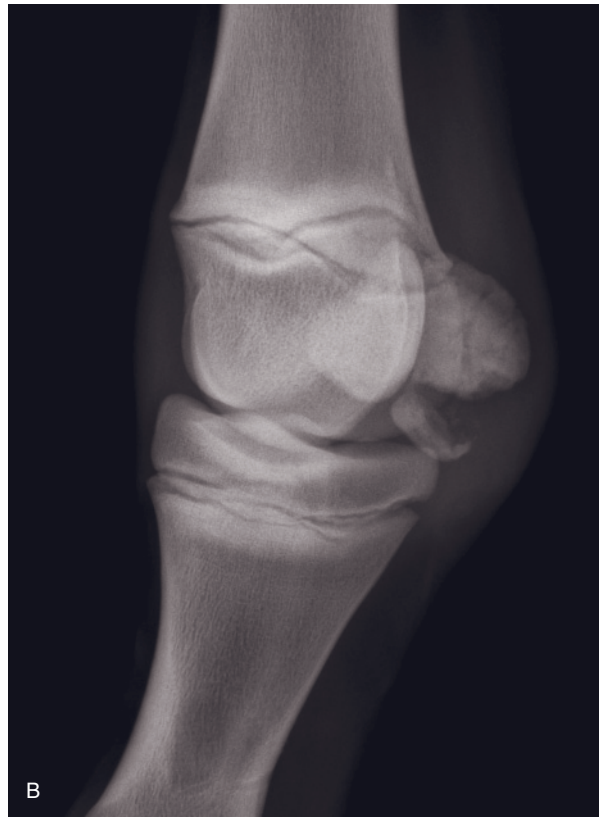
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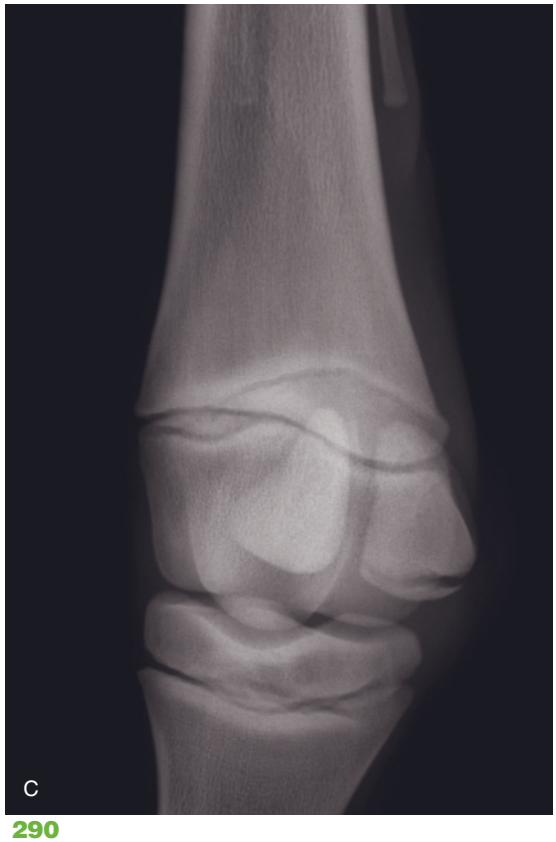
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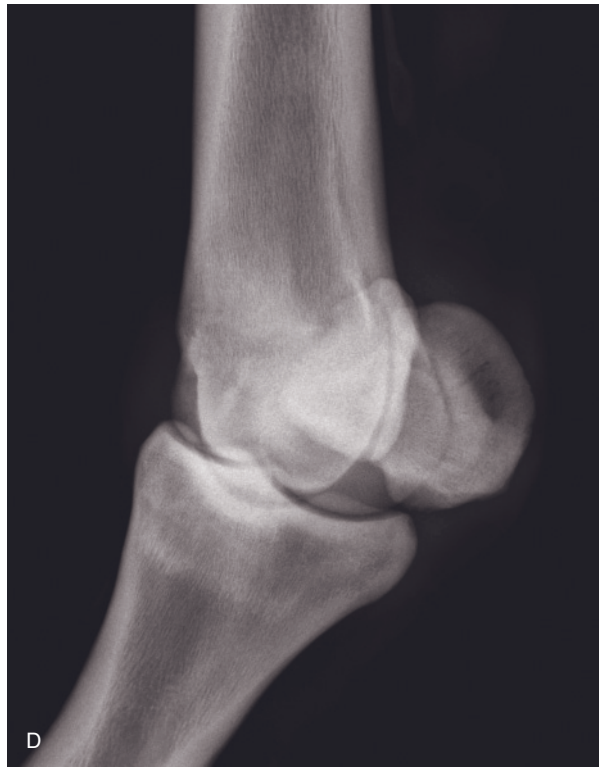
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**Figure 6.29.** (A) Lateromedial radiographic view of a nondisplaced apical sesamoid fracture. (B) Dorsomedial to palmarolateral radiographic view of a displaced basilar sesamoid fracture. (C) Dorsomedial to palmarolateral radiographic view of a mildly displaced medial basilar sesamoid fracture in a foal at time of injury. (D) Radiograph of the same foal in (C) at 16 months of age after healing. Courtesy of Robert Hunt.

- Ultrasonography is recommended with apical and basilar fractures to identify concurrent injury to the suspensory apparatus (suspensory and DSLs).

### *Treatment*

- The selection of treatment of sesamoid bone fractures is based on the location of the fracture and the intended use of the animal. Treatments include stall rest, cast application, surgical excision, lag screw fixation, and circumferential wiring.
- Generally, the conservative approach should be used in horses that will not be used for performance and in young foals without distraction of the fracture fragments.
- Stall rest (with or without external coaptation) for three to four months has been successful to obtain fibrous or partial bony union. These fractures heal by a weak fibrous union, and the fracture line will be observed on radiographic examination for prolonged periods of time.
- Surgical removal or repair when possible is the treatment of choice for fractures in horses intended for performance.
- Apical, articular abaxial, and basilar fractures involving less than one-third of the sesamoid bone are best treated by arthroscopic removal of the fragment (Figures 6.26 to 6.28).
- Midbody transverse fractures and large basilar transverse fractures of the proximal sesamoid bones have been treated successfully with lag screw fixation or circumferential wiring.
- Biaxial midbody or comminuted sesamoid bones (breakdown injury) are treated by humane euthanasia or arthrodesis of the fetlock.
- Casting or splinting is recommended for midbody fractures and comminuted fractures, or if clinical evidence of suspensory disruption accompanies the sesamoid fracture.
- Removal of nonarticular basilar fractures can be performed through an incision between the distal sesamoidean ligaments through the DFTS.

### *Prognosis*

- The reported prognosis for apical sesamoid fractures is good to excellent (88% of Standardbreds and 77% of Thoroughbreds return to racing).
- The prognosis for abaxial fractures is good (71% of Thoroughbreds or Quarter horses returned to racing).
- The prognosis for basilar fractures is fair (50% to 60% of Thoroughbreds return to racing). The prognosis for midbody fractures repaired by either lag screw fixation or circumferential wiring is also fair (44% to 60% return to performance).
- Conservative management reports are not available for comparison, but it is presumed that the prognosis is guarded for basilar or midbody fractures that are not treated surgically.
- The prognosis for fractures of the sesamoid bones that result in loss of the suspensory apparatus is poor.

## SESAMOIDITIS

### *Introduction*

- Sesamoiditis is characterized by pain associated with the proximal sesamoid bones and insertions of the suspensory ligament that result in lameness. It is observed frequently in young racehorses and hunters and jumpers, but can affect any type of horse.
- Primary disease of the suspensory ligament or distal sesamoidean ligaments can accompany this condition.
- Pain, heat, and inflammation can be detected at the insertion of the suspensory ligament during the active stages of the disease process, but marked lameness and limitations on performance can also occur with few detectable signs.
- Radiographs can reveal a range of changes from accelerated early remodeling response in the bones (increased size and number of vascular canals) to marked proliferation of bone along the abaxial margin of the sesamoid.

### Etiology

- The sesamoid bones undergo intense remodeling in response to training, and sesamoiditis only occurs if the stresses exceed the bones' capability to strengthen. Many young horses can have radiographic changes in the sesamoid bones consistent with sesamoiditis without lameness.
- Any unusual strain to the fetlock region may produce sesamoiditis. Repetitive overextension of the fetlock is thought to damage the interface between the branches of the suspensory ligament and the distal sesamoidean ligaments with the proximal sesamoid bone.
- The pain is thought to be due to the initiation of the remodeling response to bone stress and/or may reflect an inflammatory response from tearing of the attachment of the suspensory ligament.

### Clinical Signs

- In the early stage, minimal swelling will be observed, but increased heat may be palpable over the abaxial surface of the sesamoid bones.
- As the disease progresses, a visible enlargement of the soft tissues overlying the palmar surface of the fetlock can be seen due to fibrosis of the suspensory ligament.
- Firm palpation of the sesamoid bones and flexion of the fetlock usually elicit painful responses.
- The lameness can be variable in severity but is usually most evident during the first part of exercise and when exercised on hard surfaces.

### Diagnosis

- The radiologic changes of true sesamoiditis have been described as abnormalities on the abaxial surface or basilar region of the bone with increased radiodense buildups, increased number and irregularity of the vascular channels, and increased coarseness and mottling of the bone trabeculation (Figure 6.30).
- The increased vascular channels can be confused with a fracture; however, a fracture usually extends to the abaxial surface and the vascular channels do not.
- Nuclear scintigraphy can be used to document increased radioactivity in the region of the sesamoid bones.
- Ultrasound of the suspensory ligament and DSLs may not be helpful because many horses have no abnormalities in the acute stage of the disease.

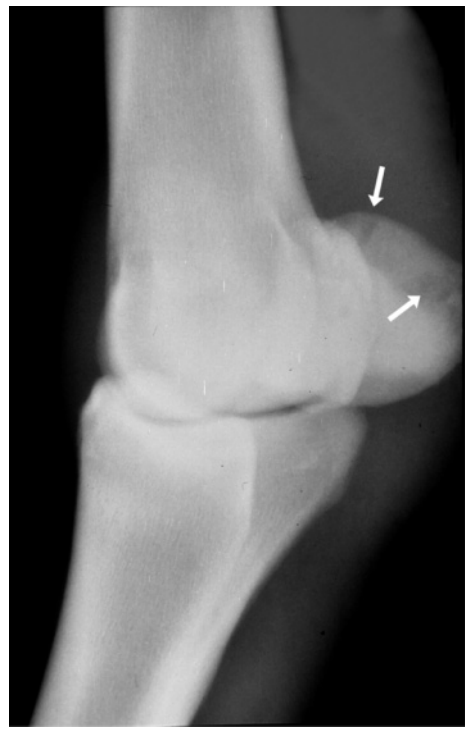


**Figure 6.30.** Typical findings in sesamoiditis. Note the osteolysis along the vascular channel and bone proliferation at the abaxial edge of the lesion (arrows). Courtesy of Alicia Bertone.

### Treatment

- The initial treatment in acute cases is focused on reducing the inflammation. Alternating cold and hot packs, as well as antiphlogistic packs, can be used.
- Rest from performance until soundness at the trot is achieved followed by slow convalescent exercise will permit the bone to continue to remodel and strengthen. Importantly, the exercise must be kept below the level that would re-injure the bones.
- Similar to other suspensory ligament injuries, convalescence is long (six to eight months) and injury often recurs when horses return to full work.
- Firing and blistering and shock wave therapy have been used in chronic cases with undetermined success.

### Prognosis

- The prognosis for return to full athletic performance is usually guarded to unfavorable.

- The prognosis often depends on the amount of periosteal reaction and new bone growth that occurs on the sesamoid bones as well as the extent of the injury to the suspensory ligament and to the DSLs.

## TRAUMATIC OA OF THE MCP JOINT (OSSELETS)

### *Introduction*

- Traumatic OA of the fetlock includes a diverse collection of pathologic and clinical states that develop after single or repetitive episodes of trauma.
- Predisposing conditions include synovitis (inflammation of the synovial membrane), capsulitis (inflammation of the fibrous joint capsule), sprain (injury of collateral ligaments associated with the joint), IA fractures, and subchondral bone injuries.

### *Etiology*

- Any type of traumatic joint injury can progress to OA within the fetlock.
- Soft tissue injury to the joint commonly occurs in horses in full work and often represents overuse of the joint.
- Joint injury can occur as a sporadic event due to trauma.
- Dorsal P1 fractures and other articular injuries often occur from overextension of the joint at speed.
- Limb conformation may predispose horses to joint abnormalities and eventually OA of the fetlock.

### *Clinical Signs*

- In mild cases, joint effusion, heat, and pain on fetlock flexion are present without lameness.
- In more severe cases, joint pain and effusion persists and lameness worsens with exercise (green osselets).
- In severe cases of OA, lameness can be severe and obvious joint enlargement and stiffening can be present.
- Injury or degeneration of the articular cartilage should be suspected in horses with signs of chronic synovitis with severe lameness.

### *Diagnosis*

- The history and clinical findings often provide tentative diagnosis of a fetlock problem.
- Radiographs can diagnose an underlying cause in many cases such as osteochondral fragments, SCLs, subchondral bone erosion, osteophytosis, or joint space narrowing.
- Radiographic abnormalities can range from negative to obvious signs of OA (Figure 6.31).
- Computed tomography, MRI, and contrast arthrography can offer improved imaging of the articular surface over plain radiography.

### *Treatment*

- Management of fetlock joint OA should address any primary problem, if possible.
- Potential treatments may include rest, physical therapy, bandages, shoeing changes, and systemic and IA medications.
- The fetlock joint is a commonly medicated joint due to its high rotary motion and distal position within the limb. Return to work should be gradual and can be supplemented with systemic joint medications and shoeing alterations to promote break-over of the foot and an easier landing (pads, remove caulks and toe grabs, etc).
- Many jumping horses and racehorses are treated with icing and wrapping of the fetlocks after workouts as a routine.
- Arthroscopic surgery is recommended in horses that have concurrent predisposing conditions that are amenable to surgery. Horses that respond to an IA block but do not respond to rest or medical therapy are often candidates for an exploratory arthroscopy.
- Confining horses with primary chronic OA often worsens their stiffness and discomfort. Regular exercise and turn-out provides the greatest longevity with this condition.

### *Prognosis*

- The prognosis for green osselets is good to excellent. Early recognition and management is key to keeping the joint in good health and continuing training.
- Once degenerative changes in the joint are visible on radiographs, the prognosis is still good with management if the horse is sound enough to perform.
- Once horses are no longer sound with medical management, an extended period of rest may permit some horses to return to training, but at a reduced level.
- Many top equine athletes can continue to perform at some level of activity with fetlock OA.

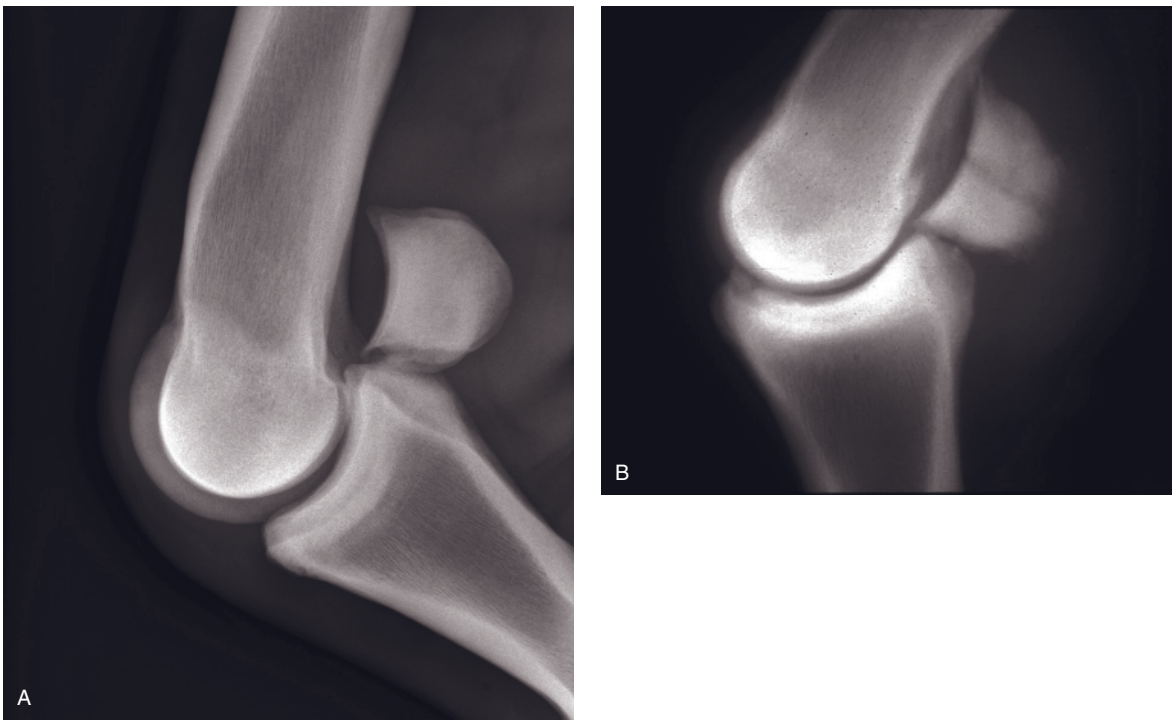



**Figure 6.31.** Lateral radiographs indicative of mild (A) and severe (B) OA of the fetlock joint. The horse in (B) had a chronic midbody sesamoid fracture that was not treated. Secondary OA developed within the fetlock, characterized by lipping of the sesamoid bones and osteophytes on the palmar aspect of MCIII.

## FETLOCK SUBCHONDRAL CYSTIC LESIONS (SCLs)

### *Etiology*

- Subchondral cystic lesions occur most commonly on the weight-bearing surface of the metacarpal condyle and less commonly on the weight-bearing surface of proximal P1.
- MCIII SCLs are usually developmental in origin and occur in young horses (Figure 6.32A).
- SCLs of proximal P1 are more often traumatically induced and can occur in horses of any age (Figure 6.32B).

### *Clinical Signs*

- Moderate lameness is a feature of the disease, together with pain to fetlock flexion and fetlock effusion in approximately 50% of the cases.
- Some horses with P1 SCLs may present with an acute onset of lameness, joint heat and effusion, and severe pain to fetlock flexion.

### *Diagnosis*

- Radiography usually confirms the diagnosis of an SCL. However, it may not be apparent early in the disease process, and follow-up radiographs should be considered.
- SCLs are characterized by defined lucent defects in MCIII (articular) or P1 (may be nonarticular; Figure 6.32).
- Nuclear scintigraphy should be able to identify a focal area of increased uptake in the bone as the SCL is developing and for several months after it has formed.

### *Treatment*

- Surgical debridement of MCIII SCLs by an arthroscopic approach is the preferable treatment if the diagnosis is made before significant signs of OA have developed.
- More conservative treatments such as IA injections, systemic joint medications, and periods of inactivity may be beneficial, depending on the severity of the lameness and the size of the lesion.

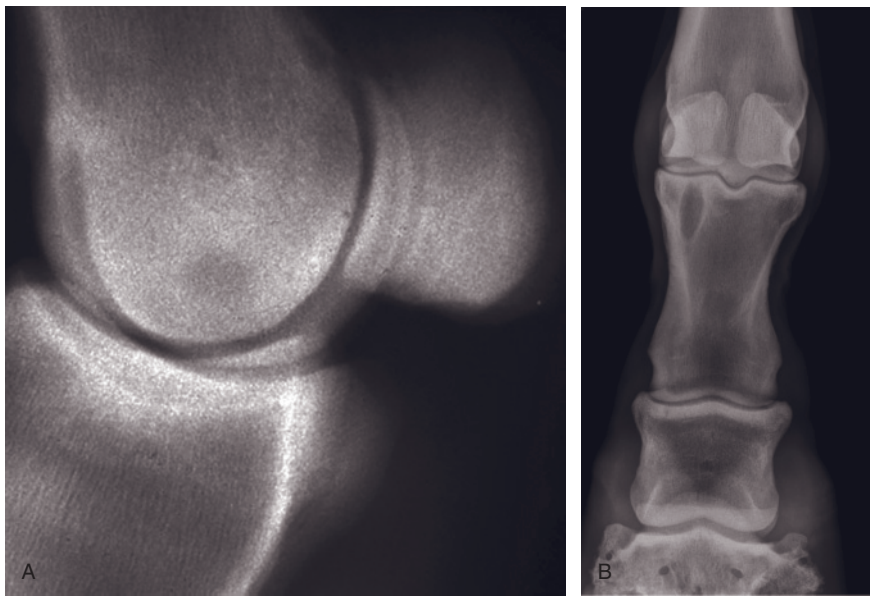



**Figure 6.32.** (A) Lateral fetlock radiograph with a subchondral bone cyst in the middle of the condyle. (B) Dorsopalmar fetlock radiograph with a SCL of proximal P1. Courtesy of Alicia Bertone.

- Proximal P1 cysts that cannot be accessed with an articular approach can be surgically debrided from an extra-articular approach. A small 2.7-mm drill bit is directed to enter the cyst from the dorsal surface of P1. Radiographic or fluoroscopic control is required to ensure entrance into the cyst and avoidance of the joint. The cyst can be decompressed and debrided from this approach.
- Use of postoperative systemic and follow-up intra-articular medications to support joint healing is indicated.

### *Prognosis*

- The prognosis for return to performance appears to be good. In one report, 80% of horses (12 of 15 horses) with MCIII SCLs treated surgically returned to their intended use.
- The prognosis for SCL of proximal P1 is less favorable than lesions of MCIII, particularly if they are traumatically induced.

## TRAUMATIC RUPTURE OF THE SUSPENSORY APPARATUS

### *Introduction*

- Traumatic rupture of the suspensory apparatus with or without fractures of both proximal sesamoid bones is a common cause of acute breakdown in the racing Thoroughbred (Figure 6.33).
- Proximal luxations of the sesamoid bone without fracture also can occur with traumatic rupture of the distal sesamoidean ligament (Figure 6.33A).
- Occasionally, open luxation of the fetlock joint can occur.
- Besides the severe trauma sustained by the supporting soft tissues and bone, the adjacent vasculature may be damaged sufficiently to result in ischemic necrosis of the hoof.

### *Etiology*

- Extreme overextension of the fetlock is the likely cause for disruption of the suspensory apparatus.
- Pre-existing pathology of the bones or suspensory ligament may contribute to breakdown.
- Factors that increase the strain on the flexor surface of the limb, such as toe grabs, may increase the risk of suspensory apparatus failure.

### *Clinical Signs*

- The horse is usually completely non-weight-bearing and the fetlock will sink to the ground when the limb is weighted.

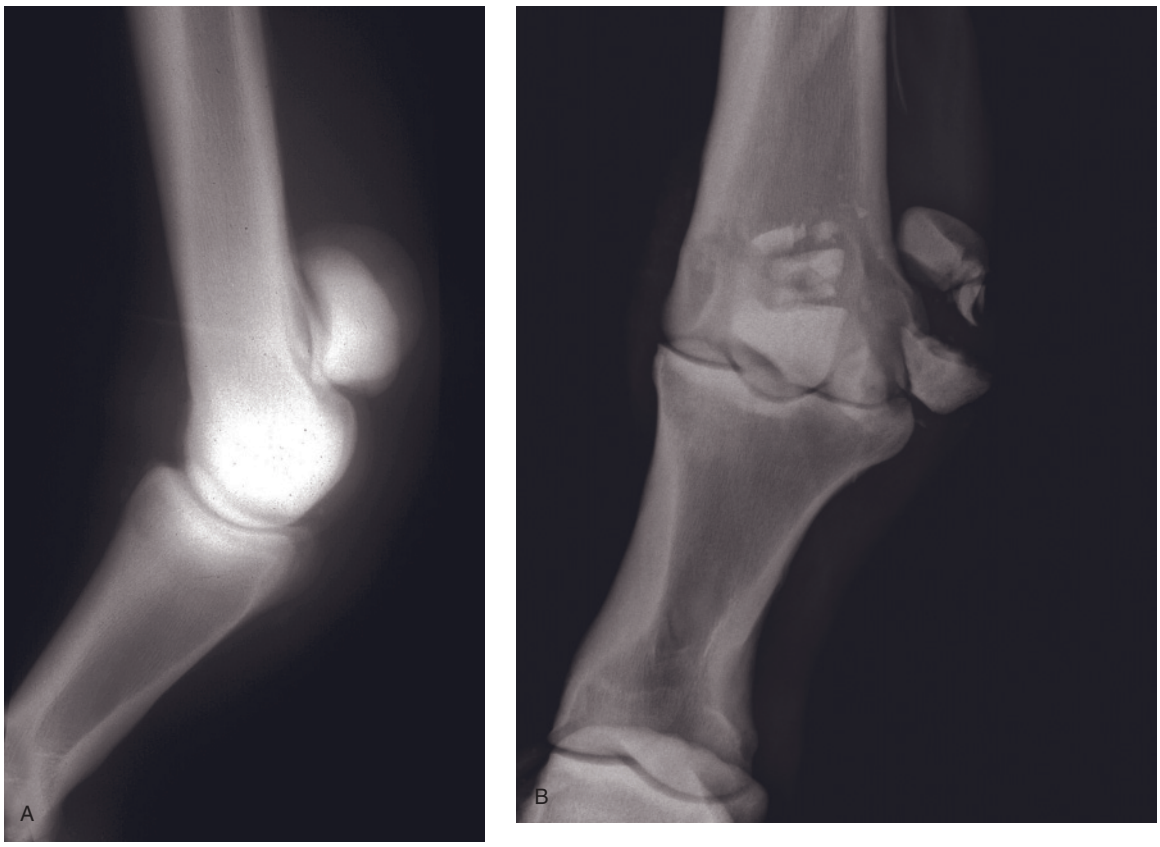



**Figure 6.33.** (A) Lateral radiograph of a horse with disruption of the distal suspensory apparatus that has permitted the sesamoid bones to luxate proximally, and (B) oblique radiograph of the MCP joint illustrating a suspensory ligament breakdown injury where comminuted fractures of both proximal sesamoid bones have occurred. Image (B) courtesy of Robert Hunt.

- The fetlock region is usually very swollen and palpation may reveal the proximal displacement of either the intact sesamoid bone or the apical fractured fragment.
- Immediate stabilization of the limb is critical to prevent rupture of the neurovascular bundles.

### *Diagnosis*

- Radiographic examination usually reveals either the proximal displacement of the intact sesamoid bone or proximal displacement of the apical portions of the fractured sesamoid bones (Figure 6.33).
- Associated swelling of the soft tissues is usually quite evident, and pre-existing degenerative lesions within the sesamoid bones and fetlock joint also may be present.

### *Treatment*

- Treatment options include humane euthanasia or surgical arthrodesis of the fetlock.
- Casting and splinting to promote fetlock ankylosis is not currently recommended because it is rarely successful.
- Treatment should be considered for horses that are intended for breeding or when there is sentimental value, because a normal gait will not be achieved.
- Management of the horse on the racetrack is critical for success in treatment. Immediate immobilization of the affected limb is required to decrease the chances of further injury to the soft tissue and vascular supply (Figure 6.34).
- Arthrodesis with implants and bone graft can be used to achieve a pain-free stable fusion of the fetlock joint if the soft tissues are intact and risk of infection is minimal (Figure 6.35).

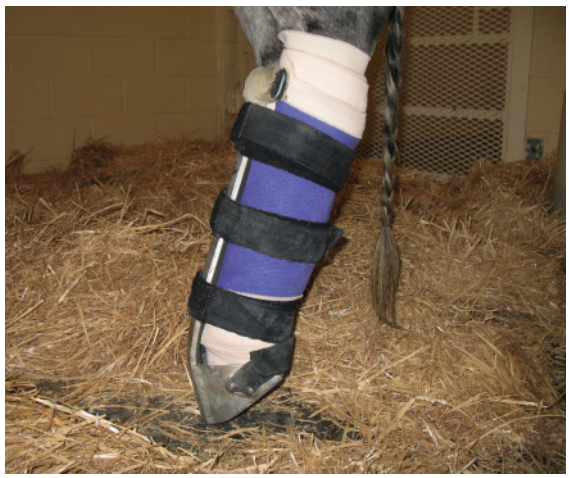


**Figure 6.34.** Immobilization of the limb in a Kimzey splint, which is designed for breakdown injuries, including severe sesamoid fractures.

### *Prognosis*

- The prognosis appears to be good for pasture and breeding soundness with preselection of cases. In one study, 32 of 54 horses with arthrodesis of the fetlock survived and were eventually allowed unrestricted activity.
- The prognosis is better for horses in which fetlock arthrodesis was elected as the primary treatment rather than as a last resort, and was better for horses in which fetlock fusion was elected for OA rather than rupture of the suspensory apparatus.
- Contralateral limb laminitis is one of the major complications of surgical arthrodesis of the fetlock.

## DIGITAL FLEXOR TENDON SHEATH (DFTS) TENOSYNOVITIS

### *Introduction*

- Digital flexor tendon sheath (DFTS) tenosynovitis is often characterized by effusion within the sheath, with or without clinical signs of pain.
- Effusion within the DFTS without lameness or other clinical signs is often referred to as "windpuffs."
- DFTS effusion associated with lameness can be due to several disease conditions of the SDFT, DDFT, annular ligament, and the sheath itself, such as previous infection.
- DFTS tenosynovitis appears to be more common in the hindlimb than the forelimb.

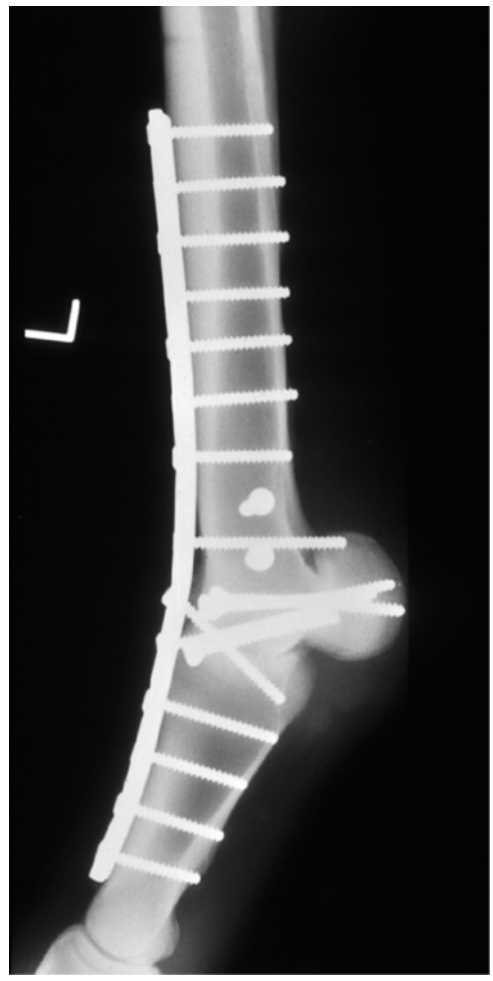


**Figure 6.35.** Surgical arthrodesis of a fetlock joint with plates and screws to treat a breakdown injury. Courtesy of Larry R. Bramlage.

### *Etiology*

- The cause of windpuffs is unknown but may represent an early wear-and-tear phenomenon.
- DFTS tenosynovitis associated with lameness or marked swelling of the sheath is usually secondary to trauma to the flexor tendons, proximal sesamoid bones, intersesamoidean ligament, annular ligament, or the DFTS itself.
- Tendinous defects within the sheath can involve the DDFT (longitudinal tears), the SDFT, and the manica flexoria of the SDFT.
- Septic tenosynovitis must always be considered, particularly if puncture wounds are present or there is a history of a previous injection.

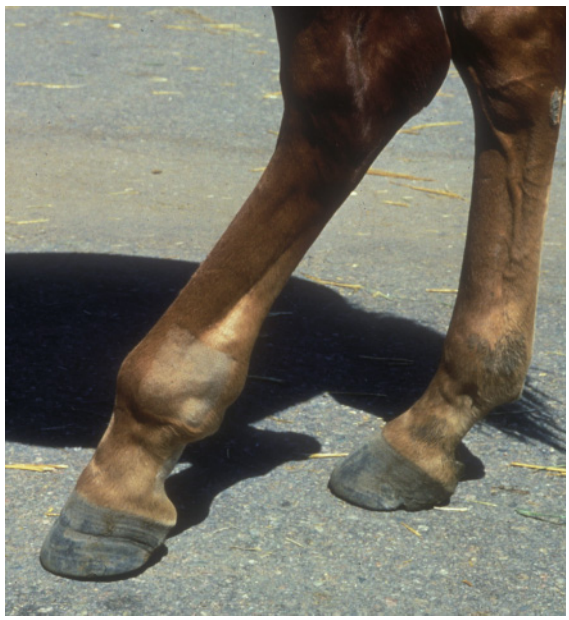


**Figure 6.36.** This horse had swelling of the digital flexor tendon sheath and evidence of constriction of the fetlock anular ligament of the left hindlimb. Courtesy of Alicia Bertone.

- With chronic tenosynovitis, permanent swelling and fibrosis of the tendons, the annular ligament, and the lining of the sheath can lead to stenosis of the fetlock canal. Adhesions and synovial masses can form within the sheath.

### *Clinical Signs*

- Effusion of the DFTS is usually visible and palpable palmar/plantar to the suspensory ligament and proximal to the sesamoid bones (Figures 6.21, 6.36).
- Effusion also may be palpable in the midpastern region at the distal most aspect of the DFTS where it becomes very superficial surrounding the DDFT.
- The most notable feature of annular ligament constriction is swelling of the palmar/plantar soft tissues around the fetlock together with a visible proximal border of the annular ligament ("notching"; Figure 6.36).
- Lameness is usually persistent, worsens with exercise, and is characterized by a decreased extension (dorsiflexion) of the fetlock during weigh-bearing. Usually, pain can be elicited with deep palpation of the DFTS and with fetlock flexion.

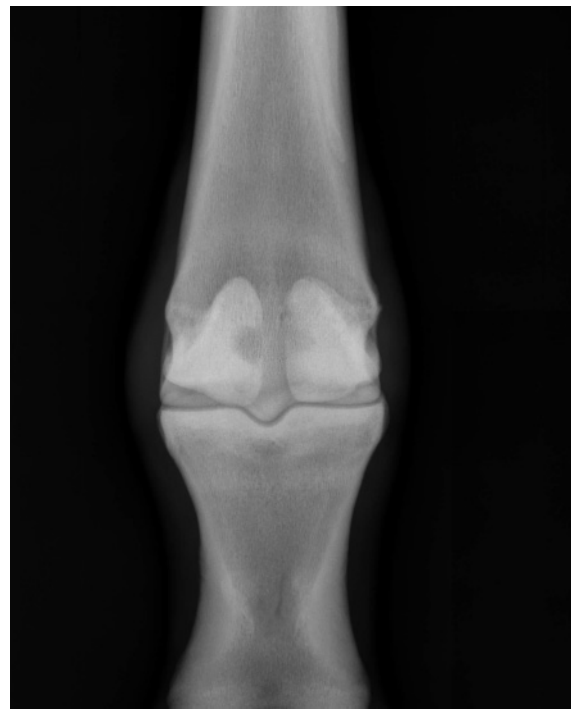


**Figure 6.37.** Dorsopalmar radiograph of the fetlock illustrating lysis of the axial borders of the sesamoids consistent with osteomyelitis. Courtesy of Ellis Farstvedt.

- Usually, acute septic tenosynovitis is characterized by a sudden onset of mild to severe lameness associated with warm, painful effusion of the DFTS.

### *Diagnosis*

- Ultrasonography is usually the most important diagnostic tool to identify abnormalities within the sheath. Lesions identified at ultrasound vary and can include core tendon lesions or longitudinal tearing of the DDFT and SDFT, fibrosis and thickening of the tendons, synovial proliferation, adhesions or masses, and thickening of the annular ligament. Longitudinal tears of the DDFT can be difficult to diagnose with ultrasound (Figure 6.22).
- Radiographs are most important in septic tenosynovitis to identify lysis of the sesamoid bones or the intersesamoidean area and dystrophic mineralization that can occur with chronic infection (Figure 6.37).
- Intrasynovial anesthesia of the DFTS is usually the best method of diagnostic anesthesia. Centesis of the DFTS can be performed at the same time in suspected cases of infection.

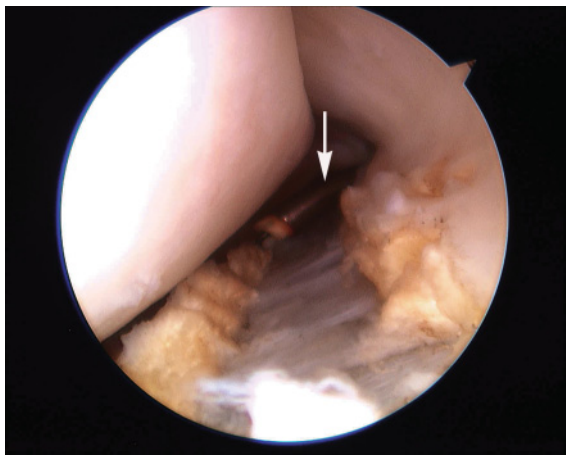


**Figure 6.38.** Tenoscopic view within the DFTS following transection of the annular ligament with a curved radiofrequency probe (arrow).

- MRI of the fetlock region can reveal abnormalities in the soft tissues not identified by ultrasound.

### *Treatment*

- Medical management of DFTS tenosynovitis is similar to that of joint synovitis/OA. Treatments include rest, cold hydrotherapy, bandaging, topical and systemic NSAID medication, and intrasynovial medication of the sheath.
- Tenoscopy of the DFTS is currently the preferred treatment method to address synovial masses and tendon lesions, and to perform annular ligament transection if indicated (Figure 6.38).
- If tenoscopy is not an option and annular ligament constriction is present, the annular ligament can be transected percutaneously through a small incision directly over the abaxial surface of the lateral sesamoid bone.
- Septic tenosynovitis should be treated with tenoscopic lavage; debridement; and systemic, intrasynovial, and IV regional antimicrobials.

### *Prognosis*

- The prognosis for soundness in horses with DFTS tenosynovitis is considered to be good, with 68% returning to soundness and 54% returning to levels of previous work.
- Horses with concurrent SDFT or DDFT lesions and those with septic tenosynovitis have a reduced prognosis.
- Persistent effusion of the DFTS is not uncommon after treatment.

## PERIOSTITIS AND FRACTURE OF THE DORSAL METACARPUS (BUCKED SHINS, SHIN SPLINTS, AND STRESS FRACTURE)

### *Introduction*

- Periostitis and stress fracture of the dorsal surface of MCIII constitute a spectrum of diseases that are commonly observed in young (2 to 3 years of age) fast gaited horses.
- Dorsal metacarpal bone failure results in a fracture of the dorsal or dorsolateral cortex of MCIII. It is usually observed in slightly older horses (3 to 5 years of age).
- It is most common in young racing Thoroughbreds, but can also occur in young Quarter horses and racing Standardbreds.

### *Etiology*

- The MCIII in young horses (2-year-olds) is less stiff, and therefore greater strains (bone movement) are measured on the dorsal cortex during high-speed exercise as compared to older horses. These high strains can induce low cyclic fatigue of bone, resulting in microdamage or ultimate bone failure. The body responds with bone modeling, but microfracture damage may develop and cause pain.
- The majority (more than 80%) of 2-year-old racing Thoroughbreds and many racing Quarter horses demonstrate dorsal cortical pain.
- It is estimated that approximately 12% develop acute failure or dorsal cortical fracture, usually within 6 months to one year of showing dorsal cortical pain.
- The incidence of fatigue failure of MCIII is greater in Thoroughbreds than Standardbreds, presumably due to different stresses on the bone during training and racing.

### *Clinical Signs*

- Early dorsal metacarpal disease usually has an acute onset and is most obvious after intense exercise.
- A visible convex swelling overlying the surface of the affected portion of the cannon bone is common.
- The dorsal cortex of MCIII is usually painful to palpation.

- With chronic dorsal metacarpal disease, lameness may be mild, but a painful enlargement on the dorsomedial cortex of MCIII is usually present.
- In horses with dorsal cortical fractures, the lameness becomes prominent after strenuous exercise, and a discrete painful area often can be palpated on the dorsolateral surface of the left MCIII at the junction of the middle and distal third.

### *Diagnosis*

- A tentative diagnosis of MCIII periostitis or stress fracture can be made from the signalment of the horse and clinical findings.
- Perineural anesthesia is usually unnecessary; direct infiltration of the painful area only provides partial relief of the lameness.
- Radiographs can usually confirm the diagnosis in cases of chronic periostitis and dorsal cortical fracture. Thickening of the dorsomedial cortex with associated periosteal new bone formation is seen.
- The DPLMO and the LM will best identify the dorsal medial bone proliferation and the DPMLO and LM will best identify the dorsal lateral cortical fractures.
- Fractures usually enter the cortex distally and progress proximad at a 35° to 45° angle. They usually appear as a straight or slightly concave fracture line (tongue fracture); occasionally they exit through the dorsal cortex (saucer fracture) (Figures 4.10, 6.39).
- Repeated radiographs at seven- to 10-day intervals may be necessary to identify a fracture that is suspected but not observed on initial radiographic examination.
- In horses with no radiographic changes, nuclear scintigraphy can provide information about the stage of disease. The sensitivity of this technique allows detection of abnormalities in horses in the acute-subacute stages.

### *Treatment*

- The goal of treatment with acute disease is to gradually increase the stress to the dorsal surface of MCIII at such a rate that this surface can model (form new bone) according to compressive demands without producing structural damage.
- Most horses with dorsal metacarpal disease are removed from training and put on a convalescent exercise program to provide time for the early acute changes to subside. Hand walking, ponying, cold water hosing, or icing and bandaging should continue until the MCIII is free of palpable pain. Initially, daily galloping distance is reduced to 50%. Speed and distance are introduced slowly. If speed is increased, distance should be decreased.
- Subacute and chronic dorsal metacarpal disease can be the most difficult to treat. Many of these horses may not be suitable for the modified training regimen described above and pain will immediately return with any sustained galloping. These horses may have marked periosteal new bone formation and may require more prolonged rest.
- Some dorsal cortical fractures in young horses may resolve with an altered exercise program but may require four to six months for the fracture to heal.
- Surgical treatment of dorsal cortical MCIII fractures include placement of a unicortical lag or positional screw, and dorsal cortical drilling (Figure 6.40). The screw may or may not need to be removed.
- Adjunctive treatments that have been recommended (with or without surgical treatment)

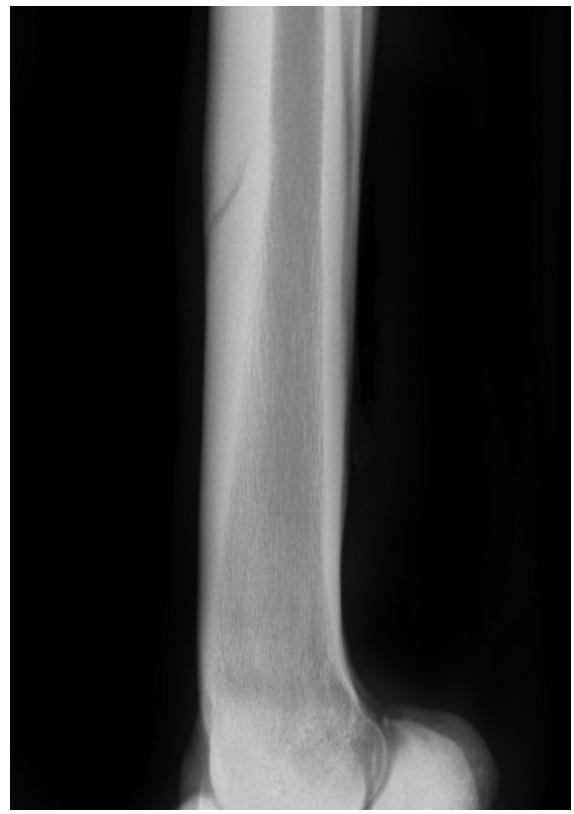


**Figure 6.39.** Lateromedial radiograph showing a dorsal cortical fracture of MCIII. Courtesy of Robert Hunt.

include electrical stimulation, shock wave therapy, thermocautery (pin firing), chemical vesication (blistering), and cryotherapy (point freezing).

### *Prognosis*

- The prognosis is good to excellent (80% to 90%) for return to racing with surgical treatment of dorsal cortical fractures.

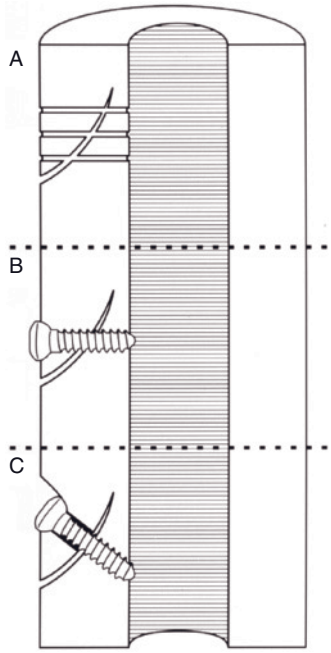



**Figure 6.40.** (A) Dorsal cortical drilling (osteostixis) of a dorsal metacarpal stress fracture. (B) Positional unicortical screw placement. (C) Unicortical screw placement in lag fashion.

- Horses with acute periostitis given adequate time to convalesce also have a very good prognosis.
- Adjustment of training regimens may assist with prevention of this problem, and training on grass, wood fiber, or softer surfaces without toe grabs is recommended.

## MCIII/MTIII CONDYLAR FRACTURES

### *Introduction*

- Fractures of the condyles of MCIII/MTIII occur most frequently in racing Thoroughbreds, less frequently in Standardbreds, and occasionally in Quarter horses and Polo ponies. They comprise a subset of catastrophic breakdowns in racehorses.
- The distribution of fractures is approximately one-third incomplete-nondisplaced, one-third complete-nondisplaced, and one-third complete-displaced (Figure 6.41).
- In Thoroughbreds, the lateral condyles of the either forelimb are most commonly involved.
- Articular comminution can occur, usually at the palmar/plantar articular margin, in about 15% of fractures.
- Concurrent axial sesamoid fractures are associated with displaced lateral condylar fractures that disrupt the collateral ligament and avulse the intersesamoidean ligament complex.
- Condylar fractures of the hindlimb are more common in Standardbreds and are more likely to be medial and to not exit the cortex. These fractures can propagate proximally or progress to a complete "Y" fracture, even with stall confinement.

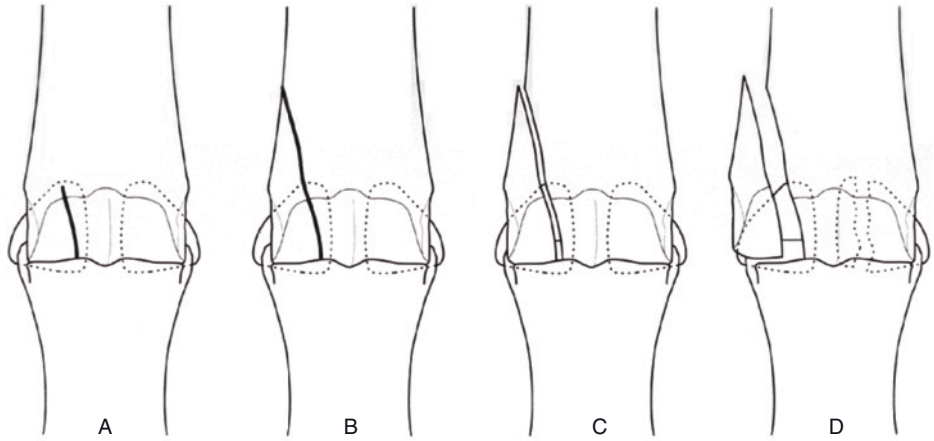



**Figure 6.41.** Condylar fractures. (A) Incomplete, (B) complete nondisplaced, (C) complete separated, (D) complete displaced.

### Etiology

- The etiology is trauma from high compressive loads, asynchronous longitudinal rotation of the cannon bone, and exercise on uneven surfaces.
- The risk of fatal condylar fractures in Thoroughbred racehorses is seven times and 17 times more likely if horses are shod with low or regular toe grabs, respectively. The toe grab changes the hoof angle and presumably places more stress on the suspensory apparatus and plants the foot more securely. This likely alters the compressive and rotatory forces on the distal MCIII.
- The palmar MCIII condyle is the site of maximum loading in racing and the bone responds by increasing density (sclerosis). Bone fatigue failure occurs, and due to the normal columnar arrangement of the bone trabeculae, acute failure of the bone propagates in the configuration seen in condylar fractures.

### Clinical Signs

- The clinical signs may vary from a mild lameness that is exacerbated by exercise with little heat or swelling with nondisplaced, incomplete fractures, to severe lameness with heat, pain, and swelling in the acute displaced fracture.
- Incomplete nondisplaced fractures often have very subtle physical examination findings with minimal lameness.
- Nearly all horses have fetlock effusion because all fractures originate at the articular surface, and most horses will be positive to fetlock flexion and rotation.
- Radiographs should be taken immediately if a fracture is suspected based on history, clinical signs, and joint effusion.

### Diagnosis

- Radiographs usually confirm the diagnosis. The exception may be very short, incomplete, nondisplaced fractures that may be difficult to detect.
- Displaced fractures are obvious on the radiographs as longitudinal lines propagating up MCIII/MTIII and exiting the lateral cortex (Figure 6.42).
- A 125° caudocranial view to isolate the palmar/plantar surface of the condyle is important to check for articular comminution.

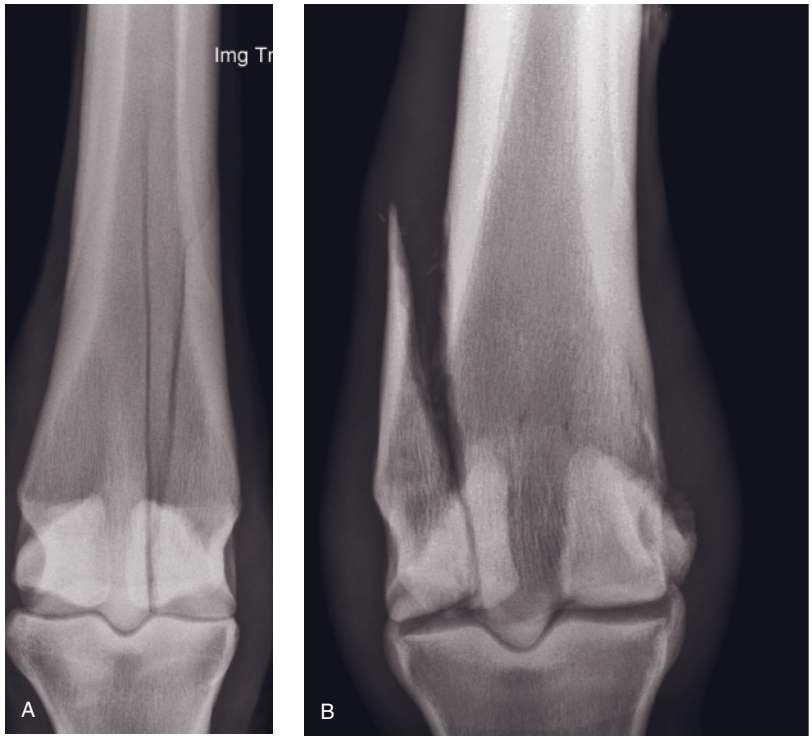



**Figure 6.42.** Radiographs of a spiraling medial condylar fracture in a racing Standardbred (A) and a displaced lateral condylar fracture in a racing Thoroughbred (B). Courtesy of Alicia Bertone.

- Lesions that have been associated with condylar fractures include proximal fractures of P1, fractures of the proximal sesamoid bone, OA of the fetlock, palmar/plantar erosive lesions of the distal MCIII/MTIII, suspensory ligament desmitis, and longitudinal fractures of MTIII.
- In horses without radiographic abnormalities, nuclear scintigraphy can detect bone damage prior to fracture or a potential fracture that was missed on the initial films.
- Computed tomography can be performed in fractures that spiral up the MCIII/MTIII to further define the fracture propagation.

### *Treatment*

- The recommended treatment of most condylar fractures is internal fixation with transcortical lag screws.
- Arthroscopic evaluation of the articular alignment can be helpful in displaced fractures, particularly if other bone fragments need to be removed, such as P1 eminence fractures or comminution of the palmar fracture line.
- Incomplete, nondisplaced fractures can be treated conservatively with successful return to racing, and surgery is not always required.
- Lag screw fixation offers the advantage of preventing fracture displacement, shorter convalescence, a reduced incidence of refracture at the same site, and primary bone healing which decreases the risk of OA within the fetlock.
- Horses with condylar fractures that spiral proximally (typically the medial MTIII) can be repaired with lag screws in the standing, sedated horse or with plates and screws or screws alone under general anesthesia.

### *Prognosis*

- General prognosis for athletic performance and returning to racing is excellent for nondisplaced incomplete fractures, whether treated conservatively or following internal fixation.
- Prognosis for athletic performance and returning to racing is fair for complete displaced and nondisplaced fractures following internal fixation.
- The prognosis for return to racing is considered poor for comminuted fractures or subchondral erosive lesions in the palmar/plantar surface of the distal MCIII/MTIII.
- The prognosis to return to racing in Thoroughbreds is significantly reduced with complete fractures, forelimb fractures, or evidence of sesamoid fracture, or if the horse is female (presumed retired for breeding).

## COMPLETE FRACTURES OF THE MCIII/MTIII (CANNON BONE)

### *Introduction*

- Fractures of the cannon bone can occur in any age or breed of horse but are most common in younger animals.
- Younger horses sustain simpler fractures than adults, possibly because of more elastic, less brittle bone. The fracture can occur anywhere along the bone length and can enter either the proximal or distal joint.
- Because of the minimal soft tissue around the cannon bone, the fractures are commonly open or become open after injury occurs, and more than half of referred MCIII/MTIII fractures are open.
- Stress fractures of MCIII/MTIII in racehorses can progress to acute and complete failure of the bone.

### *Etiology*

- External trauma in any form is the usual cause of cannon bone fractures. This often include kicks; halter breaking injuries; injuries associated with ground holes, fences, or cattle guards; slipping accidents and accidents associated with moving vehicles.
- When foals are affected, the dam has often stepped on the limb and caused the fracture.

### *Clinical Signs and Diagnosis*

- Complete yet nondisplaced fractures of the cannon bone secondary to direct trauma can occur and may be difficult to diagnose initially (Figure 6.43).
- The cannon bone may be enlarged slightly, and heat, swelling, and pain on deep palpation are usually present.
- With complete fractures, the diagnosis is usually obvious. Horses are often non-weight-bearing and the limb has an abnormal angulation.
- Approximately 50% of cannon bone fractures are open.

### *Diagnosis*

- Usually, the diagnosis can be made based on physical examination of the limb with complete fractures.

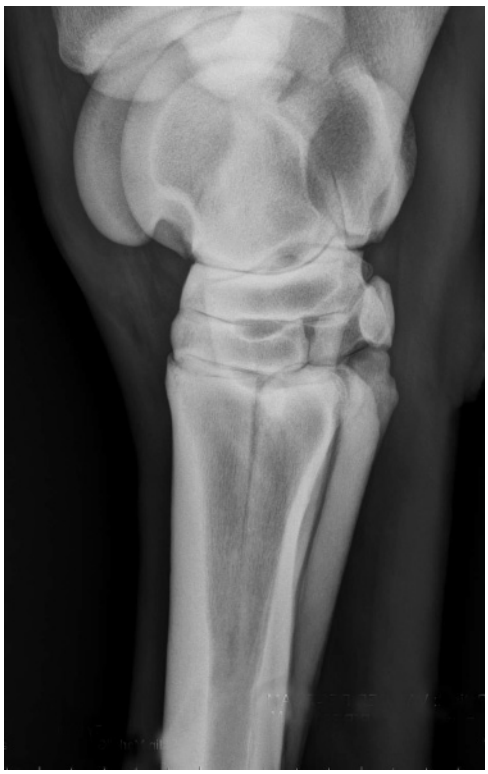


**Figure 6.43.** This oblique radiograph of the tarsus revealed an incomplete proximal metatarsal fracture. The fracture occurred from an accident at a jump and was initially thought to be a proximal suspensory injury. Courtesy of Ty Wallis.

- Radiographs usually confirm the diagnosis. A complete set of radiographs should be taken in nondisplaced fractures to help identify the fracture lines and in horses in which surgery may be considered (Figure 6.44).

### *Treatment*

- The selection of treatment of cannon bone fractures depends on the type of fracture (open vs. closed, simple vs. comminuted), the location of the fracture (articular vs. nonarticular, proximal vs. distal), the animal's age, its intended use, the presence of wounds, vascular compromise, and the economics.
- The preferred treatment for most cannon bone fractures is internal fixation with one or two DCP or LCP plates (Figure 6.44), or minimally invasive plate fixation extending the length of the bone, combined with individual screws where appropriate.
- Severely comminuted fractures may heal with transfixation pins and external fixators or casts. These methods are more successful in foals with rapid healing and low body weight.

### *Prognosis*

- In general, transverse, slightly oblique, and mildly comminuted (one butterfly fragment) fractures in the midcannon bone region in foals under 7 months of age have a good to excellent prognosis with internal fixation (Figure 6.44).
- Older horses with similar fractures have a more guarded prognosis due to their size and the risk of complications.
- Older horses with open, comminuted, or articular fractures have a guarded to poor prognosis for recovery.

## "SPLINTS" OR SMALL MC BONE EXOSTOSIS

- "Splints" is a condition of young horses that most commonly affects the proximal medial aspect of the limb between the medial splint bone and MCIII (Figure 6.45).
- A true splint refers to a sprain or tear of the interosseous ligament. The resultant enlargement is most frequently observed 6 to 7 cm below the carpus on the medial aspect of the cannon bone.
- The condition is most common in young (2-year-old) horses undergoing heavy training, but can affect horses of any age.

### *Etiology*

- The condition is associated with tearing of the interosseous ligament between the splint bones and MCIII (true splints), external trauma such as kicks or interference, or healing of a transverse or longitudinal fracture. Initially, a desmitis and periostitis occurs, with subsequent fibrous tissue enlargement and ossification of the splint bone.
- Excessive loading of MCII related to conformational abnormalities such as "bench knees" can contribute to tearing of the interosseous ligament.
- The lateral splint bones are most likely traumatized from external blows (hitting objects or being kicked).

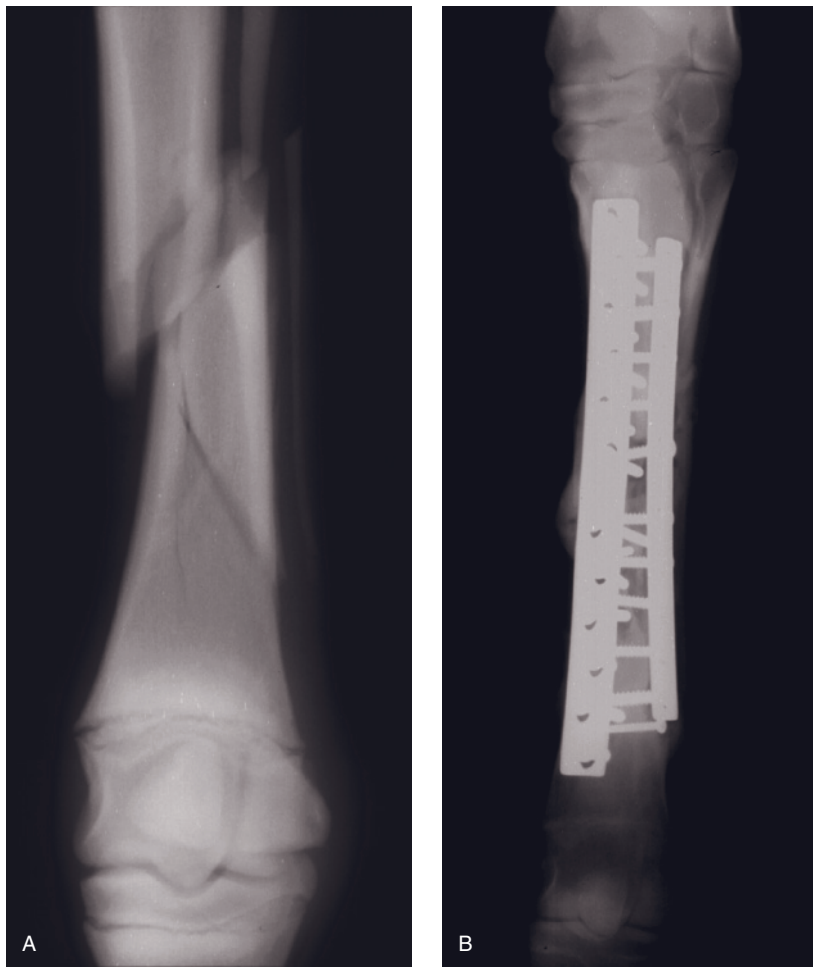



**Figure 6.44.** (A) Open comminuted mid-diaphyseal fracture of the metatarsus in a 7-month-old weanling. (B) Postoperative view demonstrating fracture repair with two broad dynamic compression plates on the dorsolateral and dorsomedial sides of the metatarsus. Additional screws were placed in lag fashion from outside the plates to secure the butterfly fragments.

### *Clinical Signs*

- Acute splints are characterized by heat, pain, and swelling in the proximal, medial cannon bone area. With time, the inflammation subsides and the resultant exostosis is much smaller than the initial swelling.
- Mild to moderate lameness is usually present and most obvious at the trot on hard surfaces.
- Incidental findings of chronic exostosis of the splint bones may occur with no evidence of palpable pain or lameness. However, extensive new bone formation may encroach on the suspensory ligament and cause chronic lameness.

### *Diagnosis*

- A tentative diagnosis usually can be made based on physical examination findings.
- Radiographs are necessary to confirm the diagnosis. A periosteal reaction associated with the splint bone is usually present (Figure 6.46). The horse should be examined for fracture of the splint bone because it can be confused with splints.
- Ultrasonography is usually unnecessary to diagnose splints, but may be helpful to demonstrate concomitant injury to the suspensory ligament.

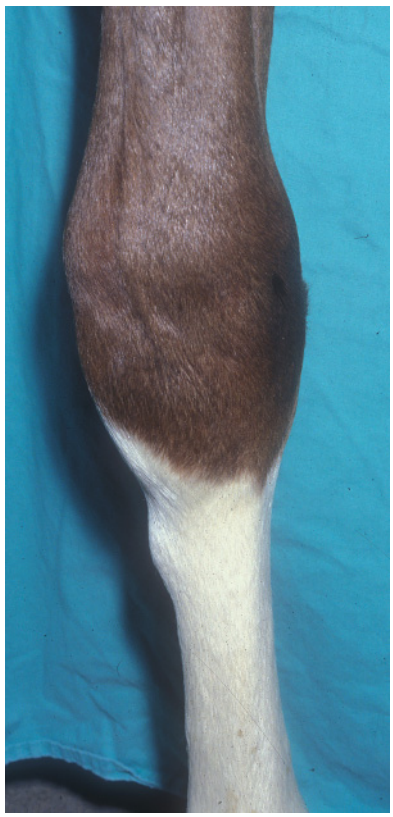


**Figure 6.45.** Visible enlargement of the medial splint area just distal to the carpus, typical of horses with "splints." Courtesy of Alicia Bertone.

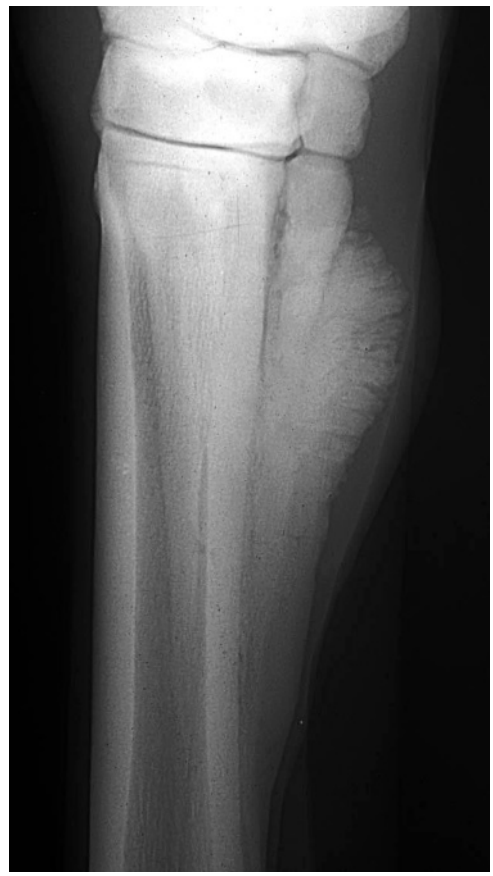


**Figure 6.46.** This large exostosis of the medial splint contributed to lameness and was removed surgically. Just the exostosis was removed and the underlying splint bone was left intact.

- Nuclear scintigraphy can be used to confirm splint bone disease if no radiographic abnormalities are present.

### *Treatment*

- NSAIDs combined with the application of hypothermia, pressure support wraps, topical diclofenac liposomal cream (Surpass®), and rest appear to be most beneficial to decrease the heat, pain, and swelling in acute cases.
- Intralesional corticosteroid in the acute stage may reduce inflammation and prevent excessive bone growth.
- Other treatments for more chronic disease include shock wave therapy and counterirritation.
- Surgical removal of the exostosis with or without the splint bone may be necessary if the exostosis causes lameness or for cosmetic reasons (Figure 6.46).

### *Prognosis*

- Prognosis is very good to excellent for horses with routine splint exostoses. Surgery is usually unnecessary.
- The prognosis for surgical removal of a splint exostosis is also very good, but recurrence is possible, depending on the location of the injury.

## FRACTURES OF THE SMALL MC/MT (SPLINT) BONES

### *Introduction*

- Fractures of the splint bones can occur anywhere along their length but are most common in the distal one-third.

- Fractures of the middle and proximal aspects are usually due to external trauma and are often open and complicated by comminution, osteomyelitis, and bone sequestration.
- Fractures of the distal splint bone are usually due to internal forces associated with exercise, and can be associated with suspensory ligament desmitis, sesamoiditis, and fetlock OA.

### *Etiology*

- Distal fractures are usually due to internal trauma from increased axial compression forces on the splint bones during exercise or increased tension on the bones from fascial attachments. However, they can occur from external trauma such as interference, kicks, and direct blows from hitting another object.
- Fractures of the middle and proximal aspects of splint bones are usually due to external trauma such as kicks and may be associated with a wound.
- Proximal fractures of MCII may be due to excessive torsional forces that may occur in the starting gate in racehorses.

### *Clinical Signs*

- With distal fractures, swelling of the distal cannon bone region and pain on palpation over the fracture are usually present. Lameness can be variable and perineural anesthesia may be indicated to isolate the site of the lameness.
- Horses with closed middle and proximal fractures usually present with variable severity of lameness and swelling in the cannon bone area. Focal pain, heat, and swelling can usually be palpated over the fracture site. A firm exostosis may be palpable, depending on the duration of the injury.
- Horses with open middle and proximal fractures usually present with a wound or draining tract and severe swelling in the cannon bone area. Bone fragments may be palpable within the wound and copious purulent drainage is often seen with sequestration and osteomyelitis.

### *Diagnosis*

- A fractured splint bone is confirmed with radiography in most cases. Oblique views should be taken to isolate the splint bones from superimposition with MCIII/MTIII (Figure 6.47).

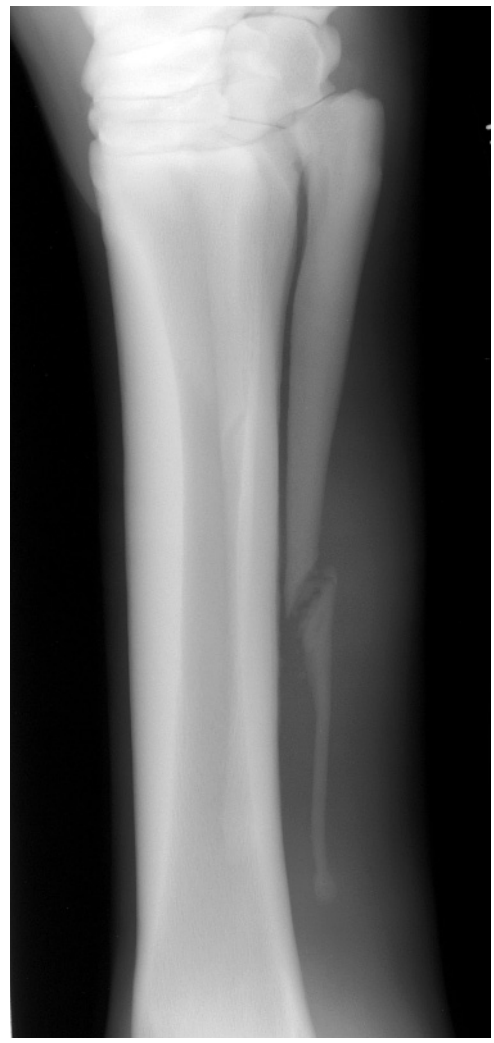


**Figure 6.47.** Fractures of the distal splint bones such as this rarely heal and are usually removed.

- Many fractures are obvious on radiographs, but some nondisplaced fractures can be difficult to identify and resemble a splint exostosis in the chronic stages.
- Radiographic abnormalities may include a fracture, periosteal proliferation, sequestration, and osteomyelitis in complicated fractures. Proximal fractures may extend toward or into the carpometacarpal or tarsometatarsal joint (Figure 6.48).

### *Treatment*

- Small distal fractures of the splint bones are traditionally treated by surgical removal, but

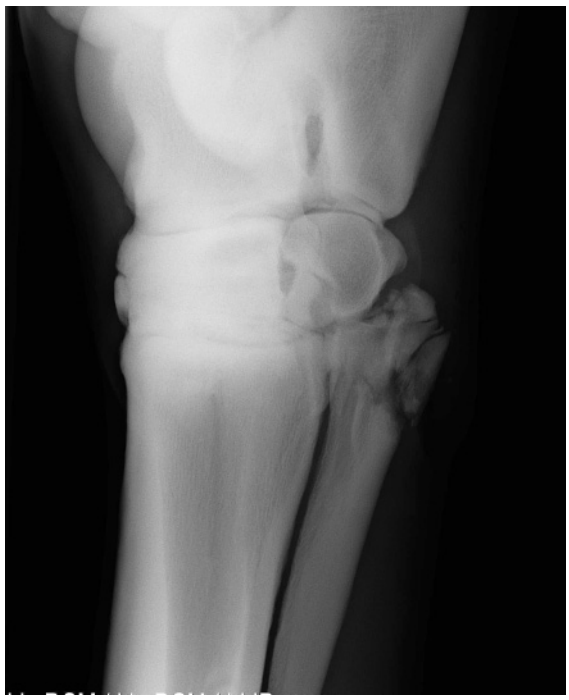


**Figure 6.48.** Proximal fractures of the fourth metatarsal bone are often comminuted and open and are due to traumatic injuries. This fracture also entered the TMT joint.

this may be unnecessary if the fracture does not cause swelling or lameness.
- Closed middle fractures also can heal without treatment, but may develop significant callus at the fracture site that may later contribute to lameness.
- Open middle fractures of the splint bone often lead to draining tracts or sequestra; open removal of the fracture with or without the remaining distal splint bone is the recommended treatment. Up to 80% of the entire splint bone can be removed without complications.
- Closed nonarticular and nondisplaced proximal comminuted fractures of the splint bones usually heal without surgical treatment. However, avulsion of the proximal fragments is much more likely to occur fractures of MCII and MCIV than MTII and MTIV.
- Closed articular fractures of MCII and MCIV are usually best treated with internal fixation using a small bone plate to prevent displacement of the proximal fragment (Figure 6.49).
- Open proximal fractures of the splint bones are the most difficult to treat. Many will heal with conservative treatment if no infection develops. Wound lavage and debridement and local and systemic antimicrobials should be performed to prevent complications.
- Infected, proximal MTIV fractures can be treated with complete removal of the bone but only if other treatments have failed (Figure 6.50).

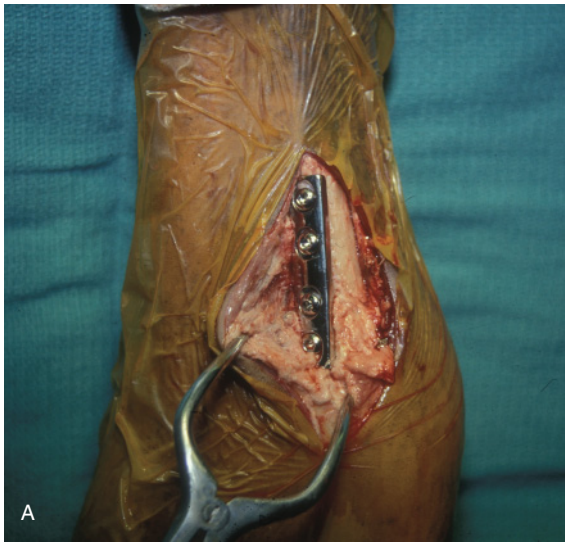
### *Prognosis*

- The prognosis for most horses with open or closed distal splint bone fractures is usually very good, and is often more dependent on concurrent musculoskeletal problems such as suspensory desmitis than the fracture itself.
- The prognosis for horses with open or closed middle splint bone fractures is also good to excellent with or without surgery if approximately one-third of the proximal splint bone remains.
- The prognosis for horses with open comminuted fractures of the proximal splint bones is more guarded and is usually determined on a case-by-case basis.

## SUSPENSORY LIGAMENT (SL) DESMITIS

### *Introduction*

- The SL originates on the proximal palmar/plantar surface of MCIII/MTIII, divides into two branches in the distal cannon bone region, and inserts on the proximal medial and lateral sesamoid bones (Figure 6.51).
- Injuries to the SL are usually based on location: proximal (proximal suspensory desmitis), middle (body lesions), and distal (branch lesions).
- Proximal suspensory desmitis (PSD) is a common cause of soft tissue injury in sport horses such as event horses and jumpers and Western performance horses. The hindlimbs are more frequently affected than the forelimbs.
- Injury to the body of the SL is less common than PSD and occurs most frequently in Standardbred racehorses. Degenerative suspensory ligament desmitis (DSLD) also usually affects the body of the SL.
- Injury to the SL branches occurs most commonly in Standardbred racehorses or jumping horses.
- The presence of upright hindlimb (straight hock) conformation is overrepresented in horses with hindlimb suspensory injury and



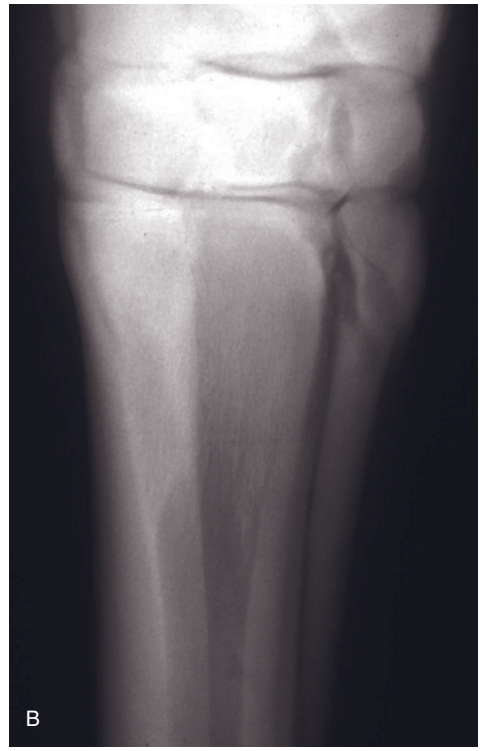




**Figure 6.49.** Plate fixation of a proximal MCII fracture (A) in a Quarter horse racehorse that was performed to prevent avulsion of the proximal fragment (B).

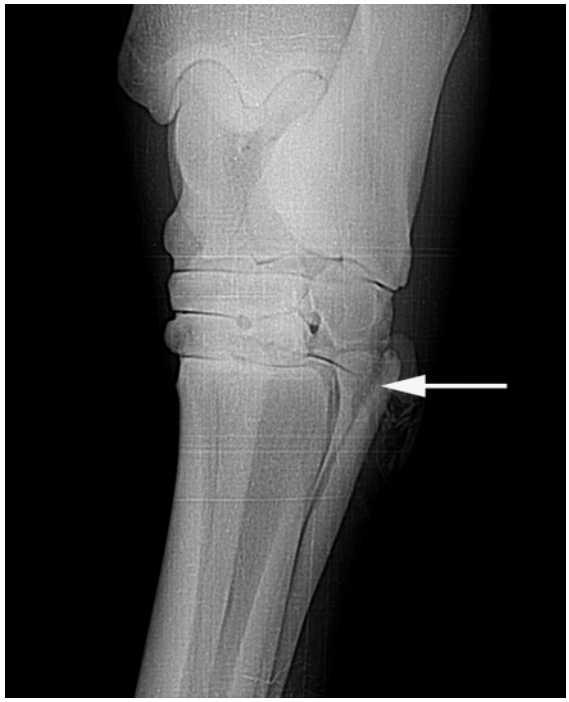


**Figure 6.50.** Open, articular, comminuted proximal fourth metatarsal fracture (arrow) that was initially debrided and treated conservatively. Infection persisted and the entire MTIV was removed.

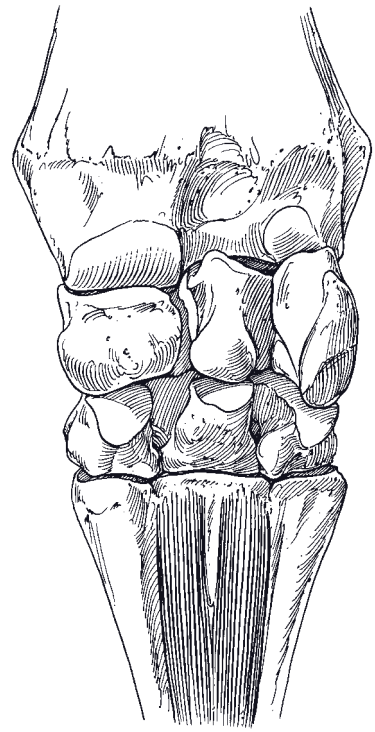


**Figure 6.51.** The attachments of the suspensory ligament at the proximal palmar surface of the third metacarpal bone. Courtesy of TS Stashak.

may predispose the ligament to injury and recurrence.

### *Etiology*

- Overloading of the SL may cause sprain trauma to any portion of the ligament. Hyperextension of the carpus/tarsus in conjunction with severe overextension of the fetlock joint has been proposed to cause proximal lesions.
- Working horses in deep, soft arenas or in eventing where there is excessive rotational movement of the limbs may increase the risk of injuries.
- Lesions within the branches are also associated with fetlock lameness and suggest that high rotary motion of the fetlock may predispose to suspensory branch injury as may occur in racehorses and horses with dropped fetlock conformation.

### *Clinical Signs*

- Most horses with PSD present with a history of intermittent lameness that is exacerbated with exercise. Heat and swelling may be palpable on the proximal cannon bone region in acute cases but typically this is difficult to detect.
- Firm digital pressure overlying the proximal SL usually elicits a nonfatiguable painful response.
- Lower limb flexion exacerbates the lameness in 50% of horses with forelimb suspensory problems and hock flexion exacerbates the lameness in 85% of horses with hindlimb suspensory problems.
- Horses with injury to the body or branches of the SL usually have visible and palpable swelling and pain at the site of injury.
- A lateral palmar in the forelimb and the deep branch of the lateral plantar nerve (DBLPN) in the hindlimb are the most specific nerve blocks to document PSD in each limb.

### *Diagnosis*

- Ultrasonography of the body and branches of the SL is the imaging technique of choice to diagnose lesions in these locations. However, radiographs should be included to identify the concomitant bone abnormalities such as distal splint bone fractures.
- Definitive diagnosis of PSD is more difficult and combinations of ultrasound, radiology, nuclear scintigraphy, MRI, and CT may be needed to document damage within the origin of the SL (Figures 4.76, 6.52).
- Radiographic abnormalities of proximal MCIII/MTIII such as bone sclerosis, fractures, and enthesiophyte formation appear to be more common with hindlimb PSD than forelimb PSD (Figure 6.52).
- Ultrasound can be difficult in the proximal cannon bone region. Abnormalities with PSD include enlargement of the ligament, poor definition of margins, central or peripheral hypoechoic areas, diffuse reduction in echogenicity, hyperechogenic foci, and irregularity of the palmar/plantar cortex of the MCIII/MTIII. However, the lack of ultrasonographic abnormalities does not rule out a problem.
- MRI is currently the most accurate and definitive imaging modality to identify abnormalities within the proximal SL (Figure 4.76).

### *Treatment*

- Immediate first aid treatment in acute cases should include NSAIDs, cold hosing or icing, and bandaging to reduce swelling and support the fetlock.
- Treatment for horses with body or branch lesions includes a convalescence program of confinement and slow return to exercise. Intralesional therapies with stem cells or PRP may be used to augment healing if indicated.
- Total healing time is often eight months and return to full competitive performance may not be possible for one year. Recurrence of injury is always a concern, especially in the hindlimbs.
- Treatment of PSD can be difficult, especially in the hindlimb. Reported nonsurgical treatments include rest and rehabilitation, shock wave therapy, local infiltration with sarapin, and intralesional therapy with stem cells or PRP. Surgical treatments have included fasciotomy alone, ultrasound-guided desmotomy and fasciotomy, fasciotomy and osteostixis of MCIII/MTIII, and fasciotomy and neurectomy of the DBLPN in the hindlimb.

### *Prognosis*

- The prognosis for PSD in the forelimb is reported to be good for return to full work in sport horses.
- The prognosis is less favorable for horses with PSD of the hindlimbs.

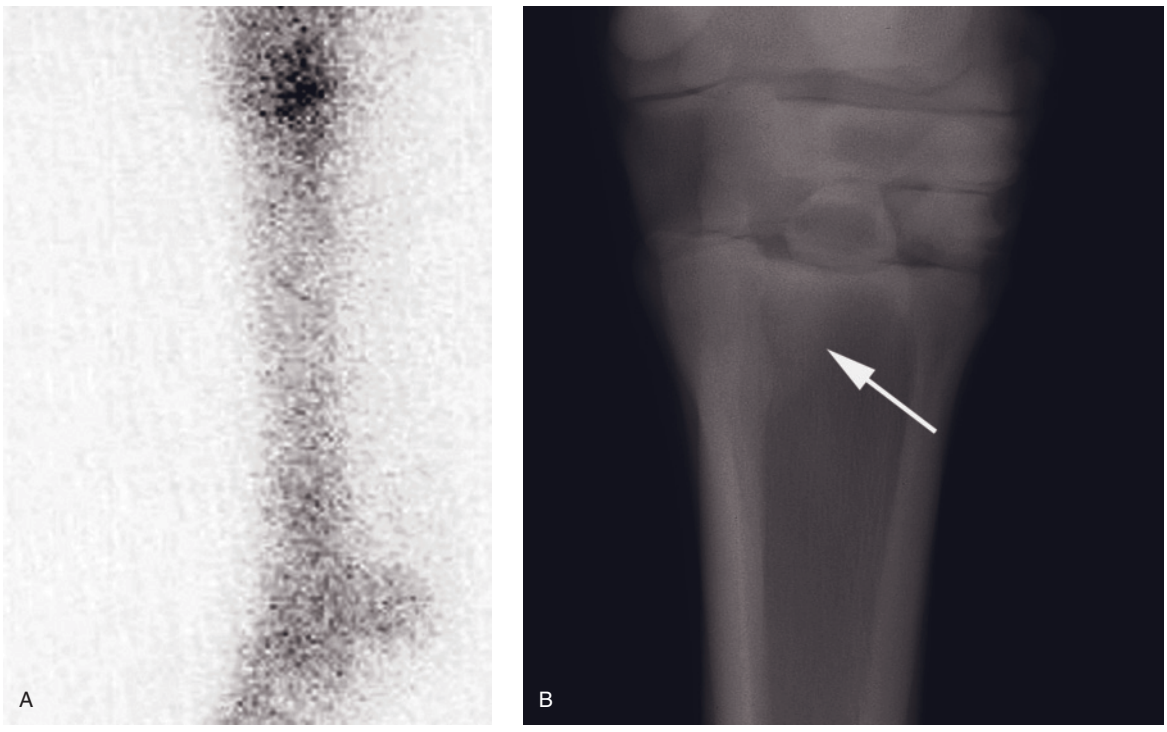



**Figure 6.52.** (A) Nuclear scintigraphy can identify bone injury at the origin of the suspensory ligament. Radiographic lesions such as sclerosis (arrow) can be subtle (B) and usually only identified on the cranio-caudal view due to the overlap with the splint bones on the lateral view. Courtesy of Alicia Bertone.

- Neurectomy of the DBLPN has been reported to result in an 80% return to performance for hindlimb PSD.
- Concurrent bone abnormalities are thought to negatively impact the overall prognosis of treatment.

## DEGENERATIVE SUSPENSORY LIGAMENT DESMITIS (DSLD)

### *Introduction*

- DSLD is a debilitating disorder thought to be limited to the suspensory ligaments of Peruvian Pasos, Peruvian Paso crosses, Arabians, American Saddlebreds, Quarter horses, Thoroughbreds, and some European breeds.
- It primarily affects the body and proximal aspects of the ligament and is often bilateral.

### *Etiology*

- The etiology of DSLD is unknown, but the disease tends to run in families, suggesting hereditary influences.
- A recent study suggests that the disease may be due to excessive accumulation of proteoglycans within the SL and other tissues in affected horses. The authors suggest that DSLD is actually a systemic disorder of proteoglycan accumulation.

### *Clinical Signs*

- Both forelimbs or hindlimbs can be involved, and the condition often leads to persistent incurable lameness requiring euthanasia.
- Many horses have excessive fetlock extension (dropping) on presentation and horses with hindlimb involvement often have the combination of straight hocks and fetlock hyperextension (Figure 6.53).
- Affected horses often have generalized thickening of the cannon bone region and palpable enlargement and pain of the SL.

### *Diagnosis*

- The diagnosis is usually based on patient signalment and history, clinical examination, and

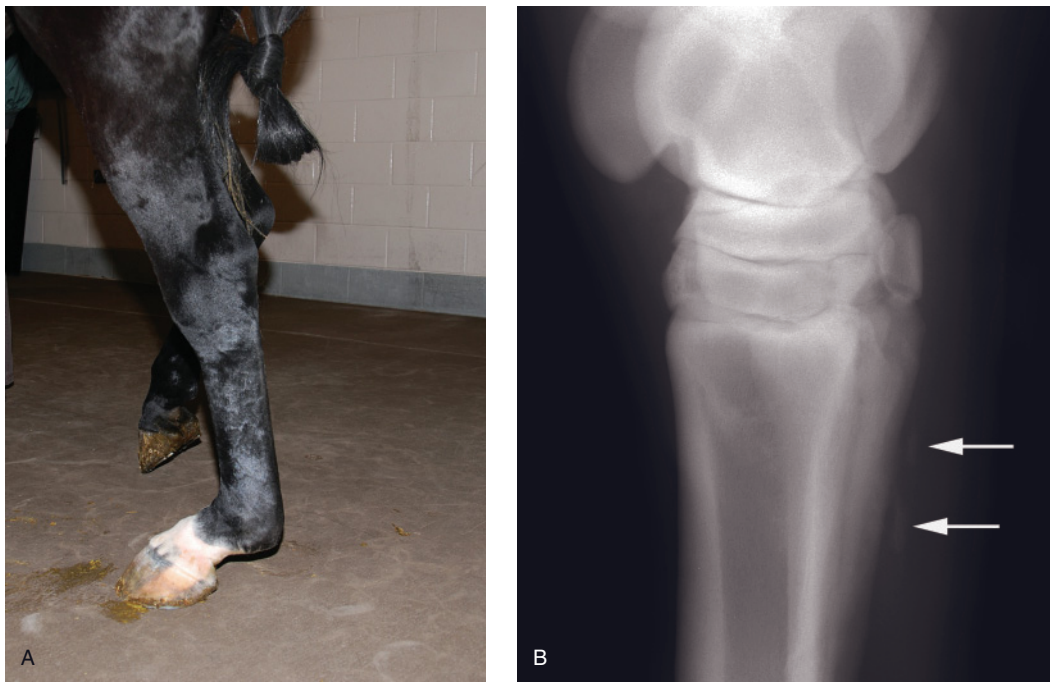



**Figure 6.53.** (A) Severe dropping of the fetlock together with a straight hock conformation is typical of horses with DSLD. (B) Dystrophic mineralization of the suspensory ligament (arrows) can sometimes be identified radiographically.

ultrasonographic abnormalities within the affected SLs.
- Radiographs may be normal but calcification within the ligament or enthesiophyte formation on the palmar/plantar MTIII may support the diagnosis (Figure 6.53).

### *Treatment*

- There is no known treatment for this condition. Treatment is empirical and supportive but often not effective in altering progression of the disease. Horses often remain lame or worsen over time.

### *Prognosis*

- The prognosis is poor for recovery and most horses are euthanized.

## SUPERFICIAL DIGITAL FLEXOR (SDF) TENDINITIS (BOWED TENDON)

### *Introduction*

- Tendinitis of the superficial digital flexor tendon (SDFT) is a common soft tissue injury in performance horses, especially racehorses.
- SDFT injury is almost exclusively a forelimb problem and most commonly occurs in the mid-to-proximal aspect of the cannon bone. Less common locations include the distal MC/MT ("low bows"), the branches of the SDFT in the pastern region, and the caudal aspect of the carpus.
- Lesions in the distal cannon bone are often referred to as "low bows" and can be associated with digital sheath tenosynovitis or constriction of the anular ligament (Figure 6.54).

### *Etiology*

- Excessive hyperextension of the fetlock associated with exercise places the SDFT under very high tensile loads, contributing to strain injuries within the tendon.
- Repetitive trauma to the SDFT can result in microdamage to the collagen structure that contributes to final failure of the fibrils.
- The strength of the SDFT appears to decrease with age, making older horses more susceptible to injury.

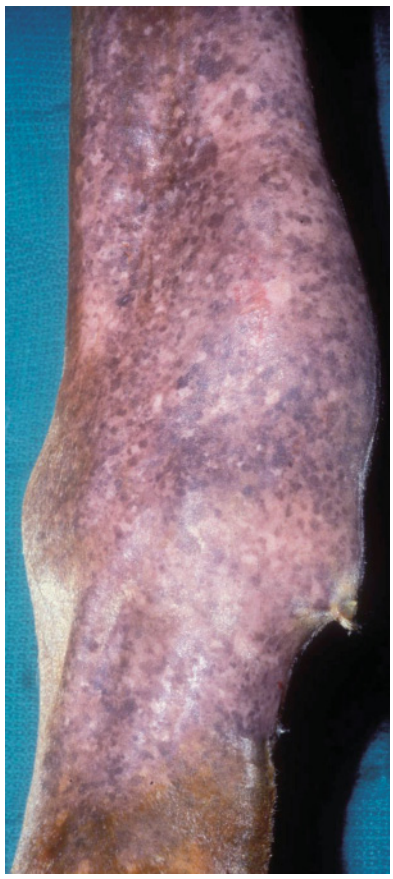


**Figure 6.54.** A "low bow" of the SDFT, as shown in this image, can be associated with concurrent digital tendon sheath tenosynovitis.

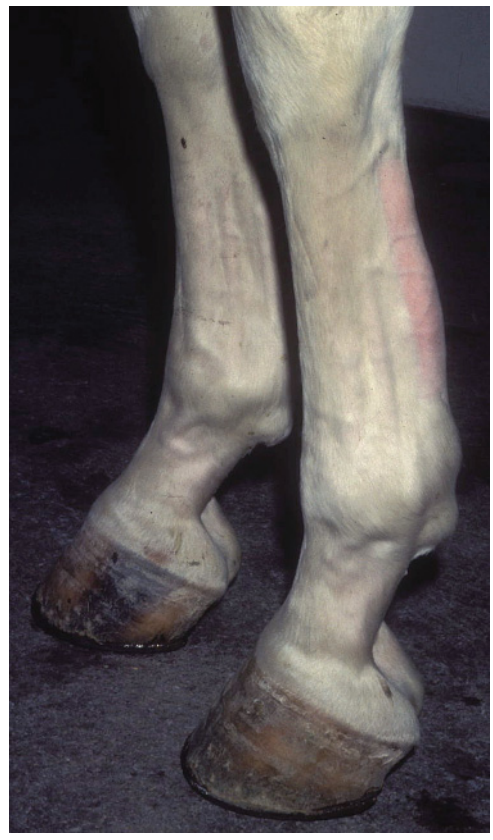


**Figure 6.55.** Classic appearance of SDF tendinitis of the left forelimb. Note the convex palmar surface to the middle region of the metacarpus.

### *Clinical Signs*

- The clinical signs are variable and usually parallel the severity of the tendon damage.
- Early focal swelling, heat, and tenderness may occur before a detectable lameness. Intervention at this early phase can prevent structural damage to the tendon.
- Classic signs of acute tendinitis include diffuse enlargement of the cannon bone region, palpable tendon thickening, heat, pain, and moderate lameness.
- Chronic tendinitis is manifested by fibrosis and firm swelling on the flexor surface of the cannon bone (Figure 6.55). Signs of inflammation or lameness may or may not be present.

### *Diagnosis*

- Ultrasonography enables initial and reasonably accurate determination of the extent and location of the lesions.
- Ultrasound abnormalities may include tendon enlargement and focal or diffuse loss of the normal echogenicity (hypoechoic areas) due to hemorrhage, edema, or fibrillar disruption (Figure 6.56).
- Repeat ultrasound after the initial signs of inflammation have resolved is recommended to more accurately determine the extent of fiber disruption.
- MRI can be performed to further characterize SDFT injuries but is not routinely performed because of the cost. MRI has demonstrated an advantage over ultrasound to distinguish fibrosis from normal tendon in chronic tendinitis.

### *Treatment*

- Initial first aid therapy such as icing, cold-water hydrotherapy, massage, pressure wraps, topical hyperosmotic sweats or diclofenac

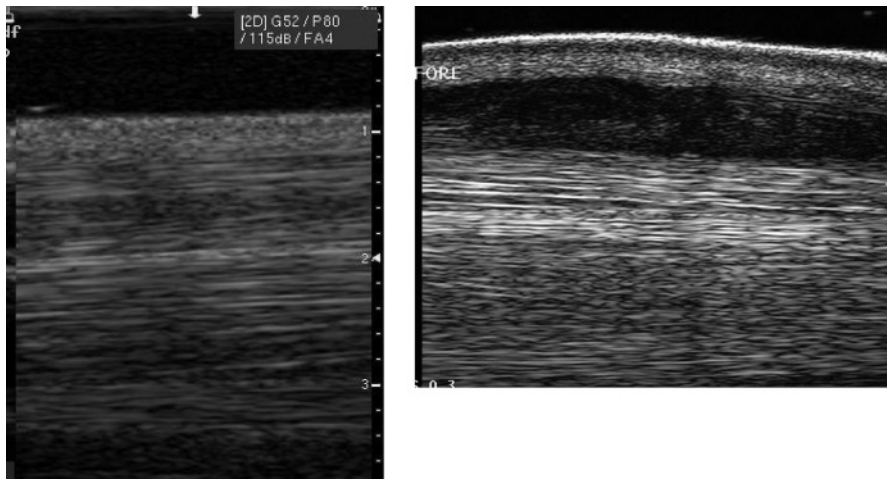



**Figure 6.56.** Ultrasound longitudinal sections through the midportion of a normal SDFT (left) and SDFT with acute tendonitis. Note the hypoechoic area (black) in the middle of the SDFT that usually corresponds to edema or hemorrhage in acute cases. Courtesy of Alicia Bertone.

liposomal cream, systemic NSAIDs, and rest is recommended. A soft cast or resin cast may be indicated if a severe injury is suspected.
- The duration of inactivity and the rehabilitation program depends on the severity of the lesion on ultrasound. If ultrasound examination is normal, then hand walking followed by a return to exercise in 30 days may be effective. Severe injuries may require a year of rest and rehabilitation (Table 6.1).
- Intralesional regenerative therapies such as stem cells and PRP are currently thought to improve healing and prevent recurrence of the injury in racehorses.
- Potential surgical treatments include tendon splitting and proximal accessory ligament transection on the affected limbs and frequently on both forelimbs. These treatments seem to have fallen out of favor in recent years.
- Proper rehabilitation and a convalescent exercise program remains paramount to success with any treatment (Table 6.1).

### *Prognosis*

- The incidence of re-injury is often used as an outcome measure for successful management of SDF tendinitis. Published reports of re-injury in horses treated conservatively with rest alone are 48% and 56%.
- Stem cell therapy reports significantly reduced a re-injury rate of 18% and surgical transection of the accessory ligament of the SDFT reports a reduced re-injury rate of 25%.
- In general, the prognosis for return to racing is often dictated by the severity of the initial tendon injury.

## COMMON DIGITAL EXTENSOR (CDE) TENDON RUPTURE

### *Introduction*

- Rupture of the CDE tendon is a bilateral or unilateral condition seen in foals shortly after birth.
- It has been reported to occur concurrently with other congenital conditions such as decreased endochondral ossification at other sites, decreased pectoral muscle mass, and prognathic conformation to the jaw.
- It is overrepresented in Arabian horses, Quarter horses, and Arab-Quarter horse crosses.

### Etiology

- It is speculated to be a heritable situation, especially if other congenital defects are present.
- Rupture of the CDE tendon is often found together with other flexural deformities of the limb.

**Table 6.1.** A standard exercise program recommended following tendon injury. This protocol can be modified based upon ultrasound recheck examinations.

| Exercise level | Weeks | Duration and nature of exercise |
|---|---|---|
| 0 | 0–2 | Box rest |
| 1 | 3 | 10 minutes walking daily |
| 1 | 4 | 15 minutes walking daily |
| 1 | 5 | 20 minutes walking daily |
| 1 | 6 | 25 minutes walking daily |
| 1 | 7 | 30 minutes walking daily |
| 1 | 8 | 35 minutes walking daily |
| 1 | 9 | 40 minutes walking daily |
| 1 | 10–12 | 45 minutes walking daily |
| **Week 12: Repeat ultrasound examination** | | |
| 2 | 13–16 | 40 minutes walking and 5 minutes trotting daily |
| 2 | 17–20 | 35 minutes walking and 10 minutes trotting daily |
| 2 | 21–24 | 30 minutes walking and 15 minutes trotting daily |
| **Week 24: Repeat ultrasound examination** | | |
| 3 | 25–28 | 25 minutes walking and 20 minutes trotting daily |
| 3 | 29–32 | 20 minutes walking and 25 minutes trotting daily |
| **Week 32: Repeat ultrasound examination** | | |
| 4 | 33–40 | 45 Minutes exercise daily, gradually increasing in amount |
| 4 | 41–48 | 45 minutes exercise daily with fast work 3 times a week |
| **Week 48: Repeat ultrasound examination** | | |
| 5 | 48+ | Return to full competition/race training |

Davis CS, Smith RKW: 2006. Equine Surgery. *In:* Auer JA, Stick JA, (eds) Philadelphia, Saunders, 1110.

- Chronic changes to the CDE tendon also have been reported to occur *in utero*.

## Clinical Signs

- Clinical signs include swelling on the dorsolateral aspect of the carpus, mainly in the form of effusion in the CDE tendon sheath (Figure 6.57).
- Forward buckling of the carpi and/or knuckling of the fetlock may exist concurrently, and affected foals can be easily confused with those with simple flexural deformities.
- Heat and palpable pain are usually not present.

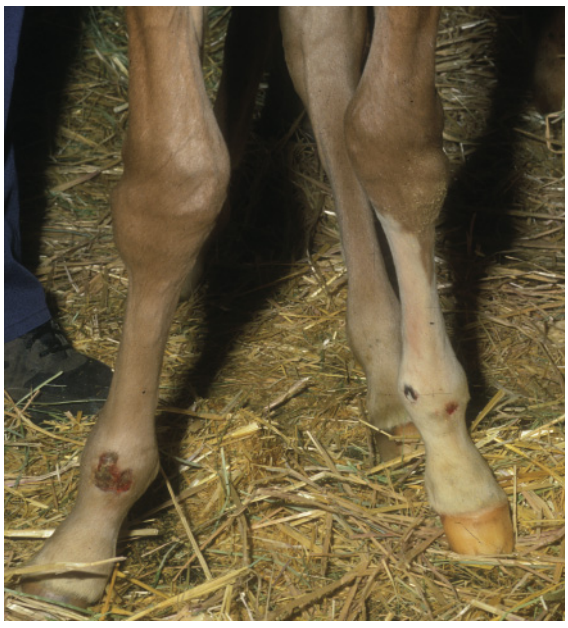


**Figure 6.57.** Bilateral symmetrical fluid swellings of the lateral aspects of both carpi in neonatal foals are characteristic of rupture of the common digital extensor tendons.

### *Diagnosis*

- The diagnosis is usually based on physical findings alone.
- Radiographs of the carpus can be taken to rule out incomplete ossification of the cuboidal bones, but are usually unnecessary.
- Ultrasound of the carpi can be performed to document the amount of separation of the tendon ends, but is also usually unnecessary.

### *Treatment*

- Treatment commonly includes confinement with or without bandaging and splinting if concurrent flexural deformities are present.
- Suturing has been advocated by some but is unnecessary because the tendon ends are unlikely to heal primarily.

### *Prognosis*

- In uncomplicated forms of rupture of the CDE tendon, the prognosis is often very good.
- The prognosis is reduced if severe flexural deformities are present concurrently.

## EXTENSOR CARPI RADIALIS (ECR) TENDON DAMAGE

### *Introduction*

- The tendon of the ECR muscle courses over the dorsal aspect of the carpus and inserts on the proximal aspect of MCIII.
- A tendon sheath encircles the tendon from about 8 cm above the distal end of the radius to the middle carpal joint.
- Swellings of the ECR tendon sheath can be confused with effusion of the carpal joints, carpal hygroma, and effusion of the CDE tendon sheath.

### *Etiology*

- Inflammation of the tendon sheath occurs most commonly because rupture or tearing of the ECR tendon is rare.
- Tenosynovitis of the ECR sheath in adults has been reported to occur mostly in jumpers and in horses that have exostoses on the distal radius. This is typically traumatic in origin from hitting the dorsal aspect of the carpus.
- Infection of the ECR tendon leading to septic tenosynovitis has been reported in a limited number of horses.

### *Clinical Signs and Diagnosis*

- Swelling and effusion of the sheath over the carpus is palpable in most cases (Figure 6.58).
- With acute injuries, pain is present with flexion of the carpus. This subsides with chronicity.
- With infection, there is usually palpable heat, pain and swelling, moderate to severe lameness, and severe pain with carpal flexion.
- Overflexion of the carpus as the foal or horse walks occurs with complete rupture.
- There may or may not be ECR muscle atrophy and a palpable defect is often detected within the swelling of the ECR tendon sheath if the tendon has ruptured.
- Ultrasonography is the imaging modality of choice to assess the ECR tendon and sheath. Radiographic abnormalities are rarely present.

### *Treatment*

- Nonseptic tenosynovitis usually responds to rest, topical and intrasynovial medication, and bandaging.
- Surgical repair in adults with complete rupture in the acute stages has been advocated, with casting for two to four weeks after surgery.

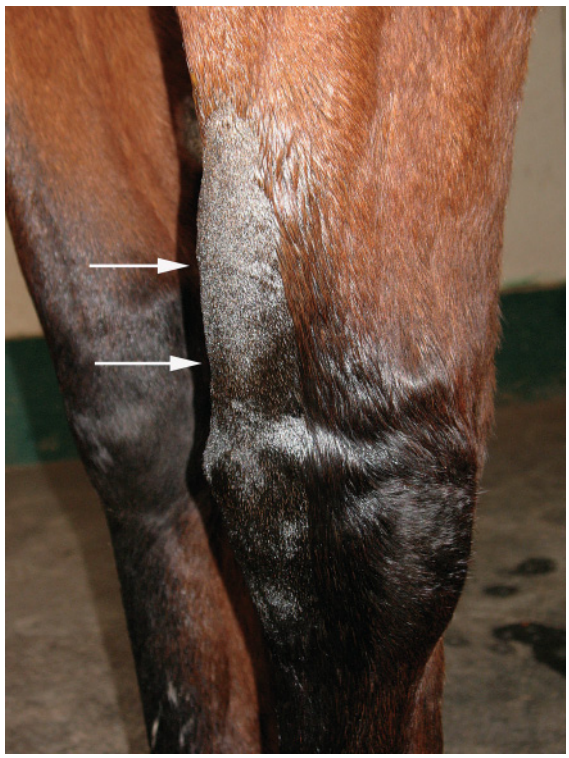


**Figure 6.58.** An adult horse with synovial effusion of the ECR tendon sheath proximal to the carpal joints (arrows). Courtesy of Ty Wallis.

- Tenoscopy and debridement of the ruptured ends also has been advocated but is rarely performed.
- Resection of the infected ECR together with the tendon sheath has been recommended in horses with chronic infection.

### Prognosis

- The prognosis for complete rupture of the ECR tendon in adults is guarded for athletic use but guarded to good for those with partial tears.
- Resection of the ECR due to infection does not preclude an athletic career, but only a few cases have been reported.

## INTRA-ARTICULAR CARPAL FRACTURES

### *Introduction*

- There are three types of fractures that can occur within the carpal joints of the horse:
  1. Osteochondral fragmentation: one articular surface
  2. Slab fractures: two articular surfaces
  3. Comminuted fractures: multiple pieces
- Osteochondral fragmentation typically occurs at consistent locations in racehorses (distal radiocarpal, proximal intermediate, distal lateral radius, and proximal third carpal bone), which reflects the chronic nature of the disease.
- Standardbreds appear to be prone to problems in the middle carpal joint, especially on the third carpal bone.
- Acute fracture and fragmentation of the carpus can occur; this is typically at unusual locations, especially in the palmar aspect of the joints.
- Slab fractures commonly occur on the third carpal bone of Thoroughbreds and Standardbreds but can involve the intermediate and radial carpal bones as well.
- Comminuted fractures primarily involve the third carpal bone but they can also involve the radial carpal, intermediate carpal, and fourth carpal bones. These horses may be axially unstable and have concurrent ligamentous damage, contributing to a poor outcome.

### *Etiology*

- It is hypothesized that fatigue of the soft tissues, increased speed, poor racing surface, poor trimming, and uncoordinated movement may contribute to hyperextension of the carpus and/or incongruent articulation among the carpal bones, predisposing to fractures.
- Subtle geometric abnormalities within the carpal joints may also predispose these horses to fracture.
- Increased axial loading that occurs with speed also leads to increased dorsal compression between opposing carpal bones.
- Histopathologic observations have demonstrated bone microdamage and intense bone modeling and remodeling at the sites of osteochondral fragmentation. Chronic repetitive stress leads to a chronic pathologic process that ultimately leads to bone failure.

### *Clinical Signs*

- Varying degrees of synovial effusion, soft tissue swelling, pain with carpal flexion, and lameness are usually present in horses with IA carpal fractures. Palpation of the dorsal aspect of the carpal bones may demonstrate focal pain of specific bones.
- The severity of clinical signs often indicates the severity of articular damage. Horses with

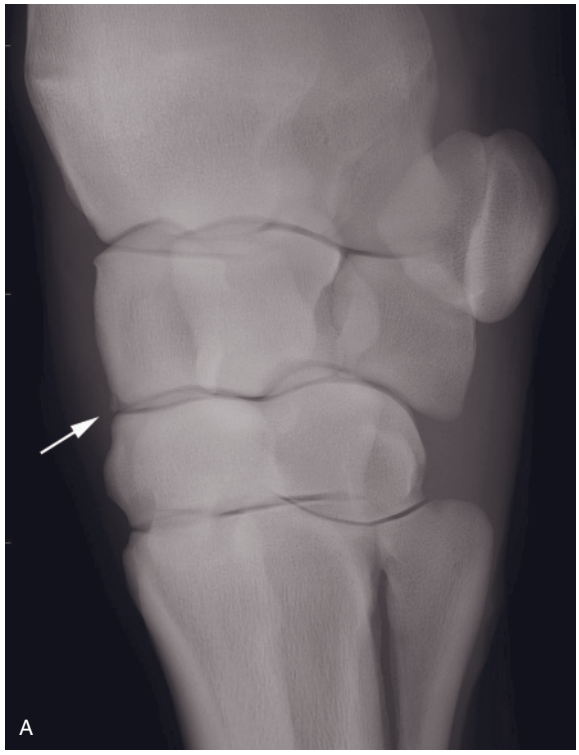
fragmentation alone may have minimal lameness but palpable synovial effusion, while horses with comminuted fractures are often severely lame and have significant soft tissue swelling.
- Horses with fractures of the palmar aspect of the joints are often significantly responsive to flexion, which is reflective of the concurrent soft tissue damage.
- IA anesthesia may or may not be necessary to document the carpus as the site of the lameness.

### *Diagnosis*

- A minimum of six radiographic views is often needed to fully characterize the carpal joints, especially those with small fragmentation (Figure 6.59). Both carpi should be radiographed in racehorses, because more than 50% will have fragmentation in both carpi.
- Images should be evaluated for osteochondral damage, the presence of OA such as osteophytes, enthesiophytes, joint space narrowing, and subchondral bone lysis.
- Fragmentation is often best seen on a flexed lateral view (Figure 6.60), while slab fractures of the carpus are often most easily seen on the standing lateromedial projection. Damage to the third carpal bone may only be seen the skyline projection (Figure 6.61).
- Nuclear scintigraphy may be beneficial, especially in racehorses, to identify remodeling of the carpal bones before fragmentation or to identify subchondral disease.
- MRI and CT also have been used to characterize subtle lesions in the carpus but are not used routinely because of the expense.
- Many times arthroscopy is used as a diagnostic and treatment tool for osteochondral fragmentation.

### *Treatment*

- A more predictable outcome is assured with arthroscopic removal of osteochondral frag-



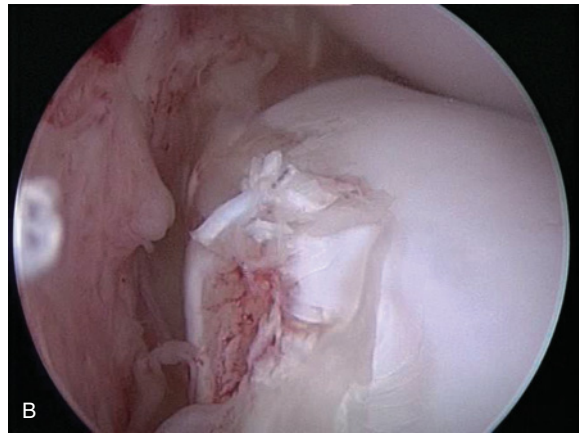




**Figure 6.59.** (A) A radiographic image showing a subtle fragment on the distal aspect of the radial carpal bone (arrow). (B) The corresponding surgical image is shown as well. This demonstrates how radiographs typically underestimate the severity of articular cartilage damage within the joint. Courtesy of Chris Kawcak.

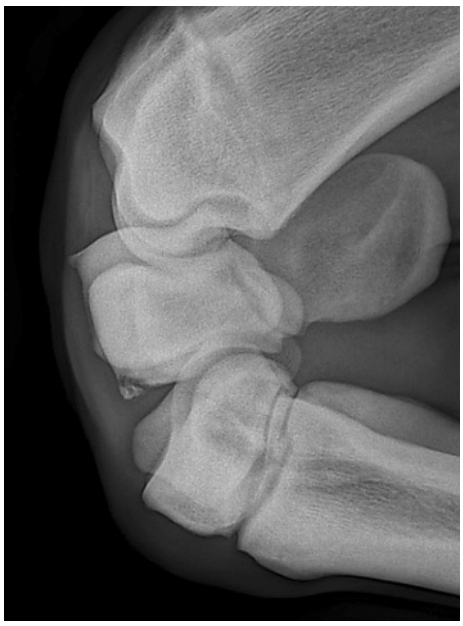


**Figure 6.60.** Radiograph demonstrating osteochondral chip fragments off the proximal intermediate and distal radial carpal bones (a common combination in the racing Quarter horse). Courtesy of CW McIlwraith.

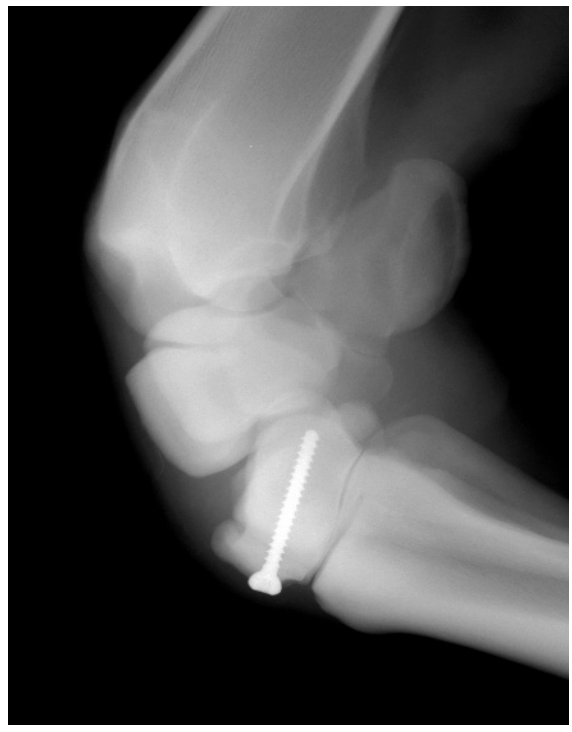


**Figure 6.62.** A flexed lateral radiograph demonstrating a third carpal bone slab fracture that was repaired with a lag screw.

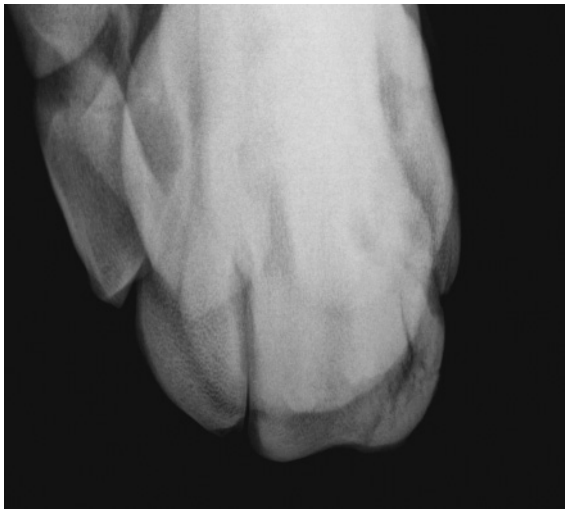


**Figure 6.61.** 30° degree flexed dorsoproximal dorsodistal oblique radiograph showing a slab fracture of the third carpal bone with frontal and sagittal components. Courtesy of Robert Hunt.

mentation whenever possible. The severity of articular cartilage and bone damage is correlated with the outcome.

- Arthroscopy is recommended for horses that block to one or both of the carpal joints but lack radiographic abnormalities.
- Slab fractures usually require internal fixation with lag screw fixation to have the best chance of achieving athletic soundness (Figure 6.62). The degree of joint surface damage, which is common with slab fractures, often dictates the prognosis of return to athletic use.
- Thin slab fractures of the third carpal bone (less than 5 mm) can be removed because they will not support lag screw repair.
- Comminuted fractures usually require internal fixation or arthrodesis of the carpus to restore axial stability to the limb. Conservative therapy with casting and/or splints may be used but often results in more prolonged lameness and complications (limb deviation and chronic lameness).

### Prognosis

- For Thoroughbreds and Quarter horses, the chance of racing at the same or increased level following arthroscopic surgery for osteochondral fragmentation is approximately 68%; for Standardbreds it is reported to be 74%.
- For slab fractures of the third carpal bone, approximately 65% to 75% of horses race after treatment but at a reduced level.
- Horses with comminuted fractures of the carpus usually do not return to racing, and salvage for breeding or light riding is the goal.

## OA OF THE CARPUS

### Introduction

- OA in the middle and radiocarpal joints usually develops secondary to previous articular trauma.
- OA of the carpometacarpal joint is a separate syndrome that predominantly involves older Arabian horses.
- OA of the carpus is often a progressive condition that may become unmanageable to the point that even pasture soundness is questionable.

### Etiology

- Carpal OA may occur in young athletes, such as racehorses, secondary to stress related bone damage that causes physical damage to the joint.
- Carpal OA also may develop in older horses without a history of trauma. These horses often demonstrate an insidious, progressive onset of disease.
- The etiology of carpometacarpal OA is unknown, but there is a suggestion that an anatomic abnormality may exist between the second and third carpal bones.

### Clinical Signs

- Carpal OA most commonly involves the middle carpal joint and horses have varying degrees of carpal effusion, joint capsule thickening, pain on flexion, and lameness.
- Many horses have an obvious visual swelling on the medial aspect of the carpus and in severe cases the limb deviates axially (varus deformity of the carpus; Figure 6.63).
- The severity of lameness is often reflective of the severity of the OA.

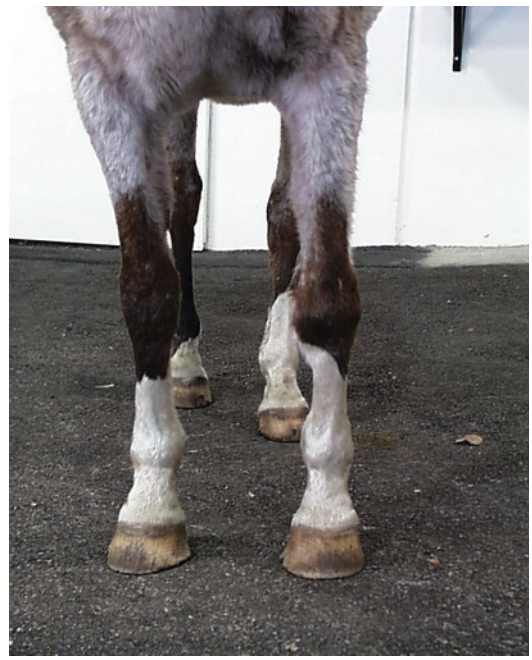


**Figure 6.63.** Aged mare with varus deviation of the left forelimb and swelling of the left carpus secondary to chronic OA of the carpus.

### Diagnosis

- Early radiographic signs of carpal OA may include mild osteophytes, enthesiophytes, and osteochondral fragmentation (Figure 6.64).
- More severe abnormalities include subchondral bone sclerosis and/or lysis and joint space narrowing.
- Carpometacarpal OA is seen radiographically as osteoproliferation and joint space narrowing medially between the second carpal and second MC bone (Figure 6.65).

### Treatment

- Horses with early signs of OA can be treated with controlled exercise and NSAIDs.
- Stall confinement is rarely helpful in these horses, and this supports the fact that strengthening of periarticular soft tissues is of benefit in other species.
- Paddock turn-out seems to help these horses, although their activity should be monitored to avoid excessive exercise.
- Horses with OA and osteochondral fragmentation may benefit from arthroscopy but the goal is usually to help relieve pain and slow but not stop the progression of the disease.
- IA medication with a variety of products (corticosteroids, PSGAGs, HA, IRAP, etc.) can be

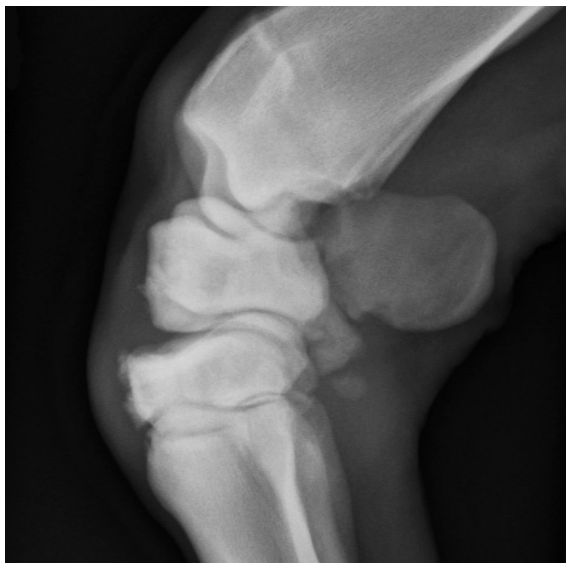


**Figure 6.64.** Flexed lateral radiograph demonstrating the characteristic osteophyte production along the dorsal aspect of the carpus that is often seen with carpal OA.

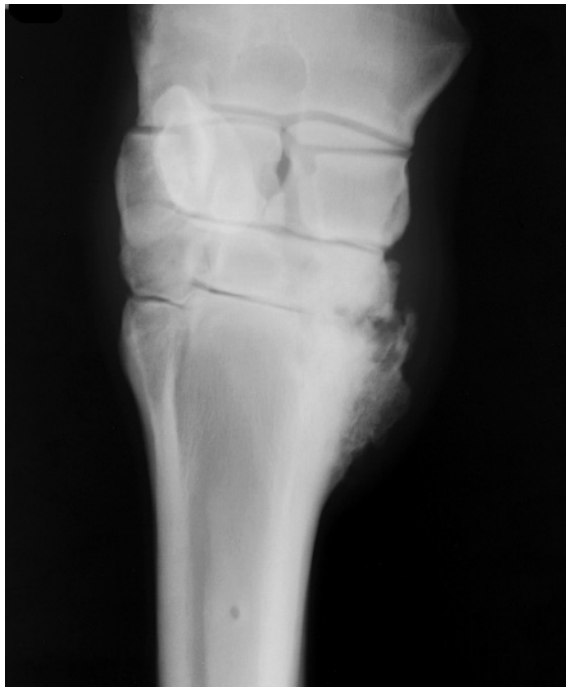


**Figure 6.65.** A radiograph demonstrating collapse of the medial aspect of the joint space and osteoproliferation typical of OA of the carpometacarpal joint.

used to primarily control the pain associated with the disease.
- A shoe with a lateral extension may help alleviate pain and lameness in horses with varus deformities of the carpus (Figure 6.63).
- In severe cases of OA, partial or pancarpal arthrodesis is often advocated to reduce the pain and prevent laminitis in the opposite limb.
- Carpometacarpal OA may be treated with IA drilling of the joint similarly to horses with tarsal OA but the success of this treatment is undetermined.

### *Prognosis*

- The prognosis for athletic performance in horses with carpal OA is usually guarded to poor, depending on the severity.
- Most horses can be managed with a combination of systemic and IA treatments, but the disease is most likely to progress.
- Horses with a varus deformity, medial joint space collapse, and significant lameness often do poorly.

## CARPAL SHEATH TENOSYNOVITIS

### *Introduction*

- The carpal sheath or carpal canal encloses the digital flexor tendons on the medial aspect of the carpus and extends from 8 to 10 cm proximal to the radiocarpal joint distally to near the middle of the metacarpus.
- Effusion of the carpal sheath usually indicates a clinical problem within the sheath, and several different conditions can contribute to the effusion.

### *Etiology*

- Causes of carpal tenosynovitis include osteochondromas at the distal end of the radius, physeal remnants or exostoses of the distal radial physis, damage to the flexor tendons within the sheath, or desmitis of the superior check ligament of the SDFT, or as a sequela to an accessory carpal bone fracture.
- All of these predisposing conditions cause an inflammatory response within the sheath that is manifested as effusion.
- Healing of accessory carpal bone fractures can contribute to later development of carpal sheath tenosynovitis.

### *Clinical Signs*

- Affected horses often present with a history of intermittent lameness that increases with exercise.

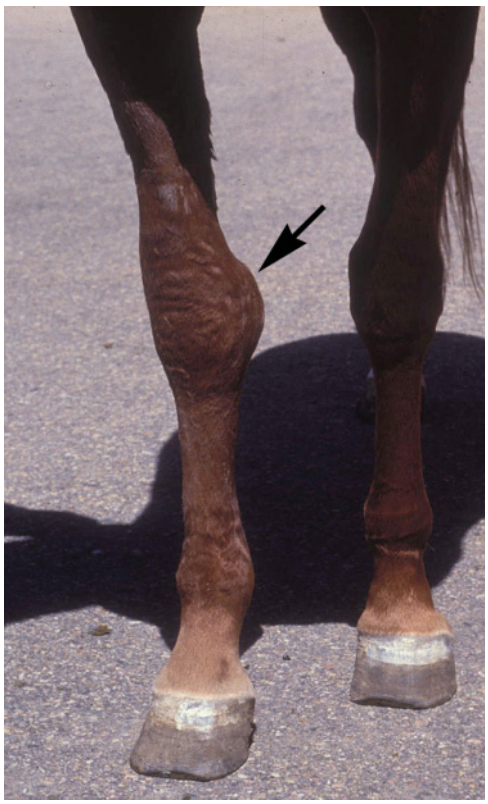


**Figure 6.66.** Carpal canal swelling (arrow) appreciated on the medial aspect of the limb.

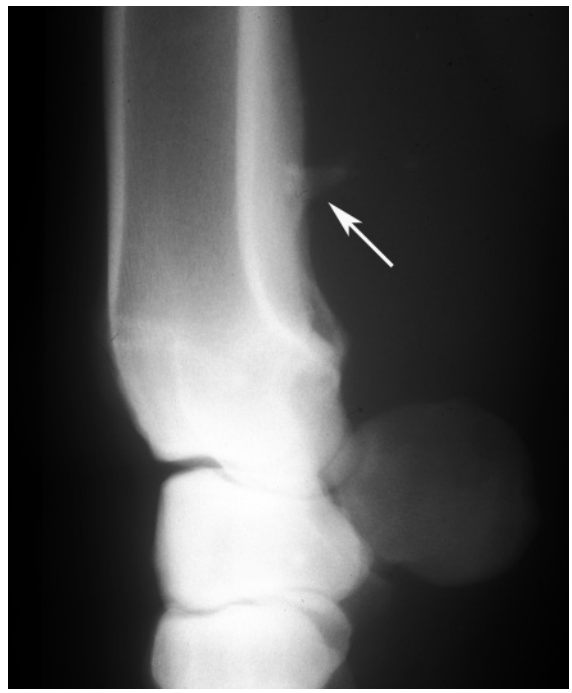


**Figure 6.67.** Lateral radiograph of the carpus, demonstrating an osteochondroma on the caudal aspect of the distal radius (arrow).

- An obvious swelling of the carpal sheath cranial to the ulnaris lateralis laterally or on the medial aspect of the carpus is often present (Figure 6.66). In some cases the swelling can be extensive.
- An osteochondroma may be palpable on the caudodistal aspect of the radius with the limb held flexed at the carpus.
- There is usually reduced range of motion to the carpus and a carpal flexion test worsens the lameness.
- In many cases, intrasynovial anesthesia of the carpal sheath is necessary to document the site of lameness.

### *Diagnosis*

- Radiography is usually used to document the potential cause of the tenosynovitis (osteochondroma, physeal remnant, or accessory carpal bone fracture). Osteochondromas and physeal remnants appear as conically shaped bony protuberances located on the caudomedial aspect of the distal radius adjacent to the physis, and may be difficult to differentiate (Figure 6.67).
- Fractures of the accessory carpal bone are usually obvious but distal migration of a fracture fragment often contributes to the carpal sheath tenosynovitis (Figure 6.68).
- Ultrasonography should be performed to determine the presence of DDFT tendinitis or damage to the superior check ligament of the SDFT.
- Diagnostic tenoscopy of the carpal sheath may be required to document the cause of the tenosynovitis.

### *Treatment*

- Surgical excision of the osteochondromas and physeal remnants via tenoscopy is the treatment of choice.
- Tenoscopy is also recommended to facilitate debridement of lesions of the DDFT that are visible on ultrasound.
- Intrasynovial medication of the sheath often reduces the effusion and lameness, but these

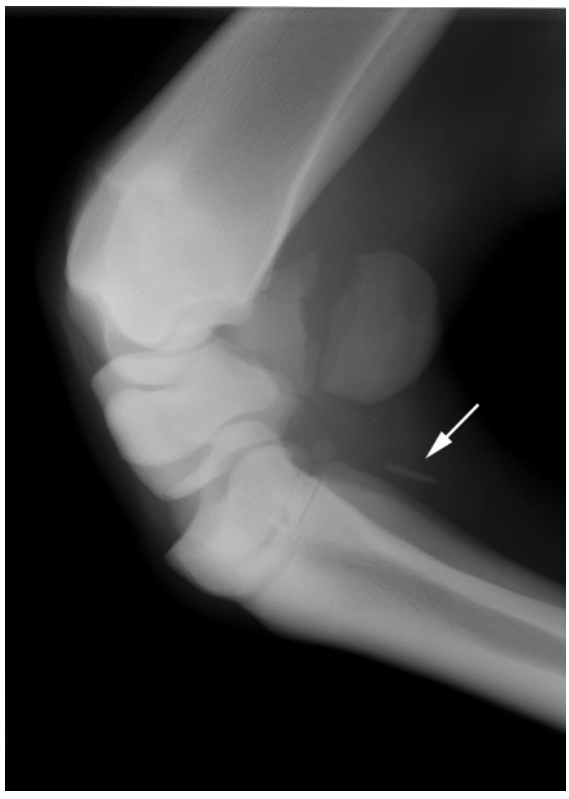


**Figure 6.68.** Flexed lateral radiograph of the carpus of a horse with a chronic accessory carpal bone fracture. A fragment from the accessory carpal bone (arrow) has migrated distally within the sheath, contributing to carpal sheath tenosynovitis.

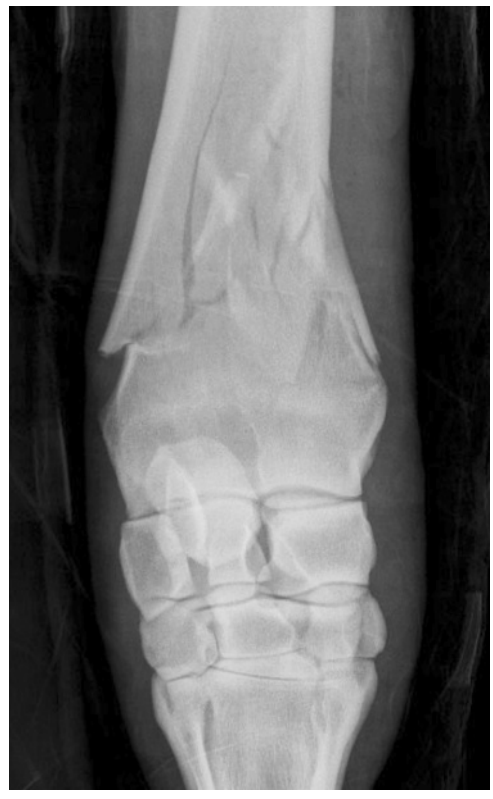


**Figure 6.69.** A cranial-caudal view of a distal diaphyseal comminuted fracture of the radius. Courtesy of Martin Waselau.

will often recur if the primary problem is not addressed surgically.

### *Prognosis*

- The prognosis for surgical excision of solitary osteochondromas and physeal remnants is good for return to performance.
- Horses with osteochondroma or physeal remnants together with significant damage to the DDFT have a reduced prognosis.
- Prognosis for accessory carpal bone fractures is usually reduced if significant carpal canal involvement occurs.

## FRACTURES OF THE RADIUS

### *Introduction*

- Fractures of the radius represent 8% to 14% of all fractures in horses; comminuted fractures are most common (Figure 6.69).
- Open fractures usually involve the medial surface of the antebrachium where there is minimal soft tissue covering.
- Young horses are more successfully treated than adults.

### *Etiology*

- Radial fractures are usually a result of high-impact blunt trauma such as a kick from another horse.
- Age may play a role in fracture configuration. Comminuted or butterfly fragment fractures are usually noted in older horses (older than 2 years), whereas simple oblique and transverse fractures occurred in younger horses (Figure 6.70).

### *Clinical Signs*

- Horses usually present with non-weight-bearing lameness, varying degrees of swelling

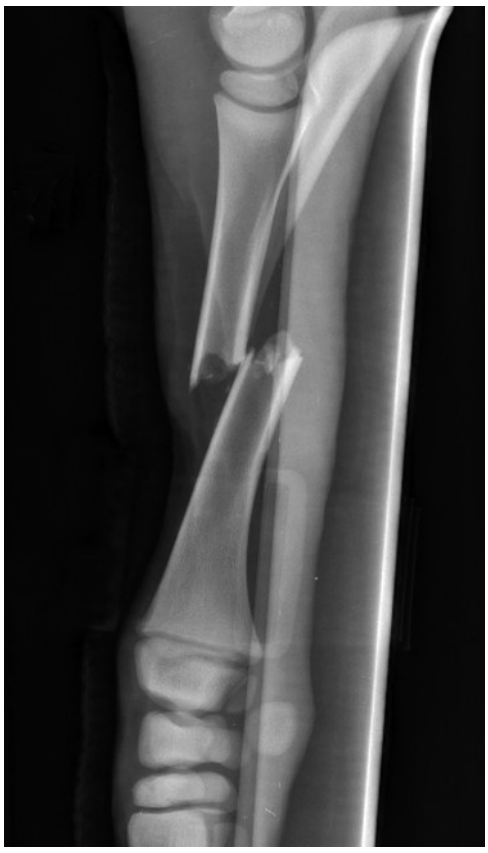


**Figure 6.70.** A transverse complete midshaft fracture of the radius of a foal. Courtesy of Martin Waselau.

in the antebrachium, and instability associated with the fracture site.
- Crepitation may be felt and pain elicited when the distal limb is manipulated.
- Wounds or penetration of the skin from the fracture on the distal medial side of the antebrachium is common.
- Horses with incomplete nondisplaced fractures are usually very lame but are willing to bear some weight on the limb.
- Horses with stress fractures are difficult to identify on physical examination alone.

### *Diagnosis*

- Radiography usually identifies the fracture, although acute nondisplaced fractures may be difficult to identify.
- Comminuted and displaced fractures are easily identified (Figure 6.69).

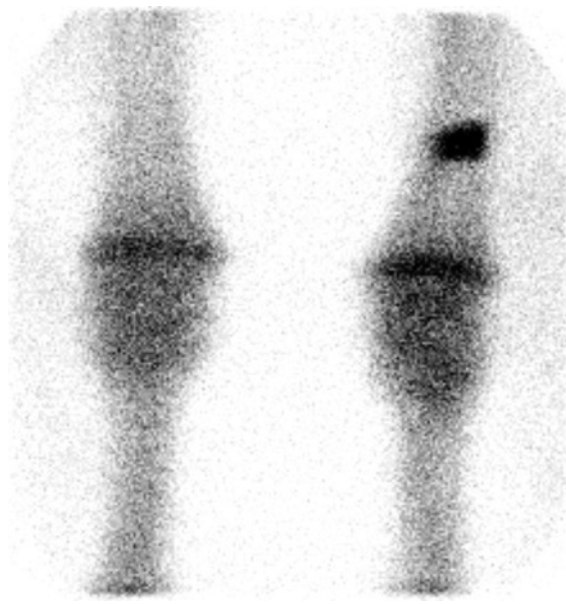


**Figure 6.71.** A nuclear scintigraphic examination of increased uptake of radioactive isotope in the distal radius, indicating a stress fracture of the metaphysis. Courtesy of Dan Burba.

- Nuclear scintigraphy can be used to identify incomplete and stress fractures (Figure 6.71).

### *Treatment*

- Immobilization of a displaced radial fracture is very important if surgery is anticipated (Figure 6.72).
- Internal fixation of complete displaced fractures of the radius is the preferred method of treatment. A single plate may be used in young foals; however, two plates are recommended in most horses.
- Transfixation-pin-casts may be considered for open, contaminated fractures in which the chances of successful plate fixation are poor or when there are economic constraints.
- Cast application alone has been used to successfully treat some distal radial fractures when there are economic constraints.
- Non-displaced, incomplete, and stress fractures are often candidates for conservative treatment.

### *Prognosis*

- The prognosis for displaced radial fractures is best for horses under 2 years of age.

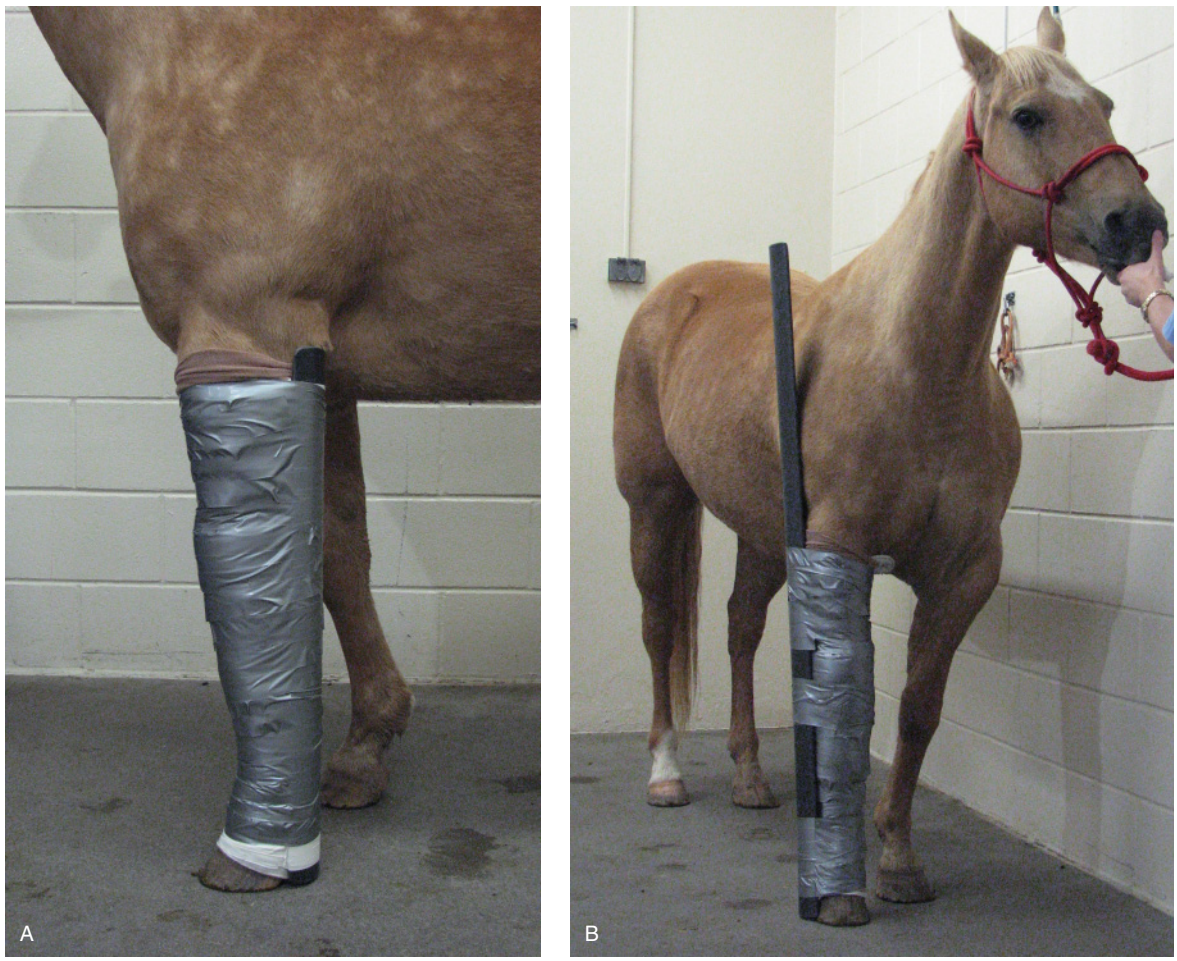



**Figure 6.72.** Proper bandaging and splinting a horse with a radial fracture. A bandage and caudal splint are applied (A) and then a lateral splint extending proximal to the shoulder is used to help prevent limb abduction (B). Courtesy of Jeremy Hubert.

- Most adult horses have an unfavorable prognosis for survival no matter what treatment is selected.
- Horses with incomplete and stress fractures appear to have a good prognosis with conservative treatment.

## FRACTURES OF THE ULNA

### *Introduction*

- The ulna is a non-weight-bearing equine long bone that transmits the extensor function of the triceps apparatus to the distal limb.
- It is a common fracture site, especially in young horses (79% of ulnar fractures were in horses under 2 years of age).
- Ulnar fractures are classified as Types I through VI (Figure 6.73). Type I and II fractures involve the apophysis and Types III through VI involve the diaphysis of the olecranon.
- Proximally located fractures tend to displace more than fractures located distal to the level of the elbow joint.

### *Etiology*

- Direct impact or trauma is the most common cause. Kick injuries appear to be a common cause in adults and may be associated with wounds on the lateral aspect of the elbow.
- Type I and II fractures can occur from excessive tensile load of the triceps apparatus in foals.

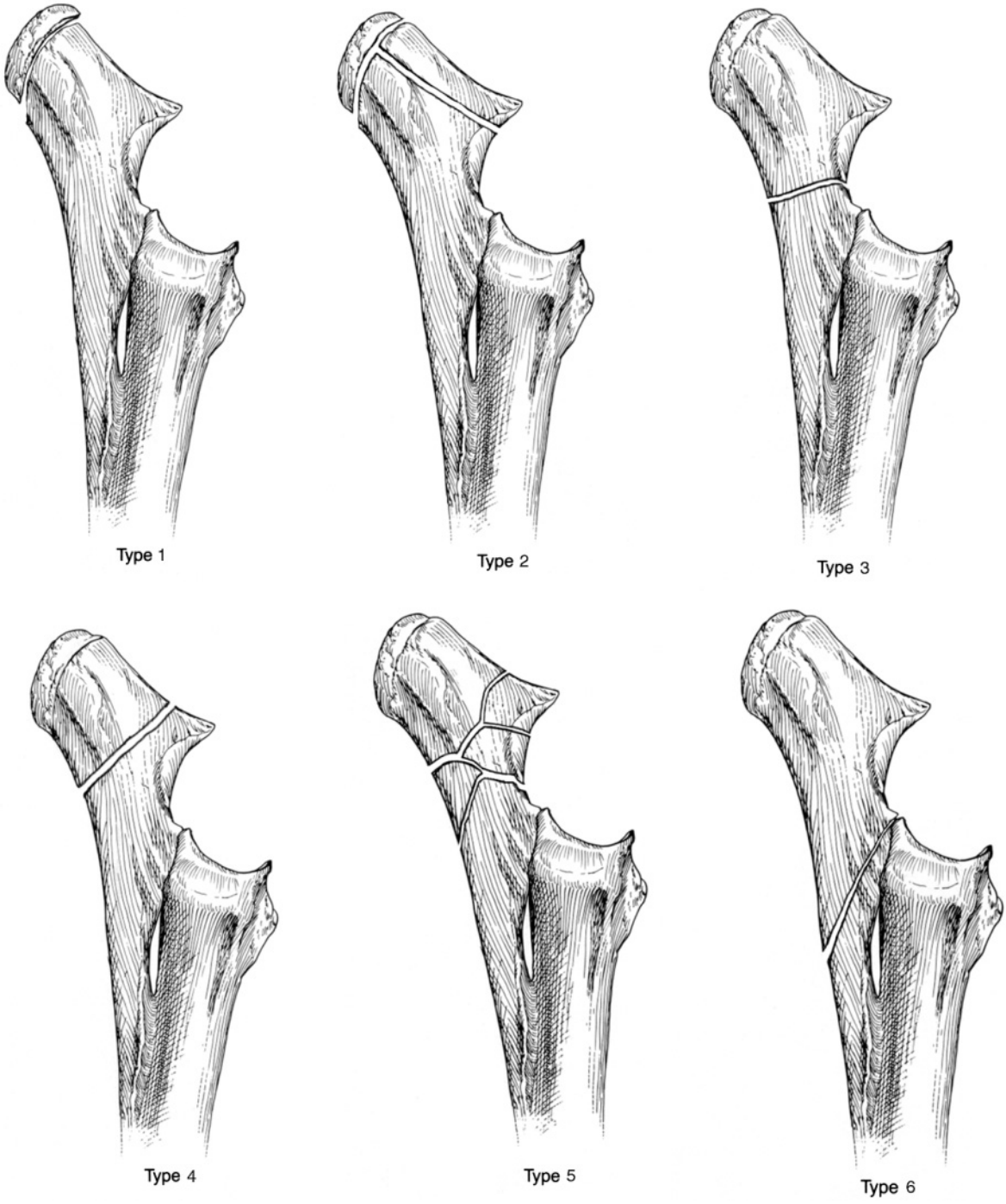



**Figure 6.73.** Classification of equine ulnar fractures.

### *Clinical Signs*

- Fractures of the ulna usually result in an acute non-weight-bearing lameness with a classic dropped elbow appearance (the carpus is flexed and the horse will not bear weight on the limb; Figure 6.74).

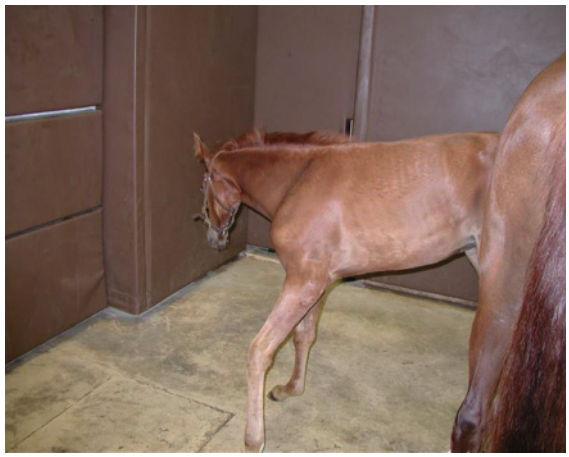


**Figure 6.74.** Typical dropped elbow appearance and the inability to extend the limb that can be seen in horses with fractures of the humerus or olecranon. Courtesy of Robert Hunt.

- Soft tissue swelling is usually present around the point of the elbow, especially in displaced fractures.
- Crepitus may be palpable and skin wounds are not uncommon in adult horses. Limb manipulation is usually resisted.

### *Diagnosis*

- A tentative diagnosis can be made based on the classic appearance of the limb and palpation. Differential diagnoses may include humeral fractures, radial nerve paresis, or joint sepsis if a wound is present.
- Lateral and craniocaudal radiographs of the elbow usually provide a definitive diagnosis (Figure 6.75). It may be necessary to take a flexed-lateral view to provide distraction of the fragment to identify a Type I fracture in foals.

### *Treatment*

- Horses with nondisplaced nonarticular ulna fractures may be treated conservatively with stall rest alone or combined with external coaptation (bandage plus caudal splint from the ground to the elbow; Figure 6.72A).

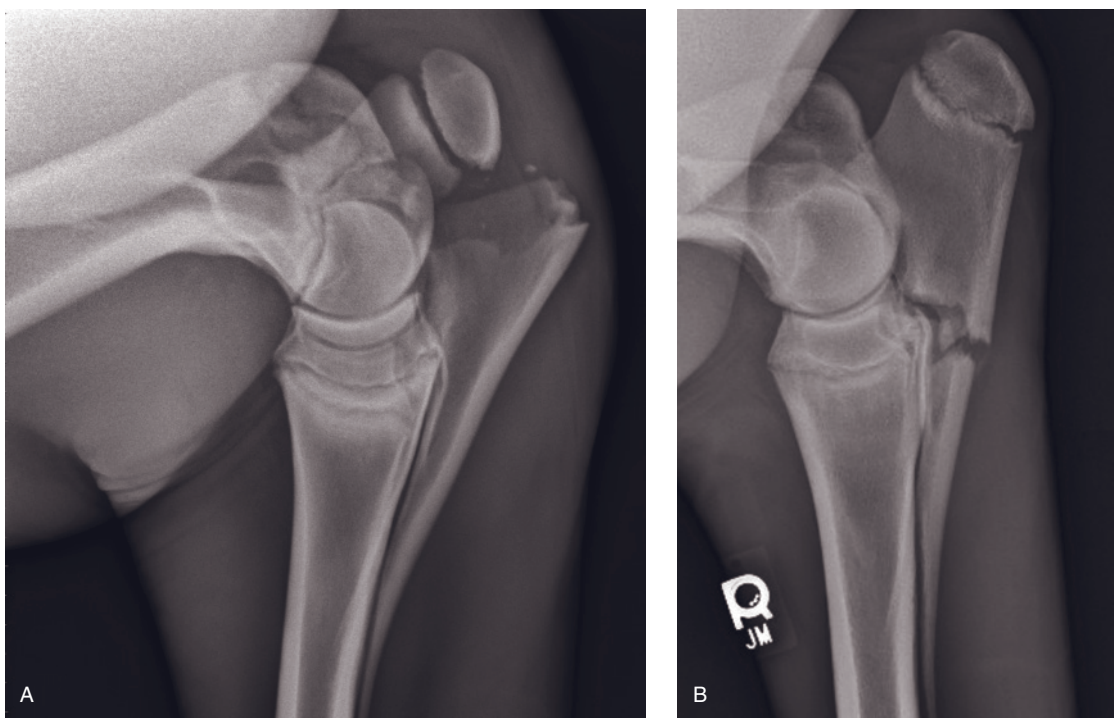



**Figure 6.75.** (A) Type III ulnar fracture. Courtesy of Lorrie Gaschen. (B) Type VI ulnar fracture. Courtesy of Martin Waselau.

- Horses with articular fractures with minimal displacement also may be treated conservatively, but better results are usually obtained with internal fixation. Nonunions can occur in cases treated conservatively.
- In young horses (under 6 months) internal fixation may be performed using a combination of screws, pins and tension band wires, or plating.
- In older horses, application of a plate along the caudal aspect of the ulna is the recommended technique (Figure 6.76). The plates counteract the tension forces of the triceps brachii muscle. Screws should only penetrate the caudal aspect of the radius in older horses (2 years and older) to prevent subluxation of the radiohumeral joint.

### *Prognosis*

- The prognosis for conservative management of nondisplaced fractures, nonarticular fracture Types I and IV of the ulna, is good.
- Nondisplaced articular fractures, especially Type VI fractures, also respond well to conservative treatment. One retrospective study reported 70% of affected horses becoming sound.

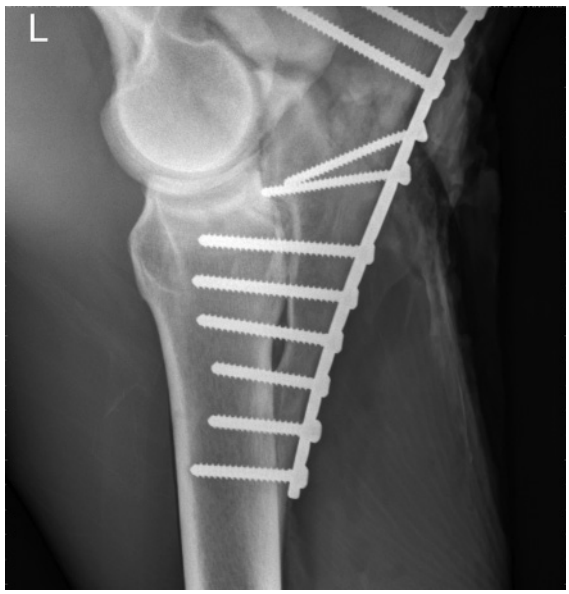



**Figure 6.76.** Type III ulnar fracture repaired with a narrow DCP applied to the caudal aspect of the ulna. Because the fracture is in an older horse, the distal screws penetrate the radius. Courtesy of Katie Amend.

- The prognosis for internal fixation of displaced Type III, IV, V, and VI fractures is also considered good to very good, depending on the technique used and the intended use of the horse.

## SUBCHONDRAL CYSTIC LESIONS (SCLs) OF THE ELBOW

### *Introduction*

- SCLs of the elbow joint have been observed in a wide variety of horse breeds of all ages.
- They are typically located on the proximomedial radius and they communicate with the radiohumeral joint (Figure 6.77). Occasionally they are found on the distal medial condyle of the humerus.

### *Etiology*

- The two proposed causes for SCLs, regardless of location, are either development associated with osteochondrosis or trauma secondary to damage to the cartilage and subchondral bone plate.

### *Clinical Signs*

- Horses often present with a history of an acute onset of lameness that may wax and wane with use.
- On physical examination there are usually no localizing signs other than lameness. Palpation of the caudolateral joint pouch may reveal effusion.
- Flexion and extension of the elbow region often elicits a painful response and usually exacerbates the lameness.

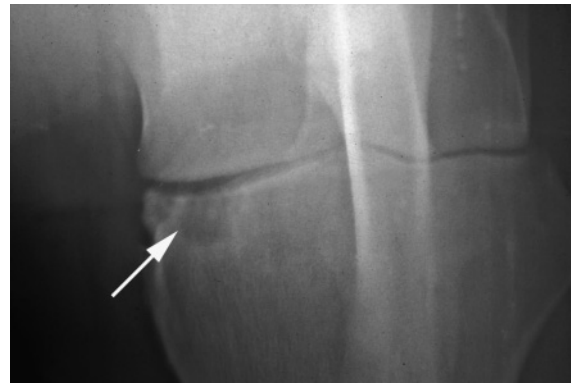


**Figure 6.77.** Subchondral cystic lesion located on the medial aspect of the proximal radius.

- Intrasynovial anesthesia of the elbow is usually needed to locate the site of the lameness.

### *Diagnosis*

- Radiographs are required to make the diagnosis. The craniocaudal view often identifies the SCL on the proximal medial aspect of the radius (Figure 6.77).
- Nuclear scintigraphy may help identify the site of the SCL in cases of early traumatic lesions.

### *Treatment*

- SCLs may be treated conservatively with rest and IA injections or surgically with extra-articular osteostixis with or without corticosteroids, depending on the duration. Injection of the cyst with steroids either via arthroscopic guidance or in an extra-articular manner is the recommended approach.
- If lesions that are treated conservatively do not appear to be resolving or the lameness does not improve, surgical intervention may be advised.

### *Prognosis*

- The prognosis appears good for conservative treatment of SCLs as long as there is no radiographic evidence of OA.
- The prognosis for horses treated with extra-articular osteostixis and steroid injection is currently unknown. There is one report of a successful elbow SCL injection.

## BURSITIS OF THE ELBOW (OLECRANON BURSITIS)

### *Introduction*

- "Shoe boil" or "capped elbows" are two common names for this condition.
- It may occur on one or both elbows and is characterized by a movable swelling over the point of the olecranon tuberosity.
- Bursal enlargement usually results in a painless swelling that does not typically interfere with function unless it becomes greatly enlarged.
- An infected bursa is painful, causes lameness, and may break open to drain (Figure 6.78).

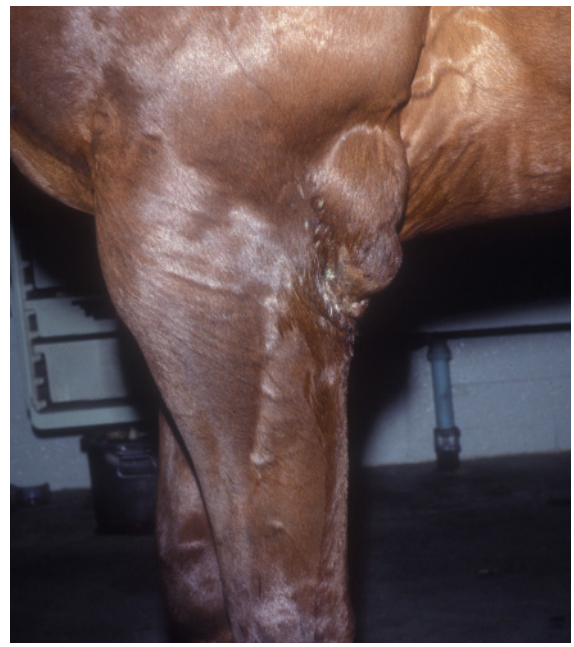


**Figure 6.78.** A chronic, infected, acquired capped elbow of the left forelimb.

### *Etiology*

- Acquired bursitis is commonly caused by repetitive trauma from the shoe of the affected limb hitting the point of the elbow during motion, or more commonly when the horse is lying down. The trauma results in a transudative fluid accumulating in the subcutaneous tissue, which becomes encapsulated by fibrous tissue. A synovial-like membrane develops, producing fluid that is similar to joint fluid.
- American Saddlebreds and Standardbreds may hit their elbows during exercise repeatedly.
- The bursae may become infected by a puncture wound or iatrogenically following intrabursal treatment.

### *Clinical Signs*

- The condition is characterized by a prominent, often freely movable, fluid-filled swelling over the point of the elbow.
- With chronicity the swelling may be comprised primarily of fibrous tissue and may be fixed in position (Figure 6.78).
- Lameness usually is not present, unless the bursa is greatly enlarged or infected. Infected bursae feel warm, and firm pressure causes pain.

### *Diagnosis*

- The diagnosis usually can be made on physical exam findings alone.
- If infection is suspected, radiographs should be taken to rule out trauma or infection involving the olecranon process.
- Ultrasound can be helpful to determine the content of the swelling (fluid vs. scar tissue) and determine whether deeper structures are involved.

### *Treatment*

- In the acute stage, the condition may resolve by preventing further trauma to the region with the use of a shoe boil roll or boot.
- The fluid can be removed aseptically and corticosteroids injected into the bursa. However, this treatment was reported to be successful in only 3/10 horses in one study.
- Intralesional injection of dilute iodine or iodine-based radiographic contrast material, or packing the incised bursa with iodine-soaked gauze also has been recommended, with variable success.
- Surgical intervention, either by placing drains or en bloc resection, appears to have the greatest success.
- En bloc resection is the treatment of choice for large and mature acquired olecranon bursae.

### *Prognosis*

- The prognosis for conservative treatment to achieve an acceptable cosmetic outcome is guarded.
- En bloc resection is regarded as a superior way to manage olecranon bursitis with good results.

## FRACTURES OF THE HUMERUS

### *Introduction*

- Fractures of the humerus can occur in horses of any age, breed, or sex, but most often affect foals under 1 year of age, racing or race training Thoroughbreds, and horses that are used for jumping or steeplechase events.
- Most humeral fractures are complete, closed, and displaced, involve the middle third of the diaphysis, and have considerable overriding of the fracture fragments.
- The radial nerve courses in the musculospiral groove of the humerus, and may be traumatized to varying degrees as a result of the fracture or during surgical repair.
- Incomplete stress fractures occur in two typical locations in racehorses: the proximal caudal lateral cortex and the distal cranial medial cortex.

### *Etiology*

- Humeral fractures frequently occur in foals and weanlings secondary to falls or other impact injuries.
- In racing breeds, they occur subsequent to falling during a race or catastrophic failure of the bone as a result of accumulated stress and microfracture.
- Deltoid tuberosity fractures and proximal humeral fractures may occur from kick injuries or running into objects.
- In an *in vitro* model, the configuration of the fracture was predictable and depended on the direction from which the insult originated. When the force was applied in a craniocaudal direction, the humerus fractured transversally; when the force was applied in a lateral to medial direction, the humerus fractured obliquely.

### *Clinical Signs*

- Horses with nondisplaced or minimally displaced fractures may present with a history of a severe lameness that quickly improved. Swelling and pain on deep palpation is usually present at the site of injury.
- Horses with complete displaced fractures usually present with an acute onset of severe non-weight-bearing lameness. Moderate to severe swelling of the muscles overlying the humerus is often seen and the elbow is usually dropped. Crepitus may be heard with limb manipulation and there is an increased range of motion when the limb is adducted and abducted.
- Horses with stress fractures often present as a nondescriptive lameness with no palpable findings. These horses are often at least a grade 3/5 lame initially but improve quickly. Manipulation of the elbow and shoulder often exacerbate the lameness.

### *Diagnosis*

- In most cases, radiography is used to confirm the fracture and define the configuration; the exception is stress fractures. Usually lateral-medial and slightly oblique lateral-medial views can be taken in the standing, sedated

**Figure 6.79.** Nuclear scintigraphic examination of the humerus with increased isotope uptake indicative of a distal metaphyseal stress fracture. Courtesy of Dan Burba.

horse, but foals can often be restrained in lateral recumbency.
- Cranial caudal views of the humerus are more difficult to obtain and may require general anesthesia, depending on the size of the animal.
- Nuclear scintigraphy is usually needed to document stress fractures within the humerus (Figure 6.79). Radiography may identify callus formation along the affected cortex, particularly in chronic cases (Figure 4.11).

### *Treatment*

- Currently three options are considered when managing a horse with a humeral fracture: nonsurgical management with prolonged stall rest (three to six months), surgical stabilization, and euthanasia. The majority of older horses with complete, displaced diaphyseal fractures are euthanized.
- Nonsurgical management is recommended to treat stress fractures and most minimally displaced fractures, regardless of the location. Some complete, displaced mid-diaphyseal fractures can be treated conservatively because the heavy musculature surrounding the humerus helps to stabilize the fracture while it heals. A better outcome is usually obtained in foals, compared with adults (Figure 6.80).
- Surgical management using intramedullary pinning, bone plating, and interlocking intramedullary nailing have been used successfully to treat young horses and ponies with humeral fractures.
- Bone plating using the cranial approach to the humerus is the most commonly used technique to repair humeral fractures. In general, the smaller the horse, the greater the chance for success for internal fixation.
- Small open fractures of the deltoid tuberosity or greater tubercle are often best removed surgically, especially if concurrent infection is present.

### *Prognosis*

- The prognosis for stress fractures, nondisplaced complete or incomplete fractures, or minimally displaced complete nonarticular fractures managed conservatively appears very good.
- The prognosis for complete displaced nonarticular fractures is guarded but appears better for horses managed conservatively. Diaphyseal fractures that are spiral and oblique with minimal overriding are the best candidates for nonsurgical treatment (Figure 6.80).
- The prognosis for fractures involving the greater tubercle and deltoid tuberosity is also very good with either conservative or surgical treatment.

## BICIPITAL (INTERTUBERCULAR BURSA) BURSITIS

### *Introduction*

- The bicipital bursa is located between the bilobed tendon of origin of the biceps brachii muscle and the M-shaped tubercles at the cranioproximal aspect of the humerus. Although uncommon, communication can exist between the shoulder joint and the bicipital bursa
- Inflammation of the intertubercular (bicipital) bursa can occur in horses of any age, breed, or sex and can be septic or nonseptic.

### *Etiology*

- Trauma to the cranial surface of the shoulder region is the most common cause of a primary bursitis.
- Falls or slips that result in flexion of the shoulder with extension of the elbow also may be a cause.

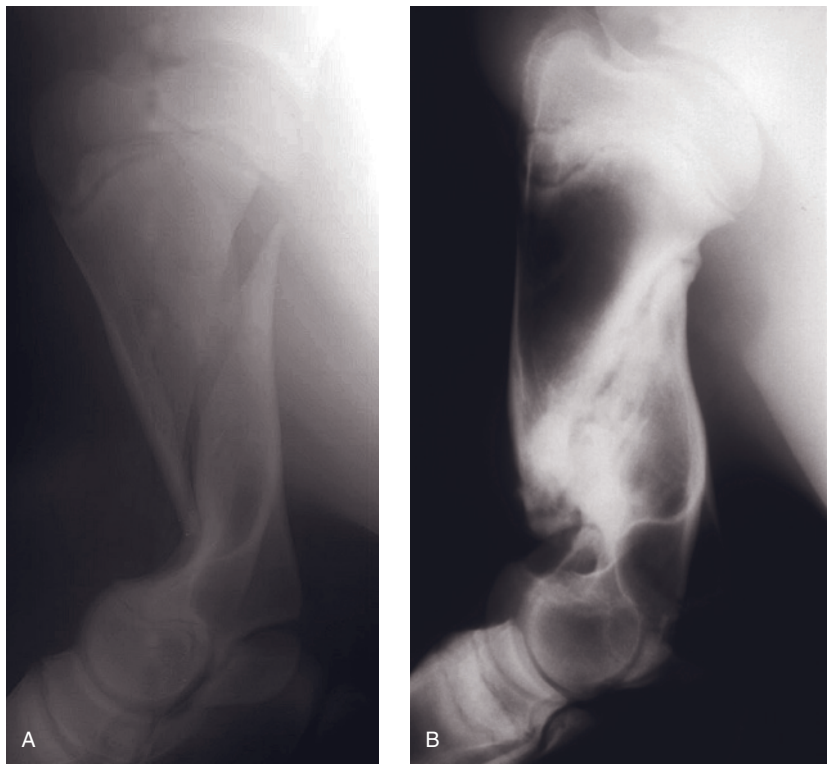



**Figure 6.80.** (A) Oblique lateral view of a spiral nonarticular fracture of the midhumerus. (B) Lateral view of a healing spiral nonarticular fracture of the midhumerus treated conservatively for five months.

- Infection, either from an open or penetrating wound or from hematogenous spread to the bursa, also may occur. Septic bicipital bursitis may be associated with "joint ill" in foals.

### *Clinical Signs*

- Swelling over the cranial aspect of the shoulder may be evident in acute cases. In more chronic cases, generalized shoulder and pectoral muscle atrophy may be seen.
- Digital pressure applied over the biceps tendon and bursal region and manipulation of the shoulder region in flexion and extension usually result in a prominent painful response.
- The lameness can be severe in acute cases and is often characterized by a shortened cranial phase of the stride, a decrease in the height of the foot flight arc, reduced carpal flexion, and a fixed shoulder appearance during movement.
- Intrasynovial anesthesia of the bursa can be performed to document the bursa as the site of the lameness.

### *Diagnosis*

- Both radiography and ultrasonography of the shoulder region are often needed to completely assess the bicipital bursa. The flexed cranioproximal-caudoproximal (skyline) projection of the cranial shoulder is useful in identifying lesions associated with the tubercles of the proximal humerus. If a wound or draining tract is present, centesis with contrast material may help assess communication with the bursa.
- Ultrasound examination of the biceps tendon, bursa, and bicipital groove can be very informative, especially when radiographs appear normal. Ultrasound changes associated with bursitis have included edema or hemorrhage in the biceps tendon or bursa, disruption of the tendon architecture with peritendinous

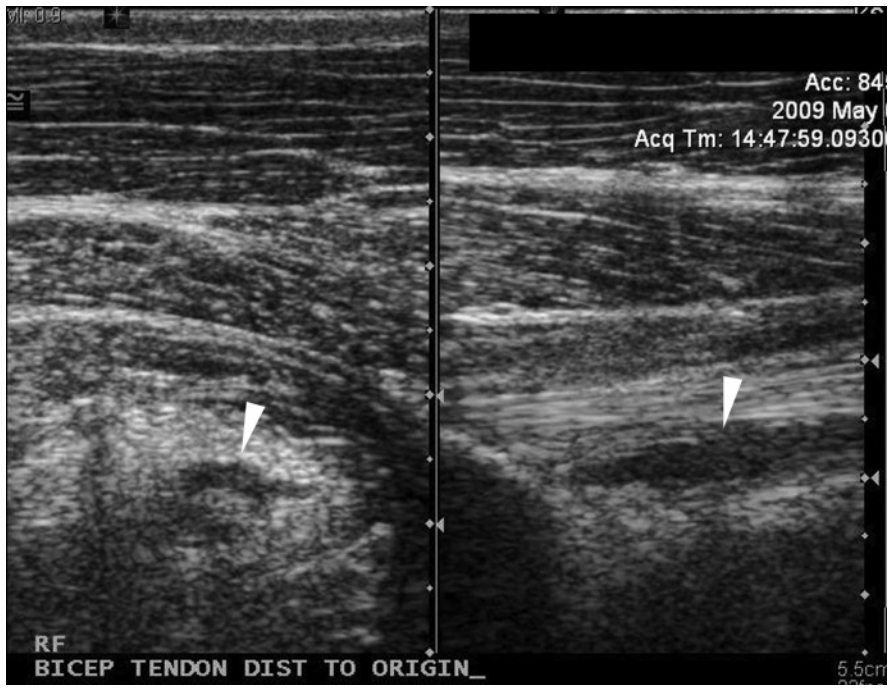



**Figure 6.81.** Core lesion of the medial branch of the biceps tendon just distal to the origin of the biceps brachii muscle (arrowheads). Courtesy of Jeremy Hubert.

thickening, an irregular surface of the bicipital groove, and hyperechoic material in the bicipital bursa (Figure 6.81).

### *Treatment*

- Noninfectious bursitis without radiographic evidence of a fracture or pathology on the cranioproximal aspect of the humerus usually respond favorably to rest and a controlled exercise program, NSAIDs, and intrasynovial injection of the bursa with corticoids and HA. Application of topical NSAIDs may also assist in reducing the pain.
- Shockwave therapy of bicipital tendon lesions may be used, but the success of this treatment is unknown.
- Ultrasound-guided intralesional treatment of tendon injuries may be performed with PRP or stem cells.
- Bursitis from either a fracture, osteitis of the proximocranial aspect of the humerus (Figure 6.82), or sepsis generally requires surgery to resolve the problem. Incisional as well as arthroscopic approaches to the bursa have been described, but bursoscopy is recommended.

### *Prognosis*

- Acute cases of nonseptic bursitis in the absence of a fracture or tendon injury often respond favorably to conservative treatment.
- Conservative therapy for more chronic cases of nonseptic or septic bursitis appears less satisfactory.
- The prognosis is considered poor for horses with chronic septic bursitis treated conservatively. Surgical intervention with debridement, lavage, and appropriate antimicrobial therapy is thought to improve the prognosis.

## OSTEOCHONDROSIS (OCD) OF THE SCAPULOHUMERAL (SHOULDER) JOINT

### *Introduction*

- This condition is most frequently diagnosed in weanlings and yearlings 6 to 12 months of age, but may be seen in horses of any age.
- Males appear to be more commonly affected than females and no specific breed predilection has been identified.
- The condition is the most debilitating form of OCD and is commonly associated with significant secondary OA.

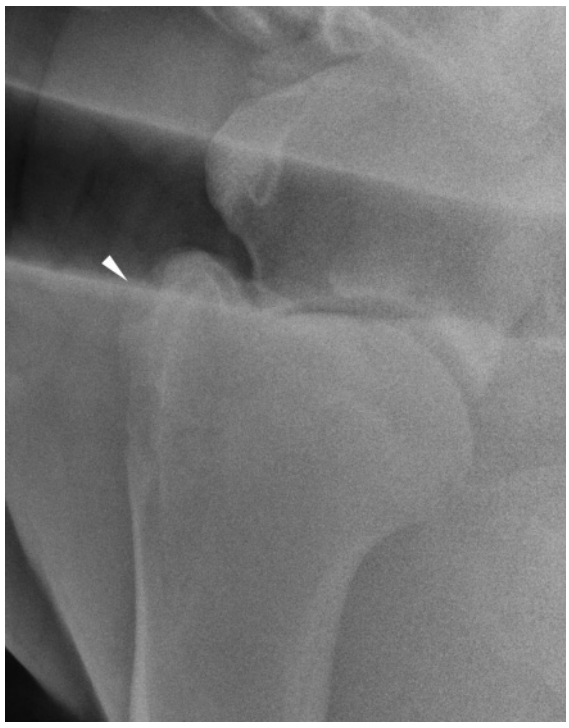


**Figure 6.82.** Osseous changes on the tubercles of the cranio-proximal humerus (arrowhead). Infiltration of lidocaine into the bursa resolved the lameness in this case. Courtesy of Jeremy Hubert.

- Lesions may be located on the humeral head, glenoid of the scapula, or both the glenoid and humeral head (most common).

### *Etiology*

- In most cases OCD is considered developmental in origin.
- Some SCLs of the glenoid may be associated with trauma and not OCD, but this is difficult to document.

### *Clinical Signs*

- Most cases present with a history of moderate to severe forelimb lameness with an insidious onset.
- Atrophy of the muscles associated with the shoulder region is a common finding in chronic cases.
- A smaller foot with a higher heel and excessive toe wear is also commonly observed in the foot of the affected limb.
- Direct, firm pressure with the thumb just cranial to the tendon of the infraspinatus muscle over the shoulder joint may elicit a painful response.
- The lameness is often characterized by a shortened cranial (extension) phase of the stride and a delay in limb protraction. A prominent shoulder lift, reduced carpal flexion, and limb circumduction is often seen in the most severely affected horses. Upper limb extension/flexion is usually painful and increases the signs of lameness.
- Intrasynovial anesthesia can be used to localize the lameness to the shoulder region.

### *Diagnosis*

- Radiographs are necessary to definitively diagnose the lesion. The most common radiographic findings include (Figure 6.83):
  1. Flattening and indentation of the caudal aspect of the humeral head
  2. Alterations in the contour of the glenoid cavity with a subchondral cystic radiolucency
  3. Osteophytes at the caudal and cranial aspect of the glenoid cavity
  4. Subchondral bone sclerosis
  5. Remodeling of the humeral head and glenoid cavity
- Arthroscopy may be needed to make a definitive diagnosis in cases in which the lameness is localized to the shoulder but no lesion is identified radiographically.

### *Treatment*

- Arthroscopic debridement of lesions is currently the recommended treatment for most horses. With very large lesions or those with severe degenerative changes, the prognosis is poor and surgery is usually not recommended (Figure 6.84).
- Rest and confinement may be considered for horses not intended for athletic performance that have mild to moderate radiographic changes.
- Euthanasia may be required in horses with severe lameness and radiographic abnormalities with a very poor prognosis.

### *Prognosis*

- Generally the prognosis for rest or surgery is considered guarded, but is dependent on the intended use of the horse and the severity of the OCD lesion.

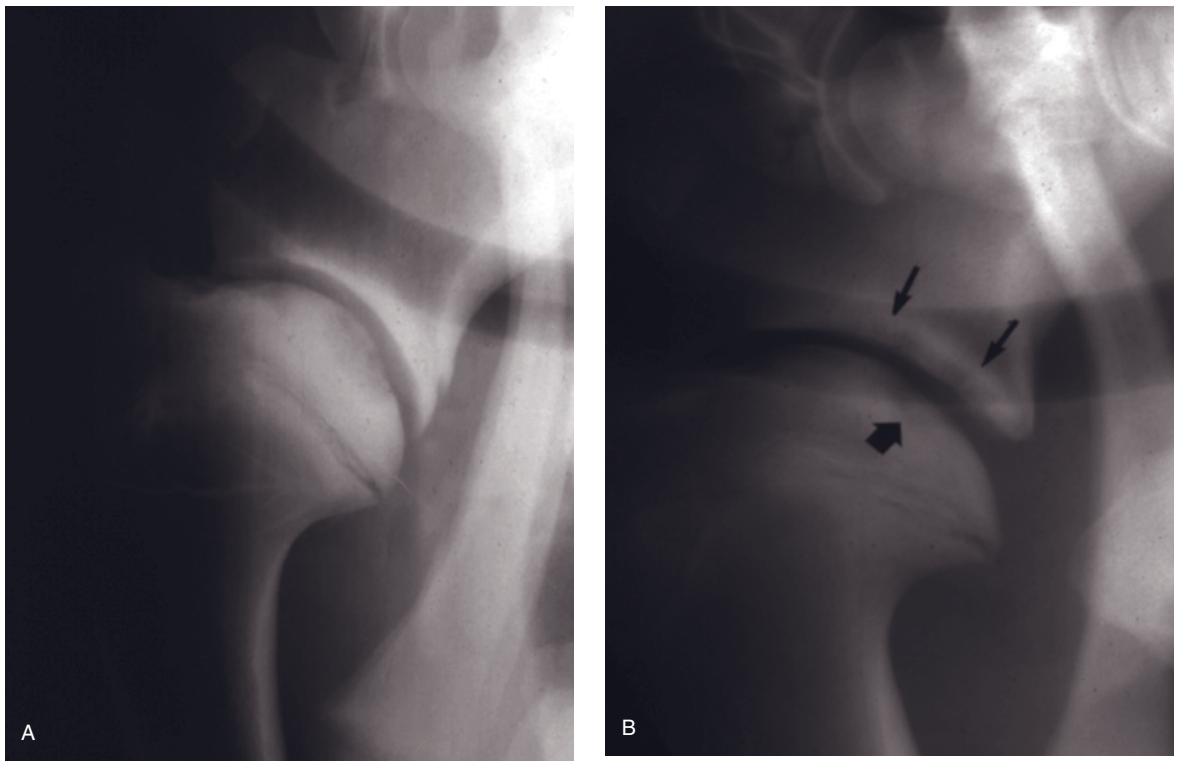



**Figure 6.83.** Radiographic manifestations of OCD of the shoulder. (A) The defect in the humeral head is the only lesion. (B) Defect in both the humeral head (large black arrow) and glenoid of the scapula (small black arrows). Courtesy of CW McIlwraith.

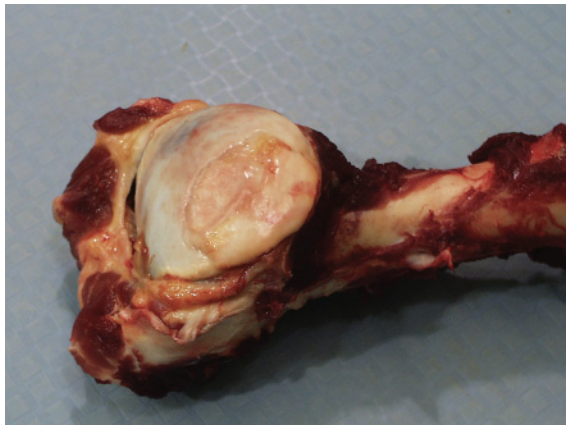


**Figure 6.84.** Post-mortem view of a large OCD lesion of the caudal humeral head that had severe radiographic signs of OA.

- Horses with mild to moderate OCD have the best prognosis and those with severe lesions usually remain lame.
- Currently the prognosis is about 50-50 following arthroscopic debridement, but depends on case selection.

## SUPRASCAPULAR NERVE INJURY (SWEENY)

### *Introduction*

- The term "sweeny" has been defined as atrophy of the shoulder muscles in horses and is a commonly used synonym for suprascapular nerve paralysis
- Suprascapular nerve injury resulting in atrophy of the supraspinatus and infraspinatus muscles and shoulder joint instability can affect any age or breed of horse.
- The condition was originally reported to commonly affect draft breeds and was believed to be associated with repeated trauma to the shoulder region from poorly fitted harness collars.

### *Etiology*

- The suprascapular nerve originates from the sixth and seventh cervical spinal segments and passes via the brachial plexus to innervate the supraspinatus and infraspinatus muscles. As the nerve reflects around the cranial edge of the scapula, it passes beneath a small but strong tendinous band and is susceptible to direct trauma and compression against the underlying bone.
- It has been suggested that chronic neuronal injury may make the suprascapular nerve more susceptible to acute trauma or that spontaneous development of the condition, without a traumatic insult, may be possible.

### *Clinical Signs*

- The clinical signs vary, depending on the extent of the nerve damage and the duration of the condition prior to examination.
- Horses with acute injuries often exhibit severe pain and are reluctant to bear weight on the affected limb. As the pain subsides and the horse begins to bear weight, a pronounced lateral instability (excursion) of the shoulder joint (shoulder slip) during weight-bearing is observed.
- Shoulder instability is due to loss of the stabilizing function of the supraspinatus and infraspinatus muscles, which serve as the major lateral support for the shoulder. The outward excursion of the scapula may cause intermittent stretching of the suprascapular nerve, leading to continued trauma and perpetuation of the paralysis.
- The progression of the condition depends on the amount of damage to the nerve. Horses with temporary neuropraxia may regain normal function within a few days to weeks, whereas those with true nerve damage will develop significant muscle atrophy as early as 10 to 14 days after injury (Figure 6.85).

### *Diagnosis*

- A presumptive diagnosis of suprascapular nerve injury can be made from the clinical signs and the history of trauma.
- Radiographs of the region should be obtained to rule out a fracture, OA, or luxation of the shoulder joint.
- Electromyographic evaluation (EMGs) of the muscles can confirm selective suprascapular nerve injury but are not accurate until a minimum of seven days after injury. EMG findings of denervation of muscles supplied by other branches of the brachial plexus should prompt further evaluation of other neurologic problems.

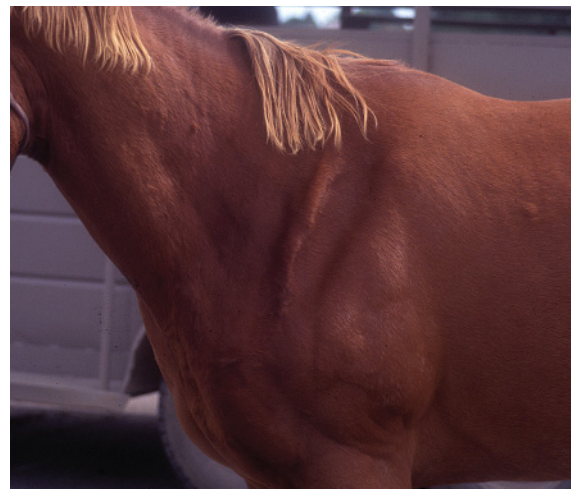


**Figure 6.85.** Prominent atrophy of the supraspinatus muscle is evident in this horse with suprascapular nerve injury.

### *Treatment*

- Initial treatment is directed toward reducing inflammation in the region of the nerve. Stall rest, systemic NSAIDs, and topical icing and anti-inflammatories (Surpass®) are usually indicated.
- Conservative treatment consisting of confinement with or without injection of the shoulder joint may be used until shoulder joint stability returns.
- Based on peripheral nerve regeneration rates (1 mm/day), nerve function should return in 10 to 12 weeks. In one study the mean time for resolution of the gait abnormality was 7.4 months and 7/8 horses evaluated had complete resolution.
- Surgical decompression or the "scapular notch procedure" may be considered in patients that continue to exhibit signs of suprascapular nerve dysfunction after 10 to 12 weeks. Entrapment of the suprascapular nerve by scar tissue between the overlying ligament and the cranial border of the scapula is thought to occur in these horses.

- Some surgeons suggest that faster resolution of the clinical signs can be obtained with surgical decompression earlier in the disease process (before 10 weeks).

### *Prognosis*

- The prognosis for return to soundness is reported to be favorable for both conservative and surgical treatments. Seven of eight horses treated conservatively became sound and 18/20 horses treated surgically became sound in two different reports.
- A severe complication associated with the surgery is subsequent fracture of the glenoid.
- Horses that have severe muscle atrophy prior to surgery may not regain normal muscle mass.

## FRACTURES OF THE SUPRAGLENOID TUBERCLE (TUBEROSITY)

### *Introduction*

- The supraglenoid tubercle serves as the proximal attachment for the biceps brachii muscle and two glenohumeral ligaments which support the scapulohumeral joint.
- Fractures of the supraglenoid tubercle (SGT) can occur in a variety of breeds, are often simple and intra-articular, and occur in young horses.

### *Etiology*

- Fracture of the SGT is most frequently associated with trauma (falling or direct blows) to the cranial shoulder region.
- Overflexion of the shoulder leading to increased tension on the biceps brachii and coracobrachialis tendons that attach to the SGT also has been proposed as a cause.
- They may also occur subsequent to the scapula notch procedure for decompression of the suprascapular nerve.

### *Clinical Signs*

- A history of trauma resulting in severe lameness that improves rapidly is common.
- Swelling over the point of the shoulder and pain on palpation are usually seen in acute cases. Crepitus also may be appreciated in some cases.
- Lameness severity may vary depending on duration, but the cranial phase of the stride is markedly shortened. A 3 to 4/5 lameness is typical in the acute phase.
- Varying degrees of muscle atrophy are usually apparent in chronic cases and upper limb flexion tests usually worsen the lameness.

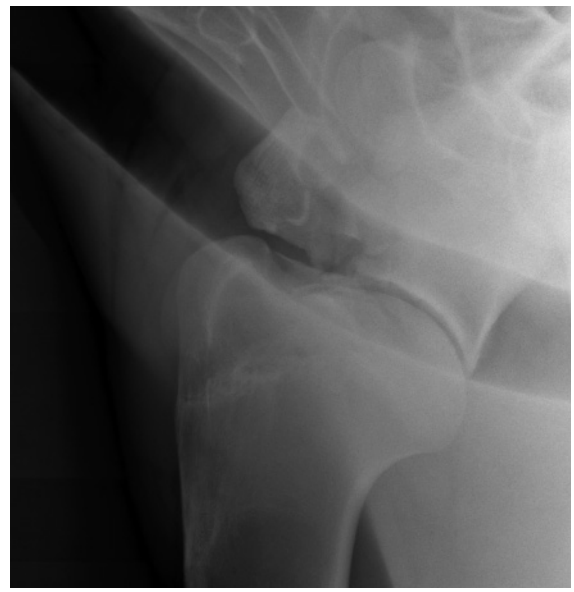


**Figure 6.86.** Fracture of the supraglenoid tubercle of the scapula with moderate displacement.

### *Diagnosis*

- Radiography is used to make a definitive diagnosis of the fracture. The size and type of fracture (simple vs. comminuted) can vary, but generally the fracture is displaced due to the pull of the biceps tendon (Figure 6.86).

### *Treatment*

- Treatment options include conservative management, removal, or internal fixation with lag screws.
- Conservative management (confinement for three to four months) can be used in horses with nonarticular or minimally displaced intra-articular fractures.
- Surgical excision of the SGT appears to be best suited for horses with chronic or comminuted articular fractures. Removal of the fragment is thought to decrease the pain caused by movement of the fracture and prevent further joint damage.

- Internal fixation is best for large articular fractures that can support screws. The goal of surgery is to prevent the development of secondary OA caused by joint incongruity. Combinations of interfragmentary compression with lag screws and tension band wires are usually used.

### *Prognosis*

- In general, the prognosis for horses with nonarticular fractures is much better than for those with articular fractures.
- A good prognosis for return to performance can be expected for horses with nonarticular fractures or minimally displaced articular fractures managed conservatively.
- The prognosis following surgical excision of the fracture is considered to be better than for conservative management, but this depends on the fracture type and complications that may occur (i.e. suprascapular nerve injury).
- The prognosis for internal fixation appears best if a combination of internal fixation and complete transection of the biceps brachii tendon is used.

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7

CHAPTER

# Common Conditions of the Hindlimb

## DISTAL HINDLIMB AND FOOT

Because of the anatomical and functional similarities between the distal forelimb and hindlimb in the horse, many of the same lameness conditions occur distal to the carpus and tarsus. However, the frequency of these lameness problems may differ between the forelimb and hindlimb, and these differences have been described in chapters 5 and 6. Therefore, the reader is referred to chapters 5 (Common Conditions of the Foot) and 6 (Common Conditions of the Forelimb) for discussions of lameness conditions that affect the distal hindlimb and foot.

## DISTAL TARSAL OSTEOARTHRITIS (OA)

### *Introduction*

- Distal tarsal OA is often referred to as "bone spavin" and is the most common cause of hock lameness in horses. It usually involves the distal intertarsal (DIT) and tarsometatarsal (TMT) joints, but may occasionally affect the proximal intertarsal (PIT) joint.
- Horses with occult spavin, blind spavin, or tarsitis are described as having similar clinical features of distal tarsal OA but without radiographic abnormalities. Scintigraphic evidence of distal tarsal inflammation may be present in these horses.
- Tarsitis and distal tarsal OA are most likely different stages of the same disease process and are most frequently observed in jumping horses; dressage and event horses; horses that pull carts; and Western performance horses used for reining, roping, and cutting.
- Icelandic horses also appear to be prone to the disease.

### *Etiology*

- Repetitive compression and rotation of the tarsal bones and excessive tension on the associated ligaments related to the type and intensity of exercise are thought to contribute to the disease.
- Shear forces due to asynchronous movement of the tarsal bones also may predispose the animal to distal tarsal OA.
- In Icelandic horses the type and severity of work did not affect the incidence, whereas tarsal angle and age were important. The incidence was also related to certain sires.
- Sickle and cow-hocked conformation cause greater stress on the medial aspect of the tarsus and contribute to the disease.
- Congenital malformation of the tarsal cuboidal bones contributes to the disease, as do specific traumatic injuries to the distal tarsus.

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

### Clinical Signs

- There is usually a gradual onset of lameness or reduced performance, especially if affected bilaterally.
- The lameness is usually worse when the horse is first used (horses with mild spavin tend to warm out of the lameness) but in severe cases exercise will worsen the lameness.
- A mild to moderate hindlimb lameness is usually observed, which worsens following hindlimb flexion. Reduced arc of the foot flight, reduced flexion of the hock, carrying the limb axially and then stabbing the limb outward, and wearing of the toe are a few specific gait abnormalities that may be present with distal tarsal pain.
- Horses with distal tarsal OA often have other musculoskeletal abnormalities (back pain and forelimb foot pain) that must be investigated.
- Thickening or enlargement of the medial aspect of the hock may be observed visually in horses with advanced disease. Palpation of the enlargement is usually painful (Figure 3.35).
- Some horses without visual enlargement of the tarsus may be positive to the "Churchill test," which suggests pain in the distal tarsal region. See Chapter 3 for details of the Churchill test.
- Tarsal flexion or full limb flexion tests usually worsen the lameness and should be performed bilaterally since the disease can affect both tarsi.
- IA anesthesia of one or both distal tarsal joints is the most specific and expedient method to document the site of the lameness.

### Diagnosis

- The diagnosis is often made based on the combination of clinical findings, response to IA anesthesia, and radiographic abnormalities. Radiographs or clinical findings alone are not reliable as the only method of diagnosis.
- Radiographic changes consistent with distal tarsal OA can be variable but include marginal bone lysis or irregularity, osteophyte and/or enthesiophyte formation, joint space narrowing, subchondral bone lysis or sclerosis, and varying degrees of ankylosis (Figure 7.1).
- Radiographic evidence of OA usually begins on the dorsomedial surfaces of the TMT and DIT joints.
- Few adult performance horses have "normal" tarsal radiographs, and some horses with significant radiographic changes may not be lame. Therefore, some clinicians rely heavily on the clinical findings.
- Scintigraphy can be used to accurately detect distal tarsal inflammation and is useful when the diagnosis is complicated by multiple problems or difficulty blocking the joints (Figure 7.2).

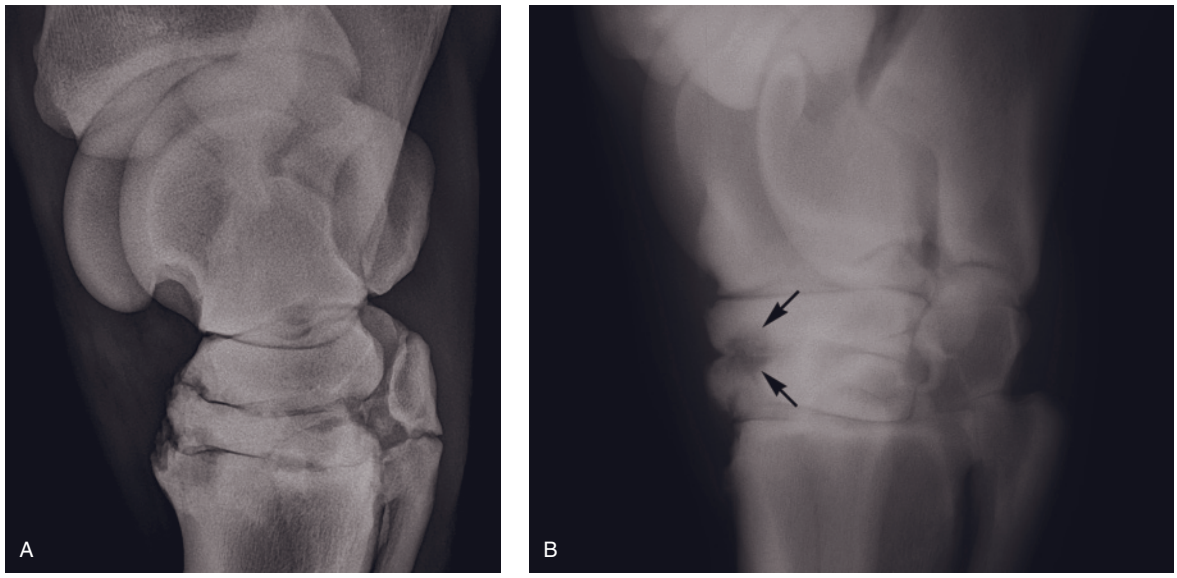


**Figure 7.1.** (A) Dorsomedial to plantarolateral oblique radiograph of a horse with severe OA of the TMT and DIT joints. These joints do not appear to be progressing toward ankylosis. (B) Dorsolateral-plantaromedial oblique radiograph of a horse with subchondral bone lysis of the DIT joint that is causing significant lameness.

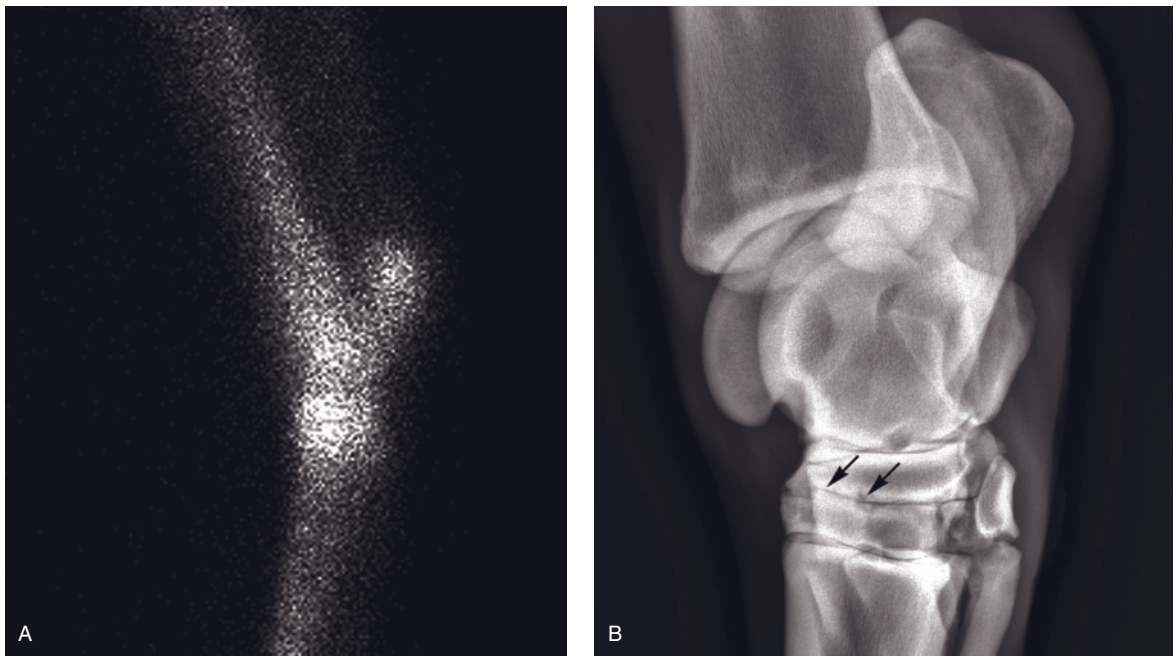


**Figure 7.2.** Lateral scintigram (A) and radiograph (B) of the tarsus of a horse with distal tarsal OA. There is diffuse uptake within the distal tarsal joints and joint space narrowing and lysis within the DIT joint (arrows) visible on the radiograph. Courtesy of Jeremy Hubert.

### *Treatment*

- Horses with mild to moderate lameness and radiographic changes usually respond favorably to a short period of reduced activity, systemic NSAIDs, corrective shoeing, and IA medication. Horses with no radiographic abnormalities may not require repetitive IA treatment.
- Supporting systemic therapies such as IM PSGAGs, IV HA, polyglycan, or oral nutraceuticals may be used to reduce the need for IA medication.
- Hoof management is aimed at reducing the rotation/shear forces at the tarsus by easing break-over and removing shoe extensions (i.e., trailers). One approach is to balance the foot and achieve a dorsal hoof angle 1° to 2° steeper than the pastern, and then square or roll the toe.
- Initial IA treatment usually consists of a corticosteroid plus HA combination. Many clinicians use triamcinolone in horses with minor signs of OA and methylprednisolone acetate (MPA; Depo-Medrol®) in horses with more advanced signs of OA.
- Horses with advanced disease are the most problematic to treat. Some may respond to the same treatment as described for horses with less severe disease but may require more frequent IA treatment and a change in career.
- Joint ankylosis or arthrodesis may be required in severe disease if other treatments fail. Exercise-facilitated ankylosis does not work in most cases because spontaneous ankylosis rarely occurs.
- Methods to induce chemical arthrodesis include IA monoiodacetate (MIA) and 70% ethanol. MIA is not readily available and rarely used. Ethanol (3 to 4 ml) is a relatively new treatment but has shown some promise in promoting joint arthrodesis in a few reports.
- Methods to surgically arthrodese the distal tarsal joints include IA laser treatment, IA drilling, and IA drilling plus stabilization with a small bone plate. Variable success rates have been reported with these techniques but no technique appears superior to the others.

### *Prognosis*

- The prognosis for horses with bone spavin often depends on the severity of lameness and radiographic abnormalities.
- Horses with mild disease often have a good prognosis, whereas horses with more advanced disease have a guarded prognosis. However, a

more accurate prognosis can usually be made after the response to the initial therapy is known.
- Horses with advanced disease requiring arthrodesis techniques have approximately a 50-50 chance of returning to athletic use.

## OSTEOCHONDRITIS DISSECANS (OCD) OF THE TARSOCRURAL JOINT

### *Introduction*

- Young horses of any breed with effusion of the tarsocrural joint should be suspected of having tarsocrural OCD.
- Many of these horses are not lame and the lesion may be found on routine radiographs taken before a sale.
- Tarsocrural OCD is quite common in Standardbreds but is found in nearly all breeds.
- Typical sites for OCD fragments to occur are the distal intermediate ridge of the tibia (DIRT), lateral trochlear ridge, medial malleolus, and medial trochlear ridge. OCD lesions in more than one location can be found in the same tarsocrural joint.

### *Etiology*

- OCD is a developmental abnormality that is defined as a focal disturbance of endochondral ossification with a multifactorial etiology.
- The most commonly cited etiologic factors are heredity, rapid growth, anatomic conformation, trauma, and dietary imbalances; however, only heredity and anatomic conformation are well supported by the literature.
- Trauma within the tarsocrural joint may be difficult to differentiate from true OCD because some traumatic events can destabilize a pre-existing OCD fragment.

### *Clinical Signs*

- Non-painful effusion of the tarsocrural joint is by far the most common presenting complaint (Figure 3.32).
- If periarticular or regional edema or swelling beyond the tarsocrural joint are present, another problem should be suspected.
- Lameness may be absent or mild; severe lameness is not consistent with this diagnosis.
- A reduced flexion angle of the tarsus may be observed in horses with excessive synovial effusion. A hindlimb flexion test may increase the lameness slightly.

### *Diagnosis*

- The diagnosis is usually confirmed with a complete radiographic examination of the tarsus. A slightly oblique dorsoplantar view may be needed to highlight a lesion of the medial malleolus (Figure 7.3).
- The radiographic abnormalities may consist of subchondral bone lucency, a partially ossified "flap," an osteochondral fragment, or multiple loose bodies within the joint (Figure 7.4).
- The opposite tarsus should be radiographed because approximately half of affected horses have similar contralateral lesions.
- A small percentage of horses may not have radiographically apparent subchondral bone involvement and require arthroscopy to make the diagnosis.

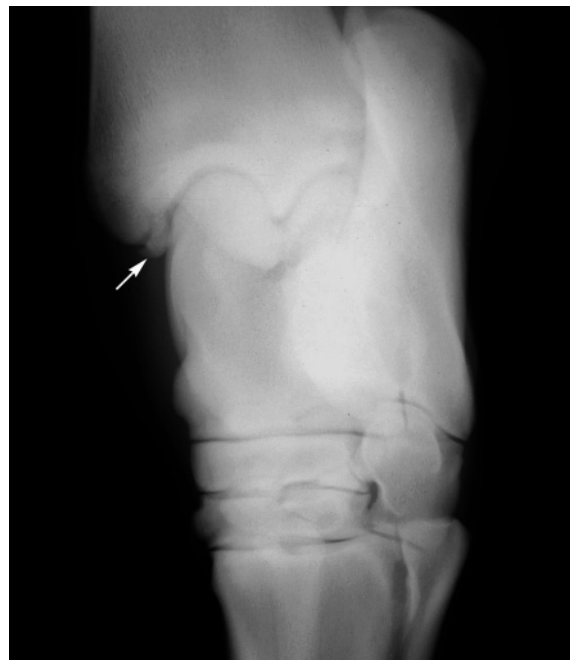

**Figure 7.3.** Dorsolateral-plantaromedial radiograph of the tarsus demonstrating a medial malleolar OCD lesion (arrow). The fragment is often located on the axial surface of the medial malleolus adjacent to the medial trochlear ridge.

### *Treatment*

- Most horses benefit from arthroscopic removal of the OCD fragment. Lesion location or type has not been correlated to differences in outcome.
- Prolonged effusion of the tarsocrural joint before surgery usually increases the chance that the bog spavin will persist after fragment removal.

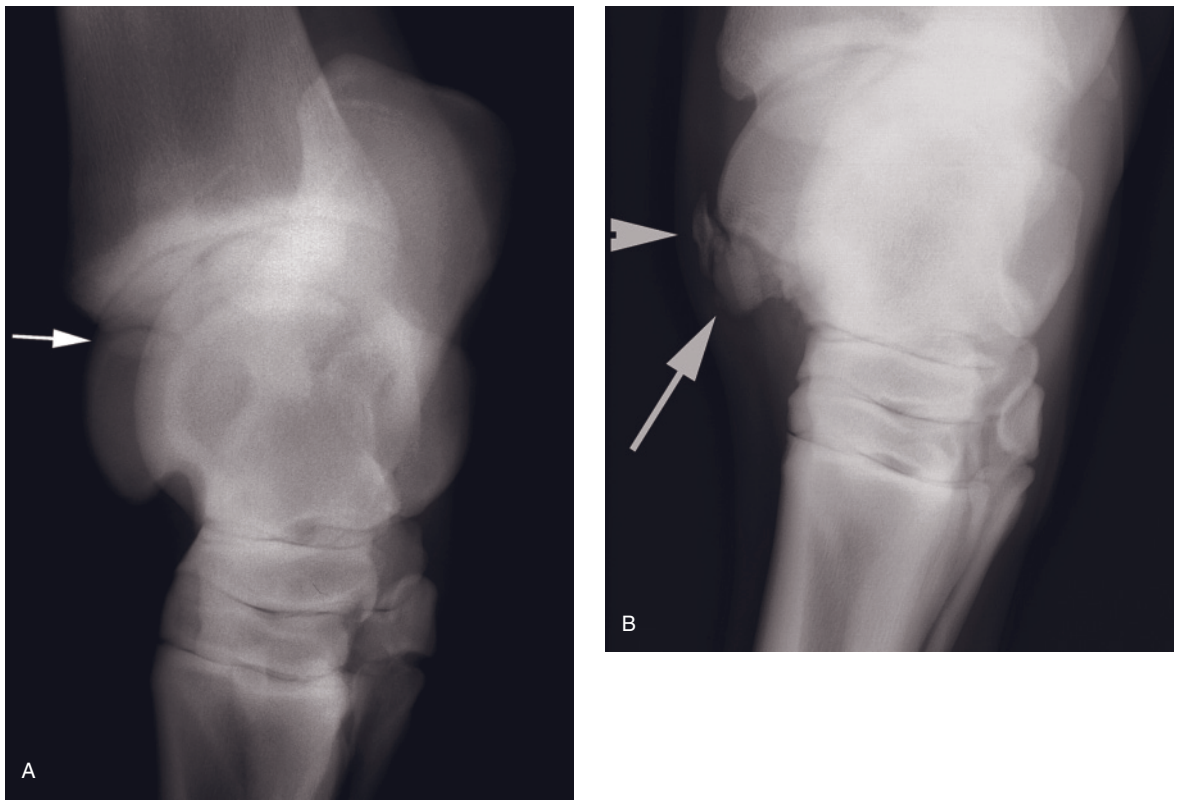


**Figure 7.4.** Dorsomedial plantarolateral radiographs demonstrating an OCD lesion of the distal intermediate ridge (A) of the tibia (DIRT) and the lateral trochlear ridge (B) of the talus (arrows). A fragment from the lateral trochlear ridge lesion has become dislodged from the parent bone. Courtesy of Ken Sullins.

- Horses with radiographic lesions but no clinical signs may not need to be treated, but clinical signs can always appear during training.
- In foals, some radiographically evident lesions will correct themselves with age. In a series of Standardbred foals, all ossification and subchondral bone abnormalities observed before 8 months of age resolved spontaneously.

### *Prognosis*

- The prognosis for athletic activity after arthroscopic debridement of tarsocrural OCD is good (75% to 80% perform successfully after surgery). Lesion location and unilateral vs. bilateral lesions do not appear to alter the prognosis.
- Effusion appears to be less likely to resolve following surgery on lateral trochlear ridge or medial malleolus lesions.
- Superficial cartilage fibrillation does not alter the prognosis, but more severe cartilage degeneration or erosion decreases the success of surgery.

## SLAB/SAGITTAL FRACTURES OF THE CENTRAL OR THIRD TARSAL BONES

### *Introduction*

- These fractures are most commonly a racing injury in a variety of breeds, but Standardbreds (left hindlimb) are over represented.
- The central tarsal bone tends to fracture along the dorsomedial aspect, whereas the third tarsal bone is most often affected dorsally or dorsolaterally (Figure 4.59).

### *Etiology*

- The distal tarsal bones are subjected to axial compression, torsional forces, and tensile forces during exercise. Their main function is to absorb concussion and neutralize these

twisting forces. When these bones are subjected to the even greater stress of racing speeds, fracture can occur.
- Asynchronous movement of the tarsal bones due to ligament damage or rapid changes in lead may result in the fracture.

### *Clinical Signs*

- Horses typically present with a history of acute onset of severe lameness that diminishes relatively quickly.
- Fractures of the third tarsal bone do not usually cause tarsocrural joint effusion, whereas effusion is common with slab fractures of the central tarsal bone.
- Heat and pain on palpation over the dorsolateral aspect of the distal row of tarsal bones may be appreciated.
- Most horses are very positive to the tarsal flexion test.

### *Diagnosis*

- Radiography is required to confirm the diagnosis, but some fractures (non-displaced) can be difficult to document on radiographs.
- Several oblique projections at different angles around the tarsus may be necessary to identify fragmentation and the specific position of these fractures (Figure 7.5).
- Radiographic evidence of OA usually appears with tarsal bone fractures of long duration.

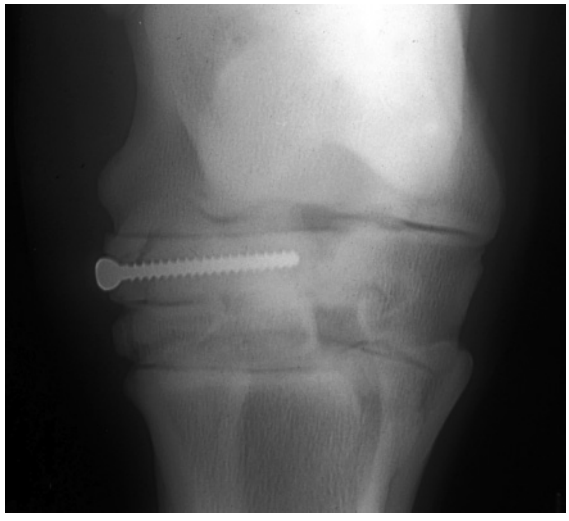

**Figure 7.5.** A slightly offset dorsoplantar radiograph of the tarsus after lag screw repair of a central tarsal bone fracture.

- With localization of the source of the lameness and no definitive radiographic lesion, scintigraphy or computed tomography may be needed to document the problem.
- Computed tomography also can be helpful to determine if a fracture is a single slab that is repairable or comminuted and not repairable (Figure 4.78).

### *Treatment*

- Acute fractures should be repaired with lag screws as early as possible to facilitate reduction and prevent degenerative changes in the joint. Fluoroscopy facilitates efficient, accurate placement of the screw or screws. The screw usually does not need to be removed.
- Comminuted fractures are usually not candidates for surgical repair.
- Satisfactory results have also been reported following stall confinement for six to eight months.

### *Prognosis*

- A combined 22 of 28 horses treated with surgery in several different reports have returned to athletic performance.
- The presence of OA at the time of surgery diminishes the prognosis for athletic function.
- The proportion of successful cases with central tarsal bone fractures is less than those with third tarsal bone fractures.
- With conservative therapy, 10 of 12 horses raced successfully.

## FRACTURES OF THE TIBIAL MALLEOLI

### *Introduction*

- Fractures of the lateral malleolus are more common and typically smaller than those involving the medial malleolus.
- A larger portion of the medial malleolus is intra-articular and therefore more likely to contribute to secondary damage within the tarsocrural joint (Figure 7.6).
- The lateral malleolus is largely invested in the joint capsule and collateral ligament; the intra-articular portion is limited to the actual joint surface.
- Many fractures of the lateral malleolus affect the most dorsal portion where the short collateral ligament attaches, but some affect the entire malleolus through to the caudal compartment of the joint.
- These fractures can be closed or open.

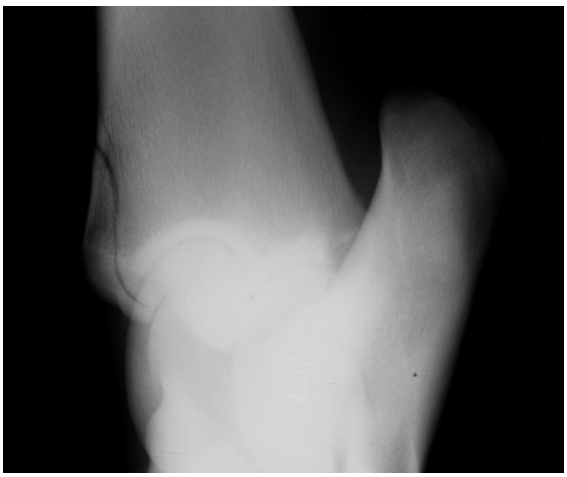

**Figure 7.6.** Oblique radiograph of a horse that had been kicked on the medial aspect of the tarsus. This articular fracture of the medial malleolus was repaired with lag screws.

### *Etiology*

- The tibial malleoli are fractured by direct trauma or avulsion of the collateral ligaments.
- The majority of horses acquire these injuries in a fall or from being kicked, with the lateral malleolus affected most commonly.

### *Diagnosis*

- Radiographs usually confirm the diagnosis. The fractures are usually demonstrated well in dorsoplantar or oblique radiographs.
- Concurrent collateral ligament injury is not uncommon and can be confirmed with ultrasonography.

### *Treatment*

- Removal is indicated when the fragments are small or comminuted, especially if the fracture is open. The fragment will usually need to be dissected from the collateral ligament but this usually does not affect the stability of the tarsocrural joint.
- Marginally sized fragments of the lateral malleoulus can be repaired by internal fixation but may split during the convalescent period and require removal.
- Three of four horses with lateral malleolar fractures treated by stall rest returned to athletic activity.
- Some large medial malleolus fractures can be repaired with lag screws (Figure 7.6) or treated conservatively.
- Much of the medial malleolus is well within the tarsocrural joint and accessible arthroscopically for removal of smaller fragments.

### *Prognosis*

- Overall good results are reported after removal of relatively small fragments from either the medial or lateral malleoli. Many of these fragments can be removed arthroscopically with minimal dissection into the joint capsule, especially those of the medial malleolus.
- A 50% return to performance following surgical repair with internal fixation of lateral malleolar fractures has been reported.
- The size of the fragment and secondary collateral ligament damage alter the prognosis in many cases.

## SUBLUXATIONS/LUXATIONS OF THE TARSAL JOINTS

### *Introduction*

- Subluxations/luxations of all four tarsal joints have been reported, but the PIT joint is most commonly affected (Figure 7.7).
- Substantial ligamentous damage around the tarsal joints must occur concurrently to permit subluxation/luxation of these joints.
- Subluxations/luxations of the tarsal joints are rarely open.

### *Etiology*

- A severe wrenching or twisting action that may occur from a sudden slip or fall is believed to be the cause.
- Kicks from other horses and entrapment in fixed objects such as fences or cattle guards also have been implicated.

### *Clinical Signs*

- Signs are usually quite obvious, with heat, pain, and swelling of the tarsus occurring in acute cases.
- The lameness is usually severe but usually improves with time. A limb deformity may be seen concurrently.
- On palpation, the limb is usually more moveable distal to the luxation, mostly in a medial-to-lateral direction.

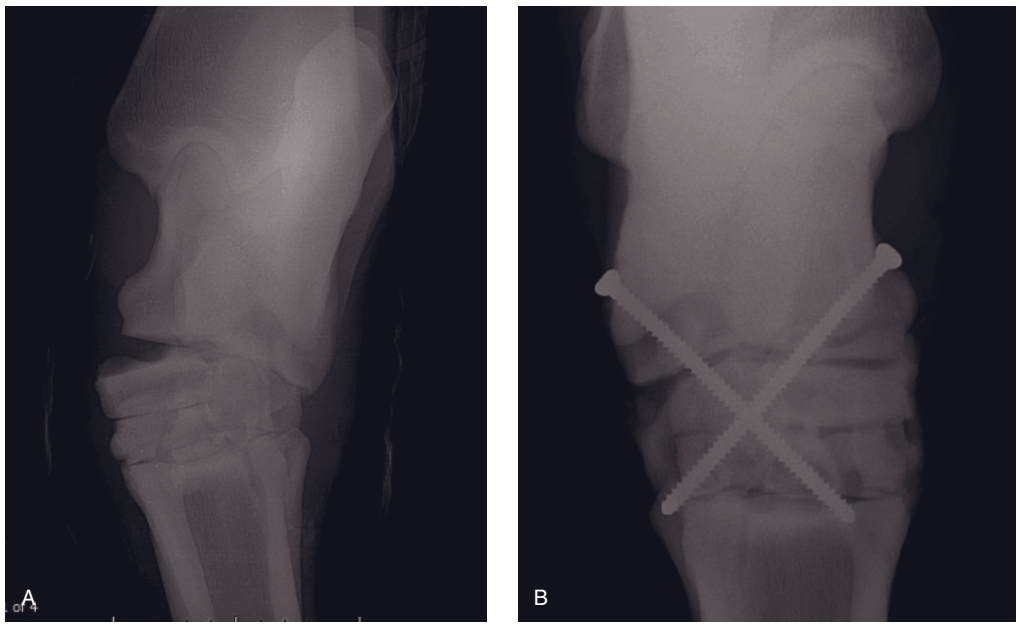


**Figure 7.7.** Pre- (A) and post-operative (B) dorsoplantar radiographs of a horse suffering a PIT joint luxation that was stabilized with cruciate screws and a full limb cast. Courtesy of Ken Sullins.

- Luxation of the tarsocrural joint is the most severe; the tibia is usually displaced distad and craniad, making it difficult or impossible to reduce.

### *Diagnosis*

- The exact location and extent of damage must be verified with radiographs. The dorsoplantar view is usually the most informative to evaluate the distal tarsal joints.
- A dorsoplantar stress radiograph holding the distal limb either medial or lateral is usually needed to document the exact location of the instability in the distal tarsal joints (Figure 7.8).
- Ultrasonography can be used to determine the severity of ligamentous damage associated with the luxation.

### *Treatment*

- Reduction and immobilization with a full-limb cast will suffice in many cases. The cast must be maintained for approximately six weeks, depending on the severity of the subluxation/luxation.
- Re-luxation of the distal tarsal joints can occur after cast removal and may require internal fixation.
- Internal fixation with a bone plate or angled screws may be required to achieve adequate stabilization of unstable luxations. Curettage of the articular cartilage from the surfaces of the distal tarsal bones facilitates ankylosis if the DIT and TMT joints are involved (Figures 7.7, 7.8).

### *Prognosis*

- The prognosis is reasonably good for simple luxation of the distal tarsal joints without fracture; however, the prognosis decreases if a fracture is present.
- Successful use of cast immobilization alone and internal fixation of distal tarsal and tarsocrural luxations have been reported.

## LUXATION OF THE SDFT FROM THE CALCANEUS

### *Introduction*

- Luxation of the SDFT from the point of the hock occurs when one of its fascial attachments (usually medial retinaculum) completely ruptures.
- This condition can be misdiagnosed as a capped hock or calcaneal bursitis, but careful

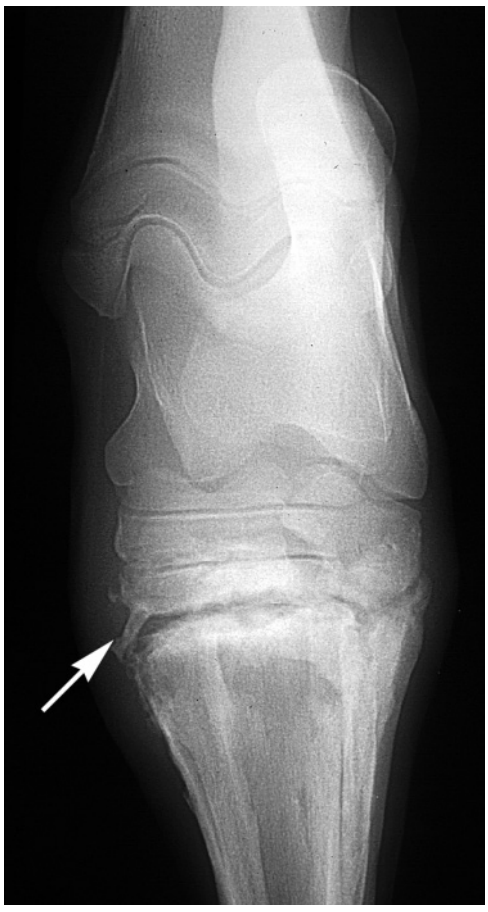

**Figure 7.8.** Dorsoplantar stress radiograph demonstrating luxation of the medial aspect of the TMT joint (arrow). A medial plate was used to stabilize the luxation and arthrodese the distal tarsal joints concurrently.

evaluation usually reveals the SDFT has displaced laterally (Figure 7.9). Concurrent calcaneal bursitis is inevitable with this injury.
- Occasionally the condition can occur bilaterally or can occur concurrently with a fracture of the calcaneus. They are rarely open.

## *Etiology*

- Trauma is the cause in most cases.
- Dislocation of the SDFT has been reported to occur as a racing injury, from simply bucking, or from unknown trauma.
- Rarely, it is associated with a calcaneal fracture.

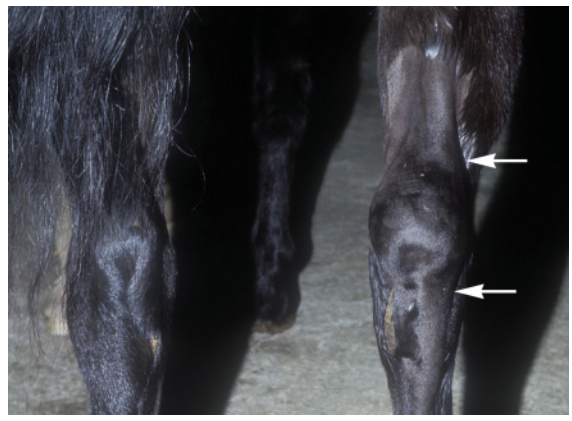

**Figure 7.9.** Caudal view of the tarsi in a horse with severe swelling within the right calcaneal bursa and evidence of lateral luxation of the right SDFT from the tuber calcanei (arrows).

## *Clinical Signs*

- Heat, pain, and swelling at the point of the hock are usually present in acute cases.
- Lameness can be severe in the acute stage but usually improves quickly. Some horses with chronic dislocation may not be lame at presentation but have swelling around the tuber calcanei.
- As the acute swelling subsides, the dislocation becomes more easily appreciated both visually and with palpation (Figure 7.9). The SDFT can usually be manually replaced over the tuber calcanei and then reluxates when the tarsus is flexed.
- Flexion of the tarsus is usually painful to the horse.

## *Diagnosis*

- The diagnosis is usually based on clinical findings (Figure 7.9).
- Radiographs should be taken to rule out a fracture.
- Ultrasonography can be used to determine the extent of damage to the medial retinaculum and to document the severity of the concurrent calcaneal bursitis.

## *Treatment*

- Treatment often depends on the degree of luxation and intended use of the horse.
- Partial dislocation of the SDFT should be treated conservatively with stall rest for three to six months.

- Some clinicians feel that horses with complete luxation do fine without surgery. Others recommend surgery. Too few numbers have been reported to determine which is best.
- Surgical options include debridement and repair of the torn fascia together with reinforcement of the suture line with mesh, or repair of the torn fascia without mesh and placing screws in the lateral aspect of the calcaneus to prevent re-luxation of the tendon. The limb is immobilized in a cast or sleeve cast for two to four weeks, after which the limb is supported in a Robert-Jones bandage.

### *Prognosis*

- The prognosis for breeding soundness or light pleasure riding is good with conservative or surgical treatment.
- Too few cases have been treated surgically with long-term follow-up to determine if surgical repair is superior to confinement alone.
- Some horses can perform normal work with the altered appearance and movement of the tendon at the calcaneus.

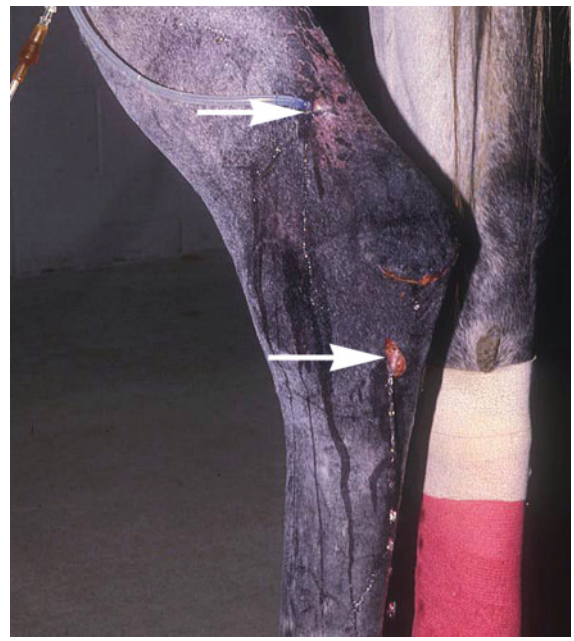

**Figure 7.10.** Lateral view of the calcaneal bursa with a drain placed to treat synovial infection. Note the proximal and distal extent of the calcaneal bursa (arrows) relative to the point of the hock.

## CAPPED HOCK/CALCANEAL BURSITIS

### *Introduction*

- Swelling at the point of the hock (tuber calcanei) is usually attributable to damage to the subcutaneous calcaneal bursa (capped hock) or to problems within the intertendinous calcaneal bursa located beneath the SDFT.
- The subcutaneous bursa at the tarsus is analogous to the bursa at the point of the olecranon but may communicate with the intertendinous bursa in about one-third of horses.
- The intertendinous calcaneal bursa is a true synovial cavity and problems within this anatomic structure are much more problematic than those within the subcutaneous bursa.
- The calcaneal bursa extends approximately 7 cm distally and 9.6 cm proximally relative to the tuber calcanei (Figure 7.10).

### *Etiology*

- Direct trauma to the point of the hock from a kick or the horse hitting a hard object such as a stall wall, fence, etc. is the most common cause of a capped hock. These injuries may be associated with a wound.
- Non-septic calcaneal bursitis is usually secondary to luxation of the SDFT from the tuber calcanei, damage to the attachment of the gastrocnemius tendon to the tuber calcanei, and nonseptic osteolytic lesions within the calcaneus.
- Septic calcaneal bursitis is nearly always secondary to penetrating wounds that enter the calcaneal bursa. Secondary osteomyelitis within the tuber calcanei is not uncommon (Figure 7.11)

### *Clinical Signs*

- A capped hock (nonseptic) is usually characterized by a soft, fluctuant swelling located directly at the point of the hock. Lameness may or may not be present, depending on the time since injury, but is usually minimal after a few days.
- Horses with nonseptic calcaneal bursitis usually have palpable effusion within the bursa (above or below the tuber calcanei) and the point of the hock is usually enlarged compared to the opposite calcaneus (Figure 3.39). These horses are often painful to direct palpa-

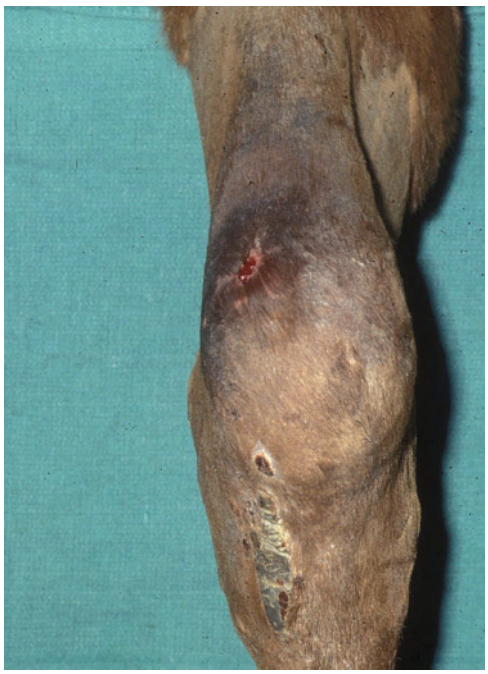

**Figure 7.11.** Caudal view of the calcaneus in a horse with synovial sepsis from a penetrating injury that occurred proximal to the tuber calcanei. The infection has resolved but the calcaneal bursa remains enlarged.

tion of the bursa, lame at the trot, and very positive to tarsal flexion.
- Horses with septic calcaneal bursitis are usually very lame, have severe swelling around the point of the hock, resent firm palpation of the bursa, and usually have purulent drainage from a previous penetrating injury (Figure 10.2).

### *Diagnosis*

- The diagnosis of a capped hock is usually based on clinical findings alone but ultrasound is useful to confirm the diagnosis.
- A definitive diagnosis of the cause of nonseptic calcaneal bursitis usually requires a combination of radiography and ultrasonography. However, endoscopy of the bursa is recommended as a diagnostic tool if other imaging results are negative.
- The diagnosis of septic bursitis is determined similarly to other sites of synovial infection and includes a combination of plain and contrast radiography, ultrasonography, aspiration of synovial fluid, and culture.

### *Treatment*

#### Capped Hock

- Small swellings over the tuber calcanei may merely be a cosmetic concern and not require treatment.
- Methods to prevent further trauma to the tuber calcanei are suggested to prevent the capped hock from worsening.
- Larger swellings are treated with topical anti-inflammatories such as ice, dimethyl sulfoxide (DMSO), or topical diclofenac liposomal cream (Surpass®) combined with bandaging.
- Other options include aseptic drainage and injection of corticosteroids or iodinated contrast agents.
- Counterpressure with bandaging is recommended for at least two weeks but can be difficult in this high-motion area.
- Surgical drainage using Penrose drains or complete removal of the bursa are rarely recommended due to the problems with wound healing in this location.

#### Nonseptic Calcaneal Bursitis

- The treatment often depends on the initiating cause but is similar to other types of synovial inflammation.
- Acute bursitis without a defined cause is often treated with intrasynovial triamcinolone and hyaluronan (HA) combined with a short period of rest.
- Known causes of calcaneal bursitis such as osteolytic lesions within the calcaneus and abnormalities within the gastrocnemius or SDFT should be debrided endoscopically.

#### Septic Calcaneal Bursitis

- Superficial wounds to the subcutaneous bursa usually resolve with routine wound care and have an excellent prognosis.
- Wounds that involve the intertendinous calcaneal bursa should be treated aggressively in the acute stage to prevent synovial infection (Figure 7.11).
- Horses with septic calcaneal bursitis should be treated with a combination of synovial lavage (endoscopy), local and parenteral antibiotics including IV regional perfusion, and NSAIDs.

### *Prognosis*

- The prognosis for horses with a capped hock is usually very good unless it becomes chronic and fibrotic.

- Based on a very limited number of cases, horses with nonseptic calcaneal bursitis, including those with osteolytic lesions of the calcaneus, have a guarded prognosis for athletic activity.
- Horses with septic calcaneal bursitis tend to have a fair to guarded prognosis for athletic use because of the high motion required in this area during limb flexion.

## TARSAL SHEATH TENOSYNOVITIS (THOROUGHPIN)

### *Introduction*

- The tarsal sheath is located on the medial aspect of the tarsus and begins approximately 5 to 8 cm proximal to the medial malleolus and extends distally to the proximal one-third of the metatarsus (Figure 7.12).
- It encloses the DDFT of the hindlimb as it courses over the sustentaculum tali on the medial aspect of the tarsus.
- Thoroughpin is a morphological description of the swelling, and while some cases may have idiopathic synovitis, potential causes for tarsal sheath tenosynovitis include damage to the DDFT, sustentaculum tali, or calcaneus and infection from penetrating injuries or hematogenous spread.

### *Etiology*

- Some horses have effusion within the tarsal sheath for what is thought to be no apparent reason (Figure 7.13).
- The most likely cause for effusion and lameness is previous trauma to the sustentaculum tali, such as kick injuries to the medial aspect of the tarsus (Figure 3.36).
- Damage to the DDFT within the tarsal sheath is also most likely trauma related and often occurs secondary to an exostosis of the sustentaculum tali.
- Penetrating injuries to the tarsal sheath may lead to infectious tenosynovitis similar to any synovial structure (Figure 10.9). Osteomyelitis of the sustentaculum tali is not an uncommon finding in many of these horses.

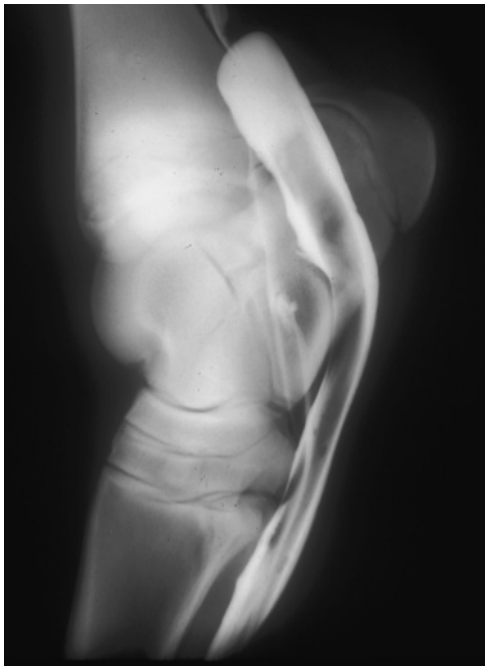

**Figure 7.12.** Iodinated contrast material has been injected into the tarsal sheath, showing the proximal and distal limits of this synovial cavity on the medial aspect of the tarsus.

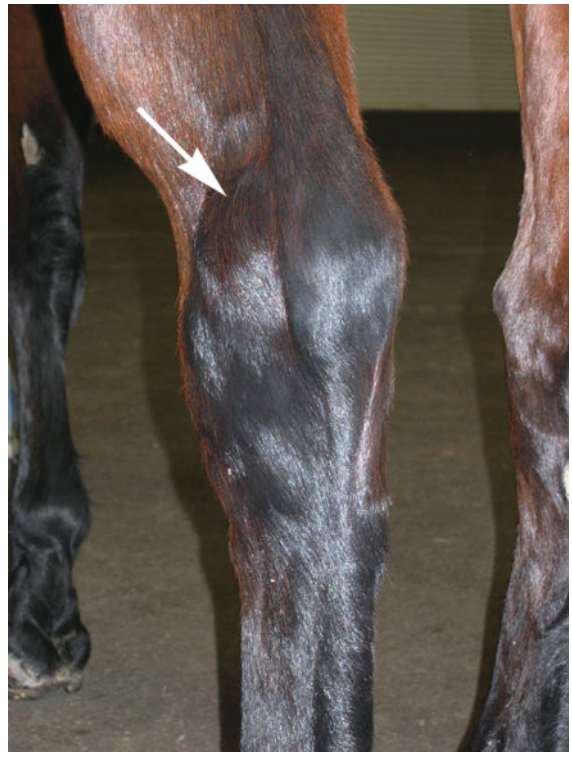

**Figure 7.13.** Thoroughpin can involve only the medial aspect of the tarsus but may also project to the lateral side, as seen in this horse (arrow).

### *Clinical Signs*

- Horses with idiopathic tenosynovitis have effusion within the tarsal sheath but no apparent lameness or performance limitations. The effusion is typically located on the medial aspect of the tarsus and courses up and down the leg in the direction of the tarsal sheath, but may also be visible and palpable from the lateral aspect of the tarsus (Figure 7.13).
- Horses with previous trauma to the sustentaculum tali often present for lameness and swelling of the medial aspect of the tarsus. Most horses are grade 2 to 3/5 lame and are positive to a tarsal flexion test. Intrasynovial anesthesia of the tarsal sheath usually improves the lameness.
- Horses with infection of the tarsal sheath are usually non-weight-bearing lame, and are painful to digital pressure applied to the sheath. Effusion within the sheath may be difficult to determine in some horses because of diffuse swelling of the entire tarsus.

### *Diagnosis*

- A presumptive diagnosis of tarsal sheath tenosynovitis is made based on the clinical finding of effusion within the sheath.
- Clinically significant abnormalities within the sheath should be suspected in horses that are lame, positive to tarsal flexion, or improve with intrasynovial anesthesia of the sheath.
- Radiographs of the tarsus including a skyline view of the sustentaculum tali are recommended. The 45° dorsomedial-plantarolateral oblique projection is also helpful.
- Contrast radiography or a fistulogram may be helpful to document synovial involvement of the tarsal sheath in horses with penetrating wounds
- Fractures, osteomyelitis, and proliferative exostosis of the sustentaculum tali are the most common radiographic abnormalities seen (Figure 7.14).
- Ultrasound should be performed to document abnormalities within the DDFT.
- Aspiration of synovial fluid from the sheath should be performed in horses with suspected sepsis or osteomyelitis of the sustentaculum tali to help document the presence of infection.

### *Treatment*

- True idiopathic tenosynovitis without lameness can be treated with benign neglect or intrasynovial injections of anti-inflammatory medication such as corticosteroids and/or HA. Bandaging the tarsus following intrasynovial treatment may help prevent recurrence.
- Persistent idiopathic effusion also may be treated with intrasynovial atropine, but a

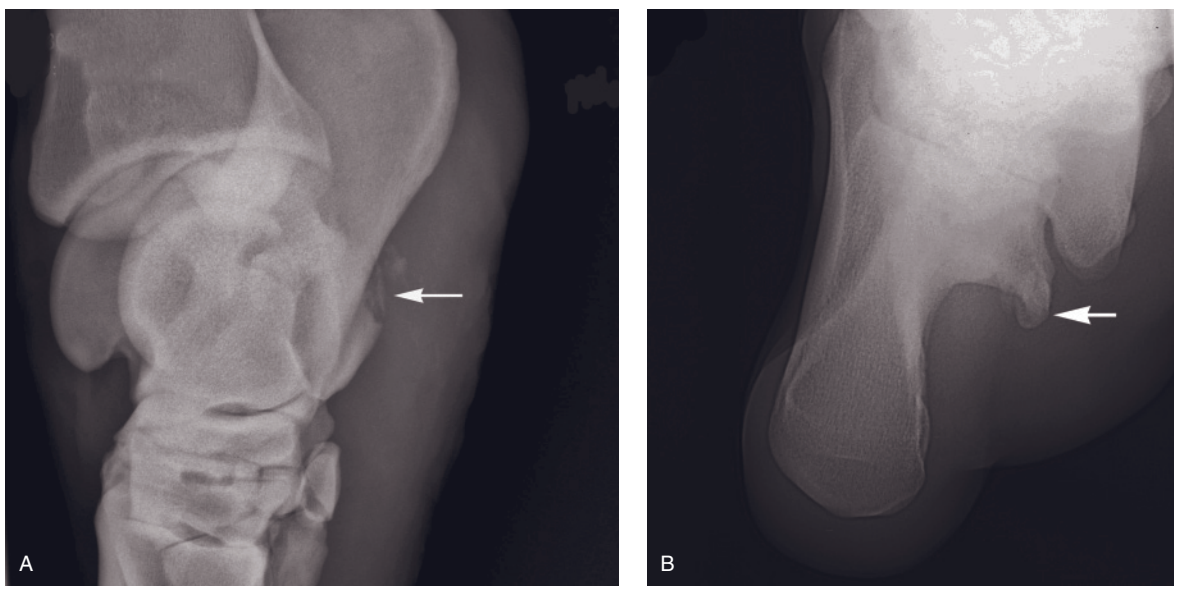


**Figure 7.14.** Oblique (A) and skyline (B) radiographs of two different horses with open (A) and closed (B) fractures of the sustentaculum tali (arrows).

predisposing cause should be documented before resorting to this treatment.
- Horses with fractures, exostosis, or osteomyelitis of the sustentaculum tali are usually best treated surgically. This is especially true for open fractures and osteomyelitis with secondary infection of the sheath.
- There is some debate whether horses with exostosis of the sustentaculum tali benefit from removal of the exostosis, but these horses often respond poorly to other treatments.
- Endoscopy of the sheath is the preferred technique, although a small incision directly over the medial sustentaculum tali also may be used.
- Horses with penetrating injuries or infection within the tarsal sheath are best treated with a combination of endoscopic lavage and debridement, parenteral and intrasynovial antimicrobials, and IV regional limb perfusion.

### *Prognosis*

- Horses with idiopathic synovitis have a very good prognosis except for the cosmetic blemish.
- Horses with small fractures of the sustentaculum tali can do very well, provided damage to the DDFT is minimal (Figure 7.14A).
- The overall prognosis for recovery from a septic tendon sheath is only fair but is improved if no radiographic changes are present.
- The majority of horses with fractures or infection of the sustentaculum tali that are treated surgically should be able to return to performance, but the prognosis is variable based on many other factors.

## RUPTURE OF THE PERONEUS TERTIUS

### *Introduction*

- The peroneus tertius is a strong muscular band of tissue that lies between the long digital extensor and the tibialis cranialis muscle on the cranial aspect of the hindlimb.
- It originates from the extensor fossa of the distal lateral femur and inserts distally as a tendinous band to MTIII and laterally on the fourth splint bone.
- It is an important part of the reciprocal apparatus, mechanically flexing the hock when the stifle joint is flexed.
- The muscle or tendon can rupture anywhere along its course and an avulsion fracture at its origin in the extensor fossa of the femur can occur in young horses. When this muscle is ruptured, the stifle flexes but the hock does not (Figure 7.15).

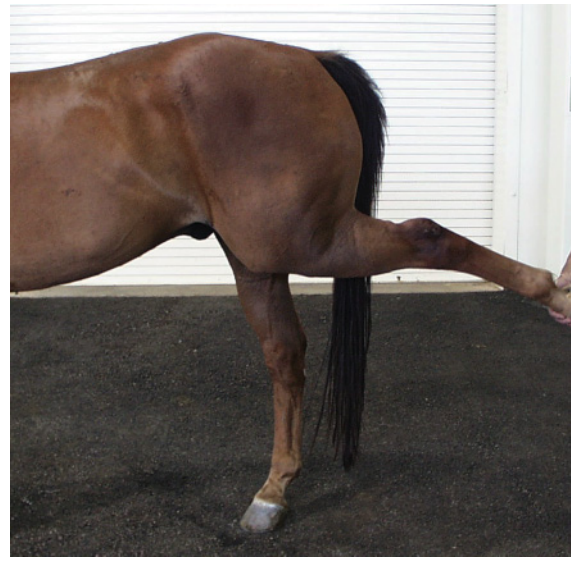

**Figure 7.15.** Rupture of the peroneus tertius disrupts the reciprocal apparatus, allowing extension of the tarsus and fetlock with the stifle flexed.

### *Etiology*

- Rupture of the peroneus tertius is usually due to overextension of the hock joint. This may occur if the limb is entrapped and the horse struggles violently to free it.
- Rupture also may occur during the exertion of a fast start, when tremendous power is transferred to the limb. Examples include jumping and barrel racing.
- It also is a complication of full-limb cast application to the hindlimb.

#### Clinical Signs

- With complete rupture, the stifle joint flexes as the limb advances and the hock joint is carried forward with very little flexion.
- The portion of the limb below the hock tends to hang limp, giving the appearance that it is disconnected from the upper limb when carried forward.
- As the horse walks, dimpling in the tendon of Achilles can be seen and there is usually minimal lameness present.
- If the limb is lifted from the ground, a dimpling in the tendon of Achilles can be pro-

duced by extending the hock; the hock can be extended without extending the stifle (Figures 3.41, 7.15).
- If the origin of the peroneus tertius fractures from the femur, femoropatellar effusion is usually present.

### *Diagnosis*

- Often, the diagnosis can be made based on the clinical findings.
- Radiographs of the stifle should be performed to document an avulsion fracture if femorpatellar effusion is present.
- Ultrasound can be used to better document the location and severity of the rupture.

### *Treatment*

- Complete rest is the best treatment currently available. The horse should be placed in a box stall and kept quiet for at least four to six weeks.
- Most cases heal and eventually have normal limb action. Some horses can return to normal work.
- Some horses will not regain a normal gait and the limb will appear "loose" or "sloppy" compared to the normal limb.

### *Prognosis*

- The prognosis is guarded to favorable for athletic performance and can be difficult to predict.
- If healing is not evident by four to six weeks, the prognosis is usually unfavorable, but final appraisal should not be made for at least three months following the injury.
- As with most soft tissue injuries, re-injury can occur.

## STRINGHALT

### *Introduction*

- Stringhalt is an involuntary hyperflexion of the hock when the horse moves that can affect one or both hindlimbs.
- The severity of the hyperflexion may vary from barely perceptible to the fetlock contacting the ventral abdomen.
- Two forms of stringhalt occur: a unilateral spontaneous form (North American stringhalt) of unknown cause (may be associated with trauma) and a bilateral form (Australian stringhalt) presumably caused by toxic plants or mycotoxins that reside on the plants.

### *Etiology*

- One form of stringhalt affects isolated horses and is usually unilateral. It may follow an injury to the hindlimbs and is thought to involve the lateral digital extensor muscle tendon unit.
- A small percentage of horses with stringhalt may have a history of previous injury to the dorsoproximal metatarsal extensor structures that healed by second intention.
- Australian stringhalt has been restricted to Australia and New Zealand and commonly occurs in outbreak proportions. It is usually bilateral and is thought to be due to certain toxic weeds including *Taraxacum officinael*, *Malva parviflora,* or *Hypochaeris radicata* (a dandelion), or mycotoxins that reside on the plants.
- A condition similar to Australian stringhalt has been reported in northern California, Washington, and southern Chile under similar conditions.
- The pathologic effect is thought to be a peripheral neuropathy (axonopathy) of the long peripheral nerves such as the recurrent laryngeal, peroneal, and tibial nerves. Neurogenic atrophy primarily affects the cricoarytenoideus dorsalis, the long and lateral digital extensors, and the gastrocnemius muscles.
- The pathophysiology of the hyperflexion remains unknown. A plausible theory is that the action-debilitated extensors are overridden by the comparatively minimally affected flexors, the biceps femoris, and semitendinosus.

### *Clinical Signs*

- Signs of the disease are quite variable; some horses show a very mild flexion of the hock during walking, whereas others show a marked jerking of the foot toward the abdomen.
- The hyperflexion also may not occur with every step. See the DVD for an illustration of stringhalt.
- The signs are usually exaggerated when the horse is backed and may occur intermittently for unknown reasons.
- Most affected horses have a nervous disposition, which may play a part in the etiology.
- Other peripheral neuropathies such as laryngeal hemiparesis may occur concurrently.

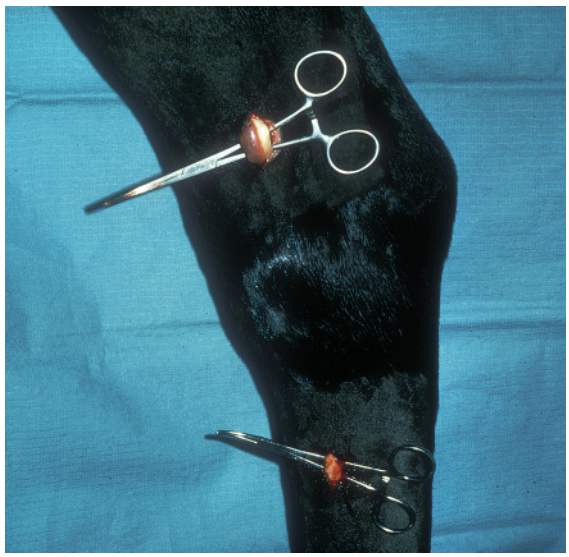

**Figure 7.16.** Lateral digital extensor myotenectomy illustrating the proximal and distal incision sites to perform this procedure.

### *Diagnosis*

- For either form, the characteristic gait is usually enough to make the diagnosis.
- The condition must be differentiated from fibrotic myopathy and intermittent upward fixation of the patella See the DVD for an illustration of these conditions.

### *Treatment*

- The classic treatment for North American unilateral stringhalt is lateral digital extensor myotendonectomy (Figure 7.16). However, the success of this surgery is unpredictable.
- Spontaneous recovery of affected horses is uncommon but has been reported.
- For horses with Australian stringhalt, the majority of horses recover spontaneously without treatment once they are removed from pasture. Often recovery can be protracted, from several weeks to one year.
- Pharmacological therapies that have been used include mephenesin, phenytoin, and baclofen, but more experience is required with these agents before their efficacy is known.

### *Prognosis*

- For North American stringhalt, the prognosis is guarded to favorable. Most horses show some improvement after surgery but the degree of improvement is not predictable.
- For Australian stringhalt, the prognosis is similar. Many horses recover after removal from the pastures, whereas others do not.

## TIBIAL STRESS FRACTURES

### *Introduction*

- Tibial stress fractures occur predominantly in young racehorses (2 and 3 year old).
- Many tibial fractures occur during training, before the horses have actually raced.
- These fractures tend to occur in the proximal caudal to lateral aspect of the tibia or the mid-diaphyseal region of the tibia.

### *Etiology*

- Remodeling due to cyclic fatigue of bone in horses that work at speed is thought to result in stress fractures.
- Continued work at speed can cause catastrophic separation (complete tibia fracture) of incompletely healed stress fractures.

### *Clinical Signs*

- An acute hindlimb lameness that gradually improves with inactivity is often observed.
- The lameness can be variable but is often moderate in severity without any palpable abnormalities.
- Deep palpation of the tibia at the typical locations for stress fractures may be painful.
- Local anesthesia is usually not helpful and is contraindicated to avoid repeated jogging of the horse.

### *Diagnosis*

- With acute fractures, radiographs may be normal and scintigraphy is often required to document the lesion (Figure 7.17).
- If radiographically apparent, a small linear crack in the tibia may be all that is visible (Figure 7.17B) unless a healing callus is present (Figure 4.11B).
- Repeat radiographs are usually used to document healing of the fracture but repeat scintigraphy can also be used.

### *Treatment*

- An initial period of stall rest (four to 12 weeks) followed by a controlled exercise program is the typical treatment.

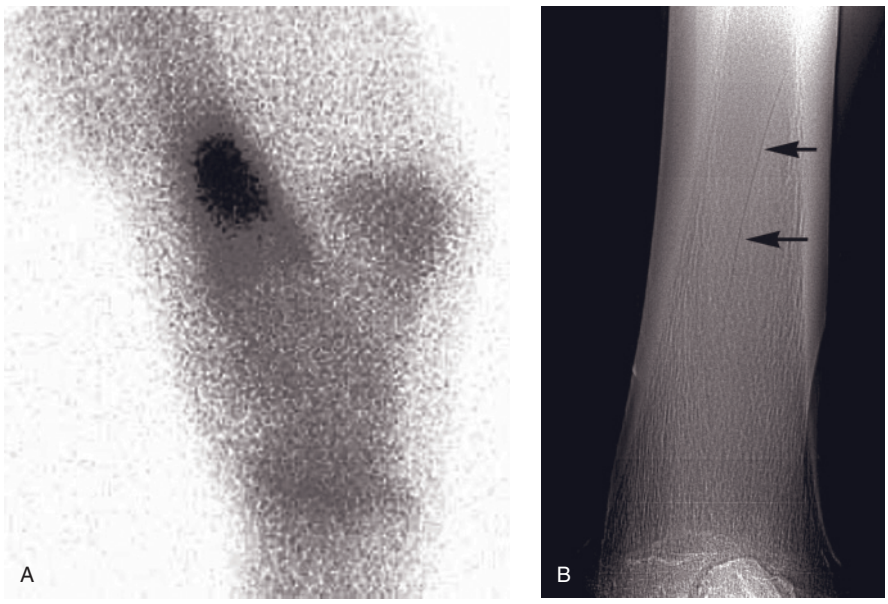


**Figure 7.17.** Lateral scintigram (A) and tibial radiograph (B) demonstrating an incomplete tibial fracture (arrows).

- Affected horses usually return to racing in approximately nine to 11 months, but some can return sooner.
- Extracorporeal shock wave therapy (ESWT) has been used to treat tibial stress fractures with variable results.
- Complete healing should be documented before allowing the horse to return to work.

### *Prognosis*

- The prognosis for return to racing is usually very good following a controlled exercise program.
- Returning horses to training too soon may predispose to complete fracture of the tibia.

## DIAPHYSEAL AND METAPHYSEAL TIBIAL FRACTURES

### *Introduction*

- Most complete fractures of the tibial shaft have a spiral configuration and/or are comminuted.
- Most fractures in older horses are highly comminuted due to the highly brittle nature of the bone.
- In general, the smaller the patient, the better the prognosis for successful treatment.
- Most adult horses with tibial fractures are euthanized because of the poor prognosis with any form of treatment.
- Open fractures are not uncommon because of the sparse soft tissue coverage on the medial aspect of the tibia.

### *Etiology*

- The cause of many tibial fractures is external trauma (e.g., kick, fall, pivot, or bad step).
- Complete fractures may occur subsequent to stress or incomplete fractures at any location in the tibia.
- Fractures due to torsion combined with bending and axial compressions have been described.
- Midshaft tibial fractures can occur from falls during a race or spontaneously for no apparent reason.
- An *in vitro* study demonstrated that direct high velocity midshaft tibial trauma produced an oblique fracture or a fracture with butterfly fragment comminution opposite the point of cortical impact. Small fragments occurred at the point of impact, and 98% had additional fissures.

### *Clinical Signs*

- Complete fracture of the tibia is characterized by a non-weight-bearing lameness, marked soft tissue swelling, angular deformity of the limb, and palpable crepitus.

- Craniomedial overriding of the proximal fragment coupled with valgus angulation frequently results in an open fracture due to the lack of soft tissue covering the medial aspect of the tibia.

### *Diagnosis*

- The obvious instability, swelling, and pain usually make a tentative diagnosis obvious.
- Radiographs are required to define the fracture configuration and to formulate a treatment plan or recommend euthanasia.
- Some incomplete or non-displaced fractures of the tibia may be difficult to diagnose and several oblique projections may be needed to demonstrate the fracture (Figure 7.17).
- If no fracture line can be identified, scintigraphy is recommended or the horse should be confined and the radiographs repeated in five to seven days.

### *Treatment*

- Euthanasia is advised for adult horses with severely comminuted fractures that cannot be stabilized.
- Conservative management can be used successfully to treat non- or minimally displaced tibial fractures, provided catastrophic separation doesn't occur during convalescence. Horses with shorter (3 to 7 cm) visible fissure lines are more likely to survive (fractures do not displace) than those with longer (12 to 15 cm) spiral fissure lines (Figure 7.18)
- The decision to use internal fixation on non-displaced tibial fractures can be difficult. Fractures traversing much of the length of the tibia probably should be repaired with internal fixation. This is also the case if the lameness remains severe.
- Any form of external coaptation to treat tibial fractures can be difficult, regardless of the size of the horse, and is generally not recommended.
- Internal fixation is usually the best treatment option in young horses with displaced fractures. Double plating is usually required and locking compression plates should provide more secure fixation than regular dynamic compression plates.

### *Prognosis*

- The prognosis for a fractured tibia in an adult horse is extremely poor.

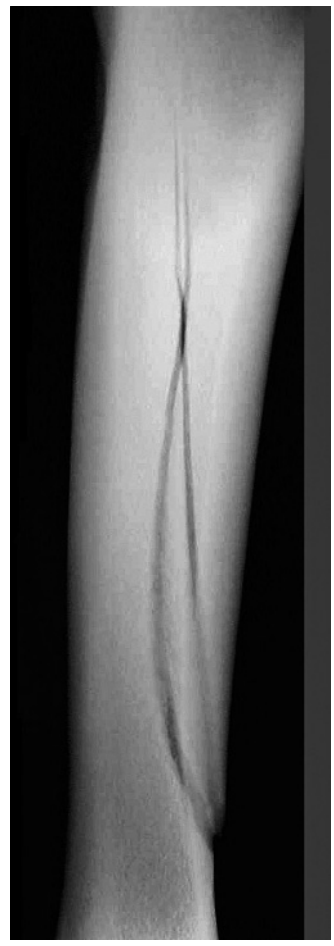

**Figure 7.18.** Lateral radiograph of a horse with a long spiral oblique tibial fracture that would most likely displace further if left untreated.

- Non-displaced fractures may heal with stall rest, but complete separation may still occur after several weeks.
- The prognosis for successful repair of tibial fractures in young horses with internal fixation is reported to be about 60% to 70% but depends on the fracture type, its duration, and the treatment selected.

## TIBIAL TUBEROSITY/CREST FRACTURES

### *Introduction*

- The physis of the tibial tuberosity is partially ossified at birth and forms a fibrocartilage union with the epiphysis during the second year of life.

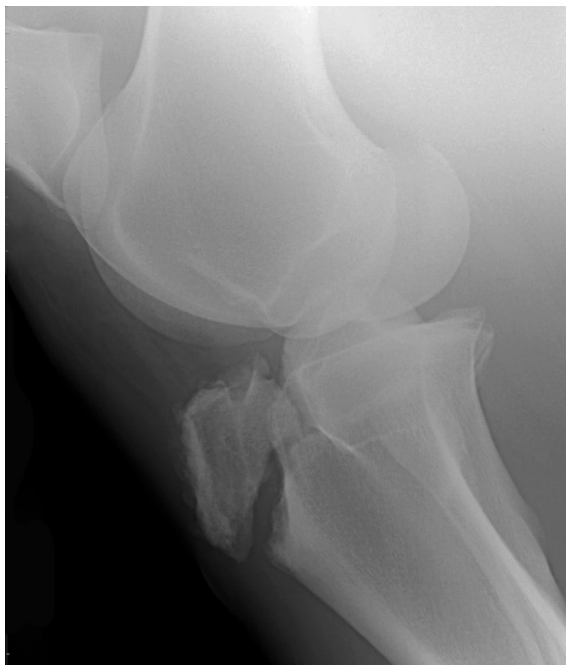

**Figure 7.19.** Lateromedial radiograph of a horse with a chronic, displaced, complete fracture of the tibial tuberosity that was repaired with a single locking plate. Courtesy of Laurie Goodrich.

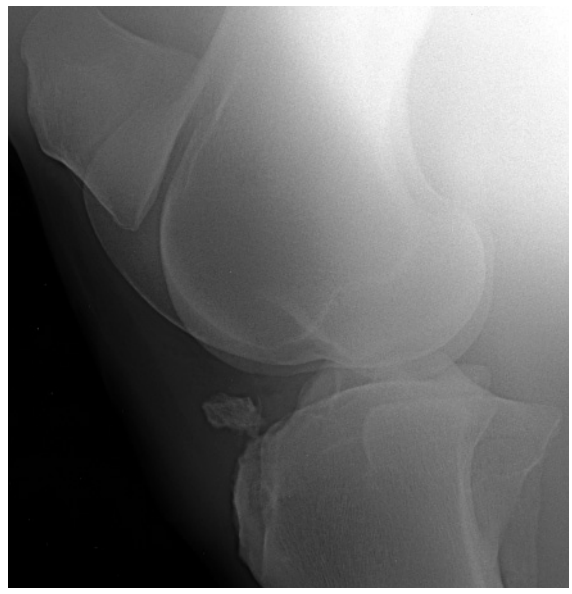

**Figure 7.20.** This small, chronic fracture of the lateral tibial tuberosity was removed because it was contributing to chronic drainage.

- The irregular physis remains radiographically visible until 36 to 42 months of age and may be mistaken for a fracture.
- The tibial tuberosity is relatively broad with regional insertions for the three patellar ligaments. The area of the insertion of lateral patellar ligament protrudes prominently and is usually involved with the fracture.
- Fractures are usually in the frontal plane and the size and configuration can vary considerably (Figures 7.19 and 7.20). Some can be open.

### *Etiology*

- Most tibial crest fractures occur from direct trauma, such as a kick, or from hitting a jump. It is a common injury in event horses.
- Horses may occasionally avulse the fragment due to sudden quadriceps tension. Displacement is in the proximal and cranial direction and quadriceps integrity usually remains intact.

### *Clinical Signs*

- The severity of the clinical signs depends on the size of the fracture.
- Most horses are acutely lame with the typical signs of inflammation localized to the proximal tibial region.
- Horses with small nonarticular fractures improve quickly but focal swelling of the tibial crest usually remains.
- Large fractures cause considerable lameness and if the middle patellar ligament is compromised, the stifle may be "dropped" and the horse may not be willing or able to fix the limb in extension.
- An open wound may be present, especially with kick injuries, which must be taken into account when planning treatment.

### *Diagnosis*

- The lateromedial radiograph usually demonstrates the fracture but the caudolateral-craniomedial oblique projection may provide additional information (Figure 7.19).
- Most tibial tuberosity fractures are nonarticular; those with fracture lines at or caudal to the intercondylar eminence of the proximal tibia may be articular.
- Secondary osteomyelitis of the fracture may be present with chronic open fractures.
- Ultrasound is useful to detect concurrent soft tissue injury and identify which patellar

ligament insertions are involved with the fracture.

### *Treatment*

- Traction by the patellar ligaments tends to distract the fragment, which impairs healing (Figure 7.19). However, non- or minimally displaced fractures can heal following stall rest for several weeks.
- These fractures take much longer to heal than expected, and complete radiographic union is not always required for soundness.
- Small fractures not involving the insertion of the middle patellar ligament can be removed, especially if they are open or infected (Figure 7.20). If left, these fractures tend to take a very long time to heal and may cause lameness.
- Unstable, displaced, or articular fractures should be repaired with internal fixation using the tension-band principle. Tension-band wire, headless screw fixation with cable tension band, or lag screws alone have been used successfully. Plate fixation improves the repair when the fragment is large and/or unstable.

### *Prognosis*

- In a series of 17 horses with non-articular fractures treated conservatively, 14 became sound and 12 returned to work in an average time of 6.3 months.
- The recovery rate for surgically repaired tibial tuberosity fractures also is generally good, provided stable fixation can be accomplished.
- Horses tend to have a very good prognosis following removal of small tuberosity fractures.

## FRACTURES OF THE PROXIMAL TIBIAL PHYSIS

### *Introduction*

- Fractures of the proximal tibial physis have been observed in foals up to 8 months of age.
- The fracture is nearly always a Salter Harris Type II fracture with the metaphyseal fragment located laterally (Figure 7.21).
- It is rarely open or articular and concurrent soft tissue injuries are uncommon.

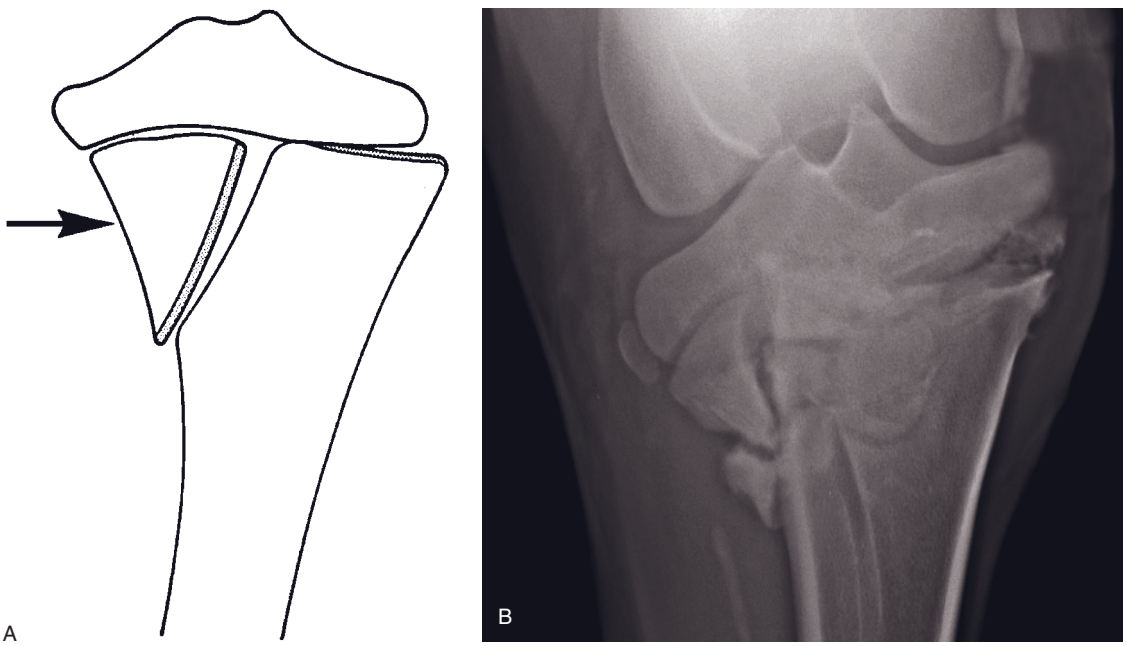


**Figure 7.21.** Line illustration (A) and caudocranial radiograph (B) of the stifle demonstrating a Salter-Harris Type II fracture of the proximal tibial physis. The metaphyseal component is always lateral (arrow) and usually involves approximately one-third of the distance across the physis.

### *Etiology*

- The injury usually occurs from direct trauma (e.g., kick) while the limb is bearing weight, or from bending while having the limb somehow entrapped or stepped upon by the dam.
- The forces apply pressure in a valgus direction, causing medial tension to separate the physeal cartilage. The epiphysis and bone fragment displace laterally due to the "ramp" defect left in the proximal lateral metaphysis (Figure 7.21).

### *Clinical Signs*

- The affected limb usually assumes a "stifle" valgus position (the distal limb is deviated outward).
- Lameness is usually severe initially and improves with time. Swelling and pain typical of a fracture are often palpable. Crepitus is usually difficult to detect.

### *Diagnosis*

- Radiographs are required to confirm the diagnosis. The caudal-cranial view is the most informative, revealing the displacement of the epiphysis and metaphyseal component.
- Most fractures are considerably displaced.

### *Treatment*

- Surgical repair with a bone plate placed on the medial aspect of the tibia is the treatment of choice (Figure 7.22). Other less desirable methods of internal fixation include lag-screw fixation alone or cross pinning using intramedullary pins.
- No external coaptation is used after surgery and the implants should be removed in eight to 10 weeks to help prevent the development of a permanent angular limb deformity.
- Conservative management has been used successfully to treat non-displaced tibial physeal fractures in a small number of horses but is usually not recommended.

### *Prognosis*

- The prognosis is generally favorable for fracture repair, barring complications such as failure of the fixation, angular limb deformity, infection, or wound dehiscence.
- The smaller the foal, the better the prognosis.

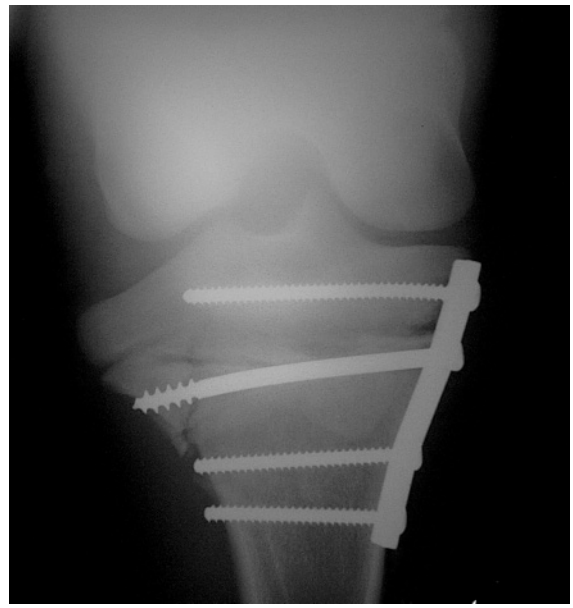

**Figure 7.22.** Repair of a proximal tibial physeal fracture in a foal using a medial plate. The first screw was placed as far as possible across the epiphysis and the second screw engaged the metaphyseal component of the proximal fragment. Courtesy of Ken Sullins.

- The prognosis for athletic activity following successful fixation is approximately 50%.

## FEMOROPATELLAR OCD

### *Introduction*

- Femoropatellar OCD is a common cause of stifle effusion and lameness in horses (Figure 3.42).
- Thoroughbreds were the most commonly affected breed of a series of 161 horses having surgery, and 78% were 2 years old or younger (mostly yearlings).
- The most common site for femoropatellar OCD to occur is the lateral trochlear ridge of the femur.
- Half or more of affected horses have bilateral lesions.

### *Clinical Signs*

- Femoropatellar OCD usually causes visible joint effusion (Figure 3.42) and variable hindlimb lameness. The lameness is usually so

mild that joint effusion is the primary complaint.
- Infrequently, bilaterally affected horses may be extremely lame and may have difficulty rising from recumbency.
- Horses presenting after training has begun generally have less severe lesions than horses presenting at an earlier age.
- Some weanlings may present with joint effusion, lameness, and no radiographic abnormalities.

### *Diagnosis*

- Lateromedial radiographs of the stifle usually demonstrate a flattened defect in the proximal portion of the lateral trochlear ridge of the femur. Ossification within the defect is variable and loose bodies may be present (Figure 7.23).
- Caudolateral to craniomedial oblique films may provide more information about the severity of lateral trochlear ridge defects.
- Other locations for OCD lesions in the stifle include the medial trochlear ridge and the patella.
- Secondary abnormalities of the patella usually can be observed in the lateromedial view.
- Ultrasound can be used as an adjunct to radiographs because the trochlear surfaces of the distal femur are readily imaged using ultrasound.

### *Treatment*

- When clinical signs are present in adults, surgical debridement produces better results than conservative therapy.
- Some OCD lesions may resolve over time in foals less than 1 year of age. Stall confinement is recommended to protect the articular surface, and both systemic (PSGAG) and IA joint medications (HA or PSGAG) may be beneficial. IA corticosteroids are contraindicated.
- Arthroscopic debridement is recommended in foals less than 1 year of age if the clinical signs persist.
- Arthroscopic debridement is indicated in nearly all horses beyond 1 year of age with femoropatellar OCD.

### *Prognosis*

- The prognosis for athletic activity following arthroscopic surgery for femoropatellar OCD is generally very good.
- Increasing OCD lesion size has a negative effect on outcome (defects less than 2 cm, greater than 2 cm but less than 4 cm, and greater than 4 cm had decreasing success rates of 78%, 63%, and 54%, respectively).
- Horses with lateral trochlear ridge lesions and patellar lesions usually have a reduced prognosis.

## FRACTURES OF THE PATELLA

### *Introduction*

- Several configurations of patellar fractures including sagittal, transverse, comminuted, basilar (proximal), and distal fragmentation of the patella occur in the horse (Figures 7.24, 7.25).
- Most patellar fractures are articular and involve the medial aspect of the bone (Figure 7.25A).
- Most fractures do not disrupt the function of the middle patellar ligament, but this should be determined before treatment is initiated.
- Open fractures associated with kick injuries occur.

### *Etiology*

- Direct trauma to the patella while the stifle joint is in a semi-flexed position is the usual cause. Horses that jump can strike jumps or the fracture can occur from a kick to the cranial aspect of the stifle.
- The prominence of the medial trochlear ridge may be a point of contact causing a relative higher incidence of fractures toward the medial side of the patella.
- Fragmentation of the distal patella may occur following a medial patellar ligament desmotomy if horses are returned to work too soon.

### *Clinical Signs*

- Most horses present with an acute onset of moderate to severe lameness and a painful swelling associated with the cranial aspect of the stifle (Figure 7.26).
- Femoropatellar effusion is usually present but significant soft tissue swelling may obscure its detection.
- Flexion of the stifle joint exacerbates the lameness and elicits a painful response.
- Weight-bearing may be difficult and the horse may stand with the limb partially flexed without locking the stifle.

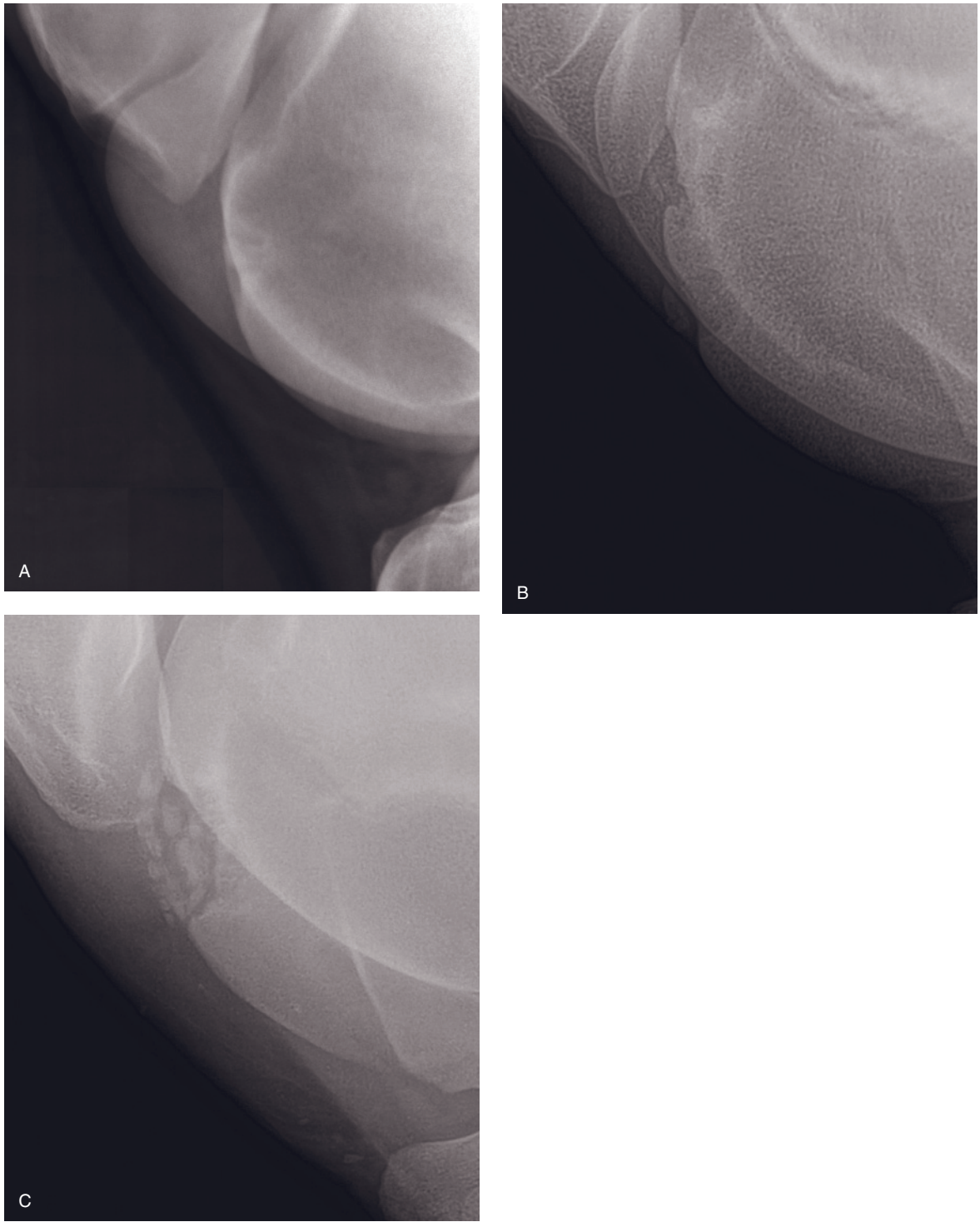


**Figure 7.23.** Radiographs of three cases of OCD of the lateral trochlear ridge of the femur in the femoropatellar joint. (A) A small defect in the lateral trochlear ridge without obvious fragmentation within the defect (treated conservatively). (B) A fragment within a defect. (C) multiple fragments in a more severe lesion. Courtesy of C.W. McIlwraith.

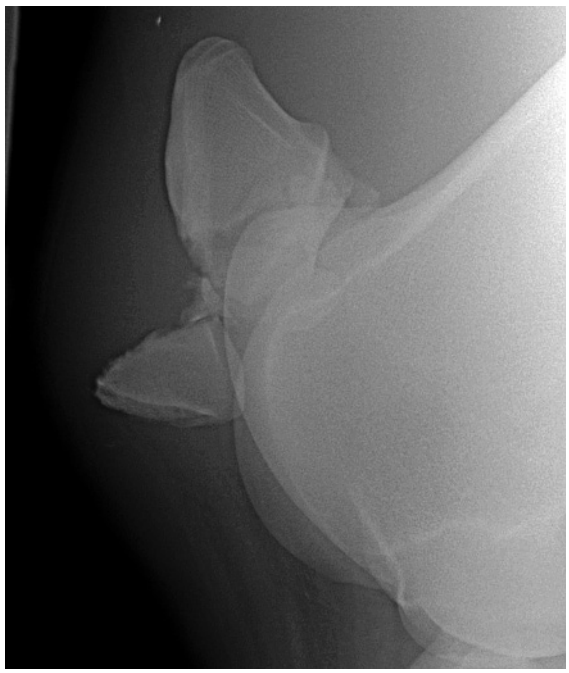

**Figure 7.24.** Lateromedial radiograph demonstrating a transverse fracture of the patella.

- Horses with smaller fractures may present for routine lameness evaluation but typically have effusion of the femoropatellar joint and are positive to stifle flexion.
- Horses with open fractures can present with severe lameness due to secondary infection of the femoropatellar joint.
- Horses with compromise to the middle patellar ligament are unable to lock the stifle and support any weight on the limb. This is usually associated with highly comminuted and displaced fractures.

### *Diagnosis*

- Radiographs are required to document the type and extent of the fracture. Routine lateromedial and caudocranial projections usually demonstrate transverse or comminuted fractures (Figure 7.24); the caudolateral-to-craniomedial oblique projection accentuates the apex (distal border) of the patella.
- Smaller medial fragments may not be visible on routine radiographs and may require a cranioproximal to craniodistal (skyline) projection (Figure 7.25).

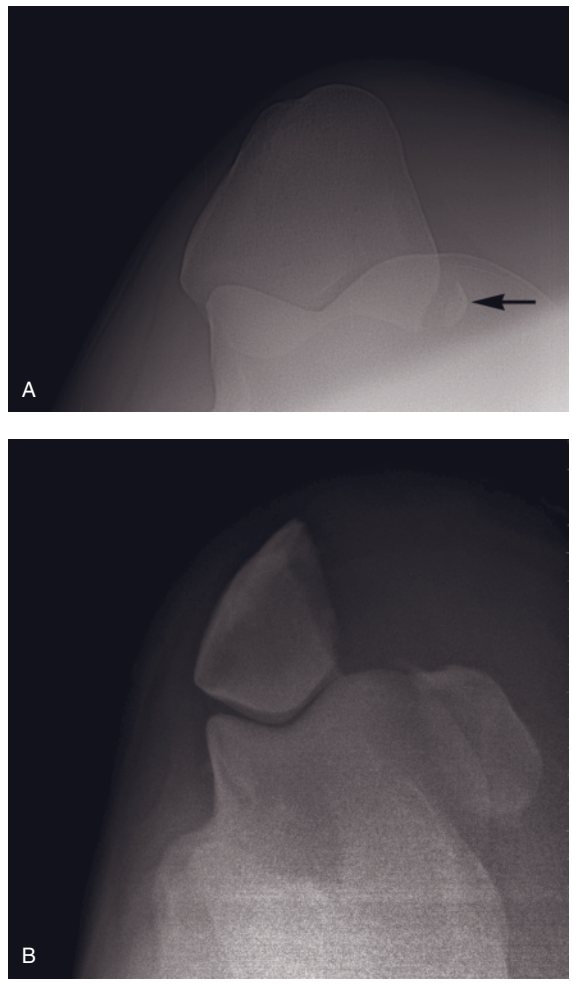


**Figure 7.25.** Cranioproximal to craniodistal (skyline) projections showing a typical medial patellar fracture fragment (A) (arrow) and a large displaced sagittal fracture (B). Arthroscopic removal is indicated in (A).

- Ultrasound is useful to identify small fracture fragments, patellar ligament disruption, or other lesions that may not be radiographically visible.

### *Treatment*

- Nonarticular or small medial or basilar fragments may heal with rest and anti-inflammatory therapy.
- Horses with intra-articular fractures seldom remain sound when returned to work. Fragments that are less than one-third of the patellar substance should be removed

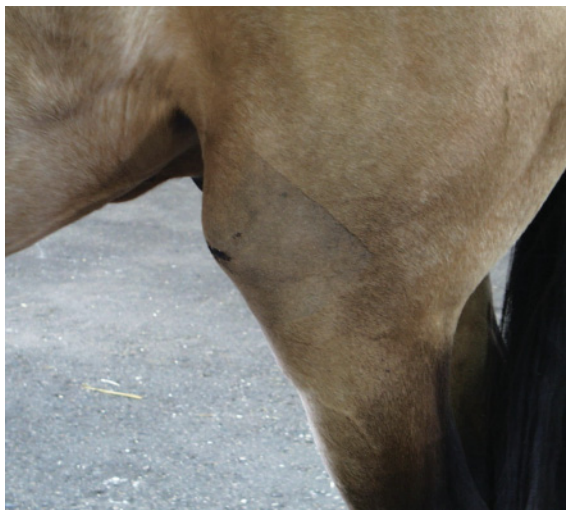

**Figure 7.26.** Physical appearance of the stifle of the horse with the patellar fracture illustrated in Figure 7.24 with associated swelling and skin abrasion.

arthroscopically for the best success (Figure 7.25A).
- Fragmentation of the distal patella secondary to medial patellar ligament desmotomy is best treated with arthroscopic debridement.
- Internal fixation should be considered for displaced fractures with sizeable fragments and transverse fractures. Successful internal fixation of transverse distracted and longitudinally displaced fractures of the patella have been reported.
- Horses with highly comminuted fractures and those with disruption of the middle patellar ligament are candidates for euthanasia.

### *Prognosis*

- The prognosis following removal of articular medial fragments is considered very good (16 of 19 horses returned to work in one study).
- The prognosis for debridement of distal patellar fragmentation is also very good if secondary articular cartilage damage is minimal.
- Internal fixation provides the best circumstances for a favorable outcome for displaced and transverse fractures, but the prognosis remains fair to guarded.
- Horses with severely comminuted fractures are likely to remain lame but some may attain breeding soundness with prolonged stall rest.

**Figure 7.27.** A horse with upward fixation of the patella. The limb is locked in extension and extended caudally and a bit laterally. The digit is also fixed in the flexed position. Courtesy of Ken Sullins.

## UPWARD FIXATION OF THE PATELLA (UFP)

### *Introduction*

- Upward fixation of the patella occurs when the medial patellar ligament (MPL) becomes caught over the medial trochlear ridge.
- When the MPL is fixed in that position, the hindlimb cannot be flexed, and the horse assumes a posture with the affected limb extended in a caudally abducted position with the fetlock flexed due to the reciprocal apparatus (Figure 7.27).
- Intermittent "catching" of the MPL also occurs and is considered a less severe stage of UFP.
- UFP appears to be common in ponies and miniature horses but can occur in all breeds of horses.

### *Etiology*

- Horses that have exceptionally straight hindlimbs are considered to be prone to UFP. To support this, hyperextension of the limb by walking a horse downhill usually exacerbates UFP.
- Loss of quadriceps muscle tone due to reduced work or debilitation functionally "lengthens" the MPL, allowing it to catch over the medial trochlear ridge. This is observed in young horses beginning training with insufficient muscle tone and in horses abruptly taken out of training and confined to a stall.
- Higher medial hoof wall and elongated toes have been reported to cause hyperextension of the stifle and outward rotation of the limb, contributing to UFP.
- Craniodorsal luxation of the coxofemoral joint predisposes to UFP by causing excessive straightening of the hindlimb.

### *Clinical Signs*

- With acute UFP, the hindlimb is locked in extension and the stifle cannot be flexed. When the horse is forced to move forward with the limb locked, it drags the front of the hoof on the ground. The condition may correct itself or remain locked for several hours.
- Intermittent UFP is described as intermittent "catching" of the patella as the horse walks or jogs. Walking the horse downhill, backing it, or moving it in a tight circle usually exacerbates the signs. When the MPL releases, the hindlimb usually jerks up quickly, mimicking stringhalt. UFP must be differentiated from stringhalt. See the accompanying DVD for a visual illustration.
- Usually both hindlimbs are potentially affected, but unilateral UFP can occur.
- Palpation of the limb when locked in extension reveals tense patellar ligaments and the patella locked above the medial trochlear ridge of the femur.
- Lameness usually is not present unless the condition is very chronic.

### *Diagnosis*

- The diagnosis is usually based on the characteristics of the gait and limb posture.
- Radiographs of the stifle should be taken to eliminate conditions that may predispose to UFP but radiographic abnormalities are uncommon.

### *Treatment*

- Horses with a persistent UFP that repeatedly re-occurs after manually unlocking the patella often require an MPL desmotomy to resolve the problem.
- Less severely affected horses respond to controlled conditioning to increase quadriceps strength and tone, which tightens the MPL. Conditioning, including going up hills without coming back down the incline (which exacerbates the hyperextension of the stifle,) strengthens and tones the quadriceps.
- Shortening the toe and lowering the medial hoof wall sufficient to move break-over medial to the toe may help alleviate UFP in some horses.
- Other treatments for UFP include "tightening" the MPL by splitting the ligament in multiple locations or injecting counterirritants(2% iodine in almond oil). This results in substantial thickening of the MPL, presumably causing a functional shortening and tightening of the MPL.
- If all treatments fail to correct the UFP, an MPL desmotomy is performed using local anesthesia in the standing horse. Fragmentation of the distal patella is a risk but is less likely to occur in horses that have persistent UFP.

### *Prognosis*

- The prognosis is very good for horses that respond to a conditioning program and maintain that level of fitness.
- Most horses also do well following injection of a counterirritant or medial patellar ligament splitting procedure.
- Horses that truly require an MPL desmotomy also have a very good prognosis and are unlikely to have complications if they receive a 60- to 90-day convalescence following surgery.

## SUBCHONDRAL CYSTIC LESIONS (SCLs) OF THE STIFLE

### *Introduction*

- SCLs within the stifle are nearly always located on the medial femoral condyle but can occur on the lateral femoral condyle and the proximal tibia (Figure 7.28 and 7.29).
- SCLs of the medial femoral condyle can be one of the most difficult lameness conditions to treat in the horse.

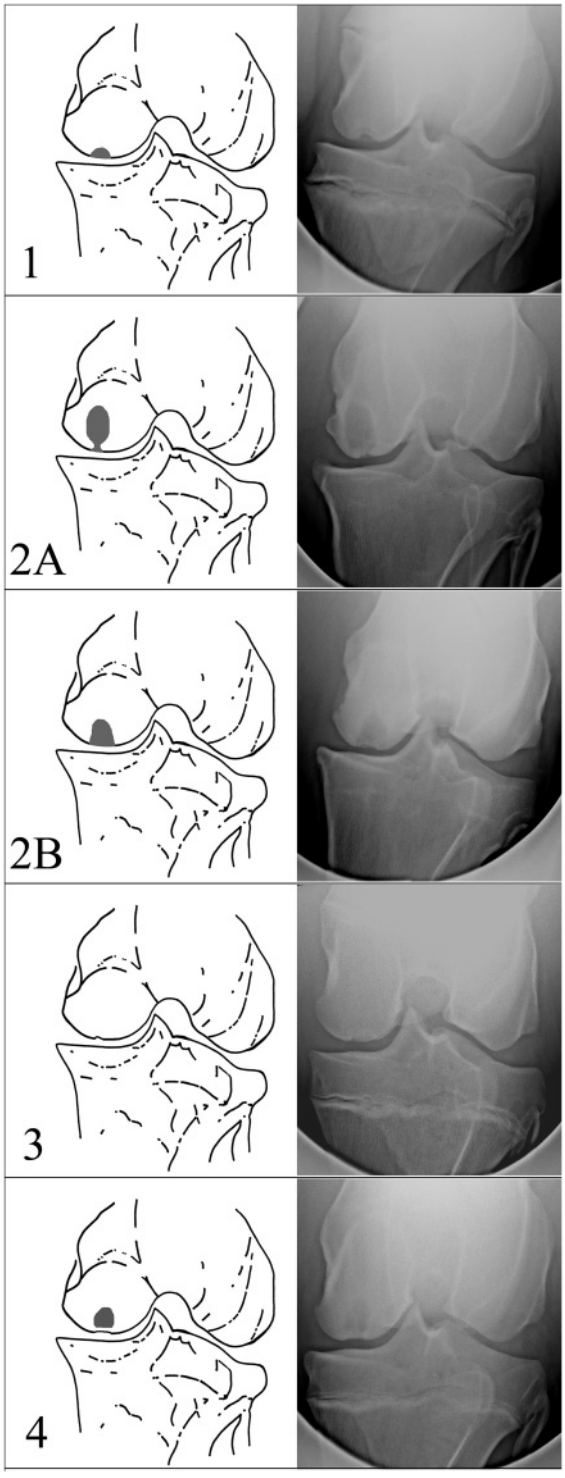


**Figure 7.28.** Grades of subchondral cystic lesions (SCLs). Reprinted with permission from Wallis TW, et al.: 2008. *Equine Vet J* 40:461–467.

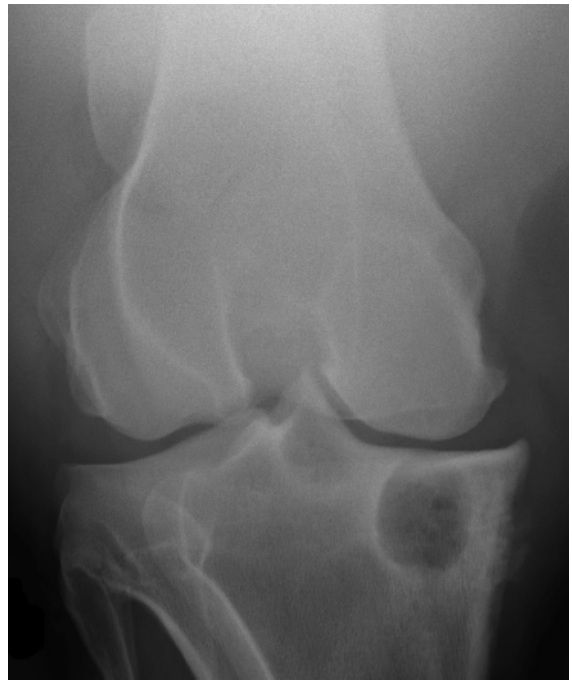

**Figure 7.29.** A caudal-cranial radiographic image of a proximal tibial SCL with concurrent signs of OA within the MFT joint.

- The presence of concurrent OA in the medial femorotibial (MFT) joint has been associated with a reduced prognosis.

## *Etiology*

- Most SCLs in the stifle are developmental in origin and are considered a type of developmental orthopedic disease (DOD) because they occur in young horses and are often bilateral.
- There is also both clinical and experimental evidence that SCLs can occur after trauma to the subchondral bone on weight-bearing articular surfaces. The SCL is usually unilateral, and concurrent abnormalities such as OA or meniscal problems are often present in the MFT joint (Figure 7.30).
- The lining of SCLs is thought to contain significant inflammatory mediators that may be responsible for enlargement and persistence of the SCL.

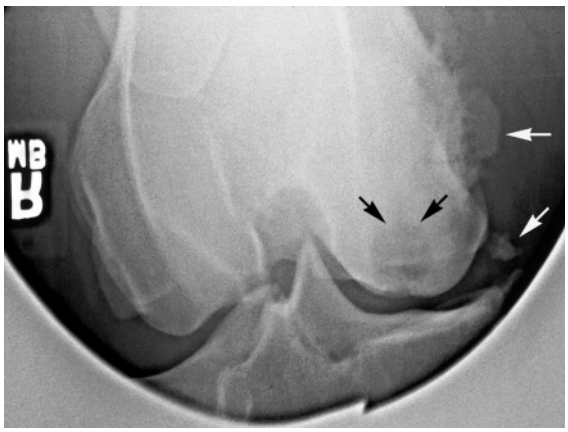

**Figure 7.30.** Caudal-cranial radiograph of the stifle of an 8-year-old Quarter horse mare with multiple radiographic abnormalities within the MFT joint (black arrows indicate a SCL; top white arrow indicates osteophyte production along the femur; bottom white arrow indicates mineralization of the medial meniscus). Concurrent medial meniscal injuries should be suspected in these types of horses.

### *Clinical Signs*

- The clinical presentation of horses with SCLs can vary from no lameness with the lesion being an incidental finding on radiographs to severe, debilitating lameness with multiple abnormalities within the MFT joint.
- Most horses with stifle SCLs become lame around the beginning of training, and a mild to moderate degree of lameness is usually present.
- Effusion may or may not be present within the MFT joints and the femoropatellar joints and horses are usually positive to upper limb flexion.
- In older horses that have been in training and competition, it is important to perform IA analgesia to prove the significance of a SCL.

### *Diagnosis*

- Radiography is the standard method of imaging for an accurate diagnosis (Figure 7.28). The caudo-cranial view is usually most diagnostic, but most SCLs can also be seen on a flexed lateral radiograph of the stifle.
- At minimum, a caudo-cranial view of the opposite stifle should always be obtained.
- A variety of shapes and sizes of lesions can be found. A grading scheme (Types 1 to 4) has been developed to help describe the different shape, size, depth, and articular involvement of the SCLs of the medial femoral condyle.
- The radiographic appearance of SCLs can change over time and should be monitored, especially in young horses.
- Concurrent radiographic signs of OA such as periarticular osteophytes and joint space narrowing are often seen in older horses.
- Ultrasound can be used to evaluate the articular surface, joint effusion, debris, and changes that may occur in the meniscus and MFT joint. SCLs typically show an irregularity on the condylar surface and thickening of the articular cartilage (Figure 7.31).

### *Treatment*

- Conservative therapy (confinement, IA and systemic medications) can be used in horses less than 1 year of age or in SCLs that are either small, have a small articular component, or may show limited communication between the SCL and the joint surface. Continued lameness and/or SCL enlargement are indications for surgical treatment.
- Surgical treatment options include debridement of the cyst contents, injection of the cystic lining with corticosteroids, or grafting of the debrided cyst with various products.

### *Prognosis*

- The overall prognosis has been estimated to be 56% to 77% of horses returning to their intended use with surgery.
- Horses in which more than 15 mm of the surface of the joint is involved have a reduced prognosis.
- Horses older than 3 years of age have a lower chance of soundness and return to work than horses 3 years of age and younger.
- The presence of articular cartilage lesions within the MFT joint remote to the site of the SCL reduces the prognosis.
- Damage to the menisci and/or meniscal ligament can further reduce the prognosis of affected horses.
- Horses with unilateral lesions have an improved prognosis compared to those with bilateral lesions.
- The presence of osteophytes within the MFT joint is thought to reduce the overall prognosis.

## MENISCAL INJURIES

### *Introduction*

- Horses of any breed and use are susceptible to meniscal lesions, especially horses that jump.

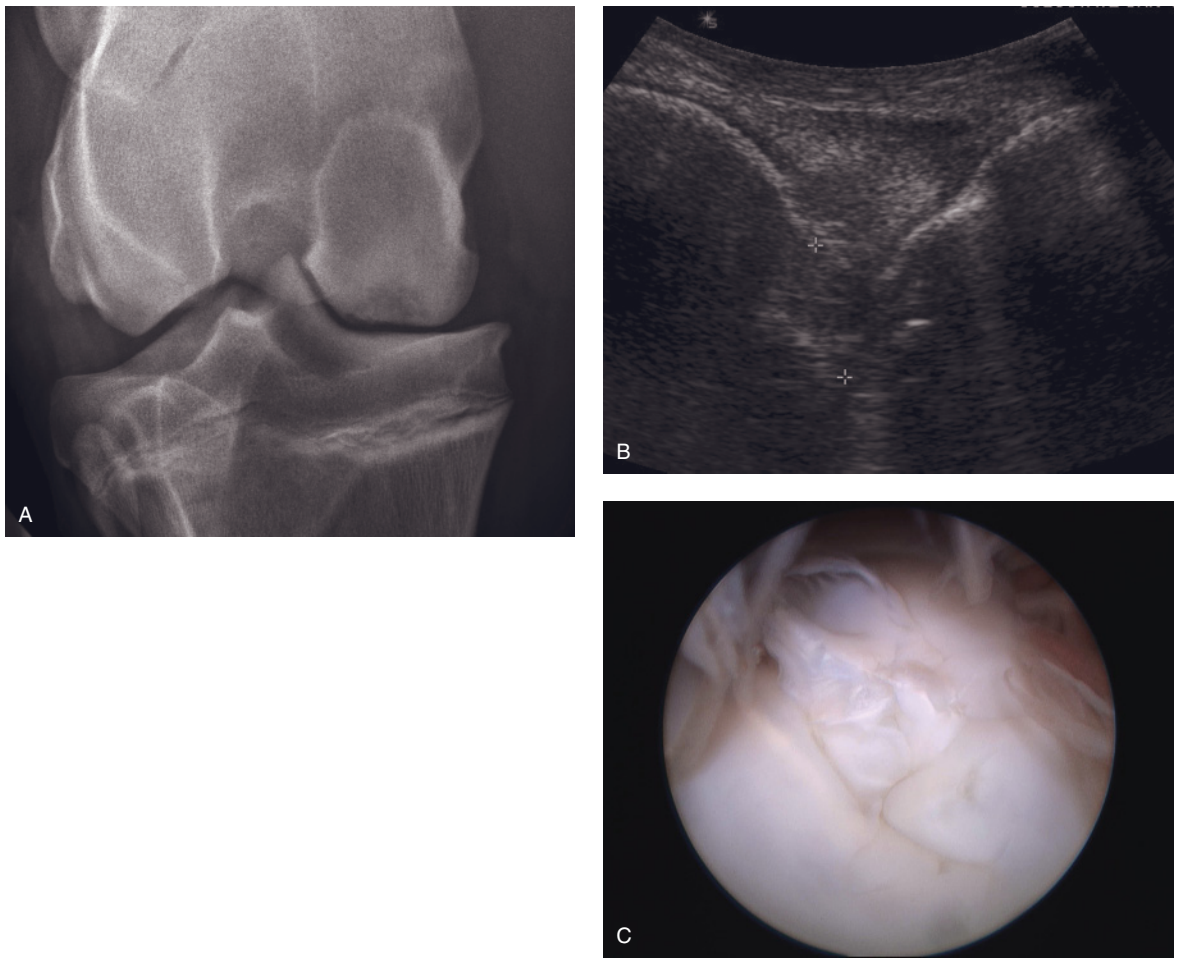


**Figure 7.31.** Radiograph (A), ultrasound (B), and intraoperative (C) images demonstrating an SCL that involved a large percentage of the articular surface. Note the joint space narrowing on the radiographic image (A), and the presence of the SCL as demonstrated by a defect in the subchondral bone on the ultrasonographic image (B). Thickening of the articular cartilage can be seen arthroscopically (C). Courtesy of Chris Kawcak.

- Western performance and sport horses have a high incidence of stifle problems, and consequently are predisposed to secondary meniscal lesions.
- Although both lateral and medial meniscal lesions occur, damage to the medial meniscus is most common.
- The medial meniscus in the right hindlimb is most commonly injured for unknown reasons.

### *Etiology*

- Meniscal lesions can be acute or chronic in nature.
- Acute damage can occur with a bad step or some sort of accident that leads to shifting or shear forces between the femur and tibia.
- It is not uncommon to see concurrent lesions in other structures such as the cruciate ligaments, articular cartilage, and medial femoral condyle (SCL).

### *Clinical Signs*

- Lameness and other clinical signs can be acute or insidious in onset.
- Lameness is usually moderate in severity and worsens with exercise and stifle flexion tests.

- MFT or femoropatellar joint effusion is present in about 50% of cases but most respond to IA anesthesia.
- Horses with severe lameness and evidence of chronic OA of the MFT joint should be suspected to have a concurrent medial meniscal injury (Figure 7.30).

### *Diagnosis*

- Radiographs of the stifle can be normal in acute cases of primary meniscal damage.
- Chronic damage to the meniscus may lead to joint space narrowing, mineralization of the meniscus, and OA within the MFT joint (Figure 7.32).
- Horses with meniscal damage appear to develop new bone formation at the medial intercondylar eminence of the tibia (Figure 7.33).
- Ultrasonographic findings can include characterization of an abnormal size, thickness, variability in echogenicity, and location of the meniscus; prolapsing of the meniscus medially; and overt meniscal tearing.
- It is important to image the meniscus in both weight-bearing and non-weight-bearing stances to better demonstrate the tear (Figures 7.34, 7.35).
- MRI can also be of use, but is limited in availability.
- Arthroscopy is an important diagnostic tool and is readily available, but much of the weight-bearing surface of the meniscus is not visible.
- CT arthrography of the stifle may provide improved imaging of the meniscus in the future.

### *Treatment*

- Treatment usually involves arthroscopic surgery and debridement of the disrupted meniscal fibers that can be seen. Lack of a visible lesion does not indicate the absence of meniscal damage because much of the meniscus is not visible with the arthroscope.
- The lesions have been graded based on arthroscopic findings: Grade I = axial tearing through the cranial ligament of the medial meniscus and into the meniscus, Grade II = same as Grade I but all damaged tissue and the extent of the damage is visible arthroscopically, Grade III = severe tear that extends beneath the femoral condyle.
- The majority of lesions are usually either Grade I or Grade II.

### *Prognosis*

- Overall, 51% of the horses may become sound with surgery.
- Sixty percent of horse with Grade I tearing were sound compared to 65% of those with Grade II and 10% of those with Grade III.

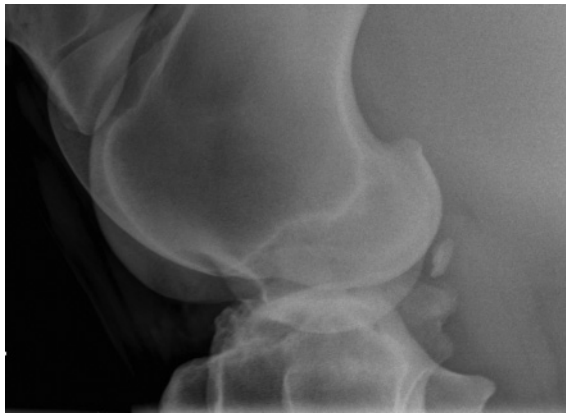

**Figure 7.32.** A lateromedial radiographic image demonstrating mineralization of the caudal aspect of the meniscus. Arthroscopic removal of this mineralization is rarely indicated because the OA is often advanced. Courtesy of Chris Kawcak.

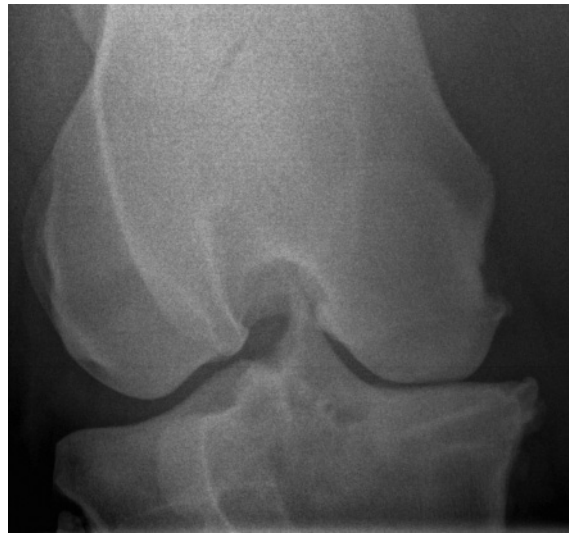

**Figure 7.33.** A caudal-cranial radiographic image showing osteophytes on the medial aspect of the tibia and the intercondylar eminence, which are common in many types of diseases involving the femorotibial joints. Courtesy of Chris Kawcak.

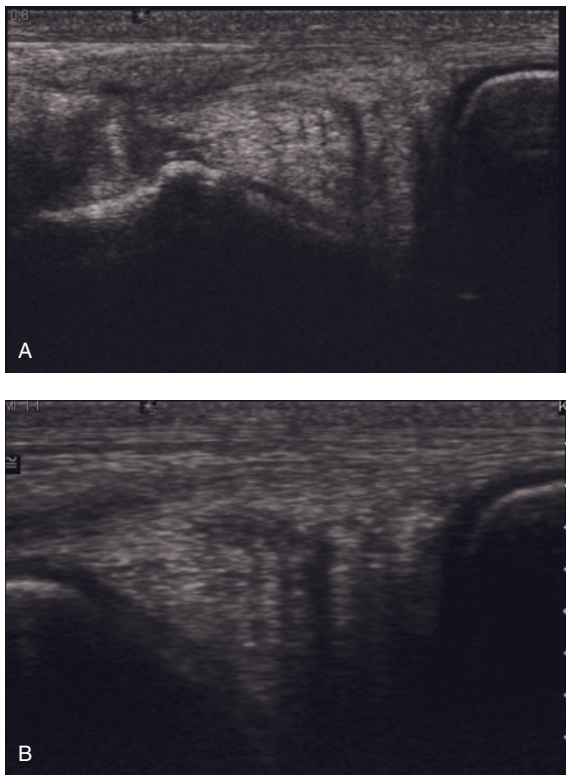


**Figure 7.34.** Ultrasonographic images of the medial meniscus in both weight-bearing (A) and non-weight-bearing positions (B). Notice how the meniscal tear becomes more apparent in the non-weight-bearing position. Courtesy of Chris Kawcak.

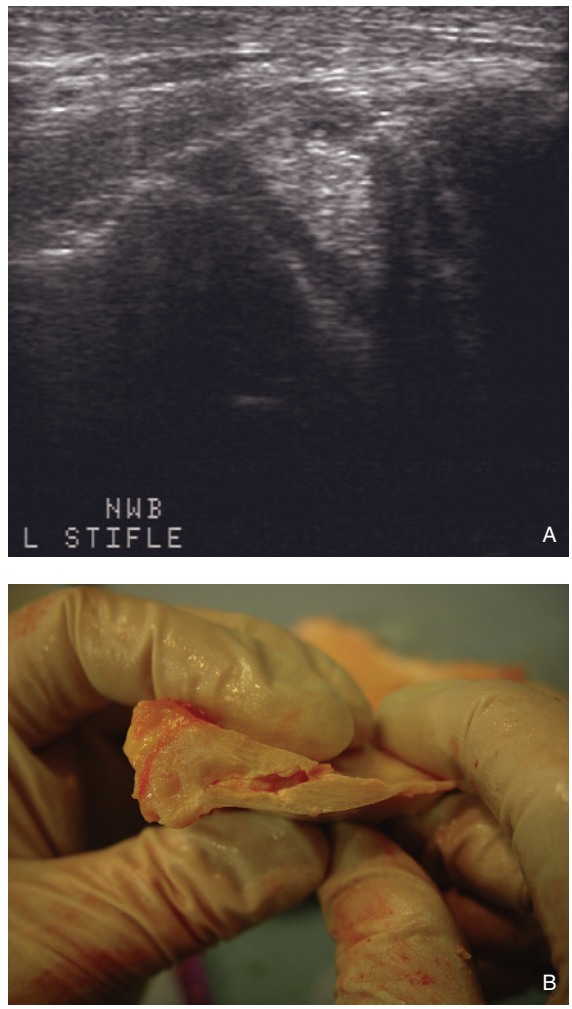


**Figure 7.35.** (A) Ultrasonographic image of a severe meniscal tear that correlated well with the gross appearance (B). Courtesy of Laurie Goodrich.

- The prognosis for lesions of the medial meniscus is worse than that for lateral meniscal lesions.
- Horses with small lesions that can be debrided can do relatively well, although horses with significant tearing carry a poor prognosis.

## COLLATERAL/CRUCIATE LIGAMENT INJURY

### *Introduction*

- The medial collateral and cranial cruciate ligaments are most commonly injured in the horse.
- Most injuries are seen in adult horses.
- Combinations of injuries that involve the medial collateral ligament, cruciate ligament, and medial meniscus are possible.

### *Etiology*

- An acute traumatic event in which the limb was stressed in an abnormal direction is usually the cause, but this is often presumed.
- Partial degeneration of the cranial cruciate ligament may occur in jumpers and racehorses.

### *Clinical Signs*

- Clinical signs are usually acute and severe; however, minor injuries cause more subtle signs.

- Horses with cruciate injuries often present for an acute lameness, with significant stifle effusion and response to flexion.
- Horses with complete rupture of the medial collateral can show significant lateral movement of the distal limb and a palpable widening of the MFT joint space on the medial aspect of the stifle.
- A tibial thrust test in the caudal direction may worsen the lameness, but this is not considered specific for cranial cruciate ligament damage.

### *Diagnosis*

- Often, radiographic findings are normal; however, the MFT joint may distract on a stressed caudocranial view (Figure 7.36).
- Radiographs are usually unremarkable with cruciate ligament injuries unless the origin of the ligament avulses from the intercondylar fossa, a midbody tear shows dystrophic mineralization, or a medial tibial eminence fragment is present (Figure 7.37).
- A medial tibial eminence fragment is not pathognomonic for cranial cruciate ligament damage because a fracture at the apex of the eminence may not involve the ligament.
- Ultrasound is usually the primary method of diagnosis for medial collateral ligament injuries but is more variable for the cranial cruciate ligament because it is difficult to image.
- Arthroscopic surgery is currently the best method to accurately diagnosis cranial cruciate ligament damage

### *Treatment*

- Horses with partial tears of the medial collateral ligament are treated conservatively; this may include rest, IA medication, topical anti-inflammatory medication such as diclofenac acid, and extracorporeal shockwave therapy.
- Horses with a complete rupture of the collateral ligament should be confined a stall for a minimum of eight to 12 weeks.
- Osteochondral fragments from the tibial eminence associated with cruciate damage can be removed and the damaged ligament debrided arthroscopically.
- There are no techniques available to actually repair the cruciate ligament in horses.

### *Prognosis*

- Overall, the prognosis depends on the extent of damage to the ligament but is often considered poor to return to athletic use.
- Mild injuries to the ligament can be successfully treated in some horses.

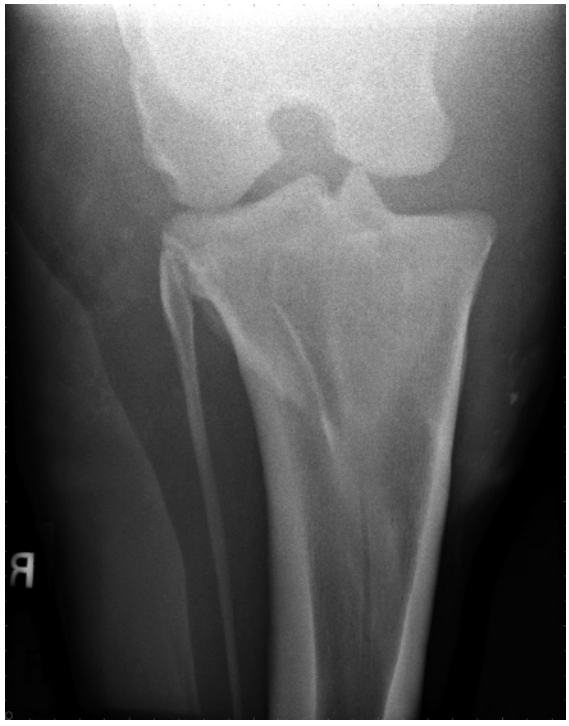

**Figure 7.36.** Caudal-cranial radiographic image of a stifle with a ruptured medial collateral ligament. Notice the widened joint space medially. Courtesy of Chris Kawcak.

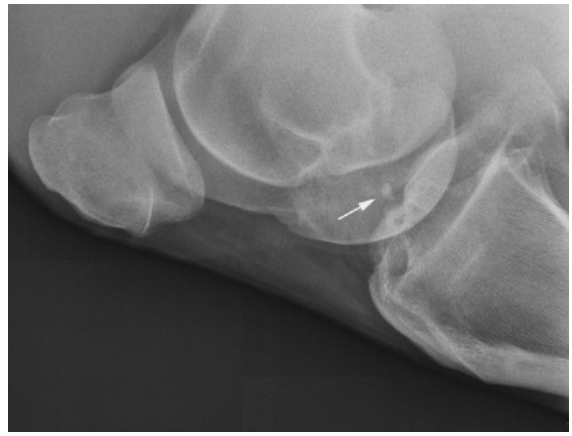

**Figure 7.37.** A flexed lateromedial radiographic image that demonstrates fragmentation of the intercondylar eminence of the tibia (arrow). The fragment was attached to the cranial cruciate ligament, but was loose during arthroscopic examination.

## SYNOVITIS/CAPSULITIS/OA OF THE STIFLE

### *Introduction*

- The MFT joint is most commomly affected.
- MFT joint synovitis is diagnosed commonly in Western performance horses, especially young cutting horses.
- Severe MFT joint OA is usually associated with other IA injuries (Figures 7.30, 7.33).

### *Etiology*

- Horses that undergo chronic repetitive stress to the hindlimbs, such as young Western performance horses, are susceptible to synovitis of the MFT joint. This situation is similar to racehorses that develop synovitis in their MCP and carpal joints.
- A primary source of synovitis usually is not apparent in many horses.
- Acute trauma to the stifle of any type leading to IA trauma can contribute to OA in any of the stifle joints.
- Repetitive use of high dosages of corticosteroids together with continued hard exercise may also predispose to articular cartilage deterioration.

### *Clinical Signs*

- Horses with synovitis of the femorotibial joints may not be lame, but have mild to moderate effusion in the MFT joint (Figure 3.43). These horses may show some response to flexion and have a gait that can be described as "stiff".
- Horses with mild OA or synovitis secondary to other primary lesions within the MFT joint usually have a history of mild lameness or a lack of performance.
- Horses with significant OA of the MFT joint are usually noticably lame with loss of muscle mass and effusion of the MFT joint. Thickening of the MFT joint capsule (medial butress) is not uncommon. Concurrent abnormalities such as SCL of the medial femoral condyle and medial meniscal damage are not uncommon.

### *Diagnosis*

- All diagnostic techniques, including arthroscopy, can be negative in cases of primary synovitis/capsulitis.
- Osteophytes on the medial tibial plateau are an early sign of MFT OA (Figure 7.33).
- Advanced OA of the MFT joint is evidenced by osteophytes on the tibial plateau, intercondylar eminence, and medial femoral condyle, with or without joint space narrowing (Figure 7.33).
- Other diagnositics that can be useful include ultrasonography and arthroscopy. Arthroscopy is best technique to evaluate the articular cartilage.

### *Treatment*

- Horses with synovitis usually respond well to IA medication, topical anti-inflammatories, and systemic joint medications. Rest or a reduction in training are often recommended and horses typically respond well to this.
- Failure to respond to medication or recurrence of lameness are often the key findings that lead to more intensive imaging and possibly diagnostic arthroscopy.
- Treatment of OA of the MFT joints is the same as for other joints. Intermittent IA medication and controlled exercise appear to help.
- Arthroscopic surgery may help reduce the progression of OA by removing debris and fibrillated articular cartilage, but the disease process is likely to progress.

### *Prognosis*

- Horses with synovitis are easily managed medically as long as a primary disease process is not present. However, there is concern that chronic, persistent synovitis can lead to secondary damage to the articular cartilage, which may predispose to the onset of OA in the future.
- The prognosis for OA of the femorotibial joints depends on the severity and appears no different than for other joints in the horse. However, severe OA can lead to significant lameness with a questionable quality of life.

## FIBROTIC MYOPATHY

### *Introduction*

- Fibrotic myopathy refers to fibrosis with or without ossification of the muscle tissue in the crus that can involve the semitendinosus, semimembranosus, gracilis, or biceps femoris muscles (Figure 7.38).
- The fibrosis and adhesions limit the action of the semitendinosus muscle, causing an abnormal gait characterized by a "slapping" down of the foot at the end of the cranial phase of the stride (Figure 7.39).
- The lesions are usually unilateral.

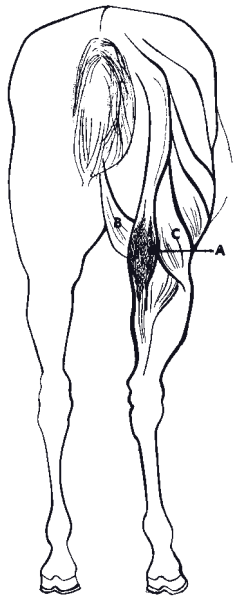


**Figure 7.38.** Drawing depicting the muscles in the fibrotic area in the gaskin of a horse affected with fibrotic myopathy. (A) Semitendinosus. (B) Semimembranosus. (C) Biceps femoris.

**Figure 7.39.** Characteristic action of the hindlimb of a horse affected with fibrotic myopathy. The limb jerks backward and downward (dotted outline of the affected foot) during the last 3 to 5 inches of the stride, resulting in slapping of the foot on the ground.

### *Etiology*

- The cause of the fibrosis is nearly always trauma, but the severity can range from a single severe injury to repetitive microdamage to the muscles due to exercise.
- Involved muscles may be injured during sliding stops in rodeo work, from slipping, getting the hindlimb caught in a fixed object or a halter, and from being kicked.
- The lack of muscle compliance and adhesions between the involved muscles that occurs with healing contribute to the gait abnormality. Ossification is believed to be a more severe progression from the fibrosis.
- Neurogenic atrophy of the affected muscles associated with a peripheral neuropathy has also been suggested as a potential cause.

### *Clinical Signs*

- With acute injuries, lameness in the injured limb together with swelling and pain on palpation of the hamstring region are common findings. A gait abnormality is usually not present.
- With chronicity, the swelling and pain subside and the classic gait abnormality develops gradually as muscle healing occurs. Lameness may or may not be present.
- In the cranial phase of the stride, the foot of the affected hindlimb is suddenly pulled caudally 3 to 5 inches just before contacting the ground (Figure 7.39). Usually the gait abnormality is most noticeable when the horse walks. See the DVD for examples of the gait abnormality.
- An area of firmness/fibrosis can often be palpated over the affected muscles on the caudal surface of the affected limb at approximately the level of the stifle (Figures 7.38 and 7.40).

### *Diagnosis*

- The diagnosis is usually based on the characteristic gait abnormality together with palpation of abnormal musculature within the hamstring region.
- Ultrasonography can be used to determine the severity of the muscle injury and whether ossification is present.

### *Treatment*

- With acute injuries, preventing permanent fibrosis of the muscles is the goal. Icing, sys-

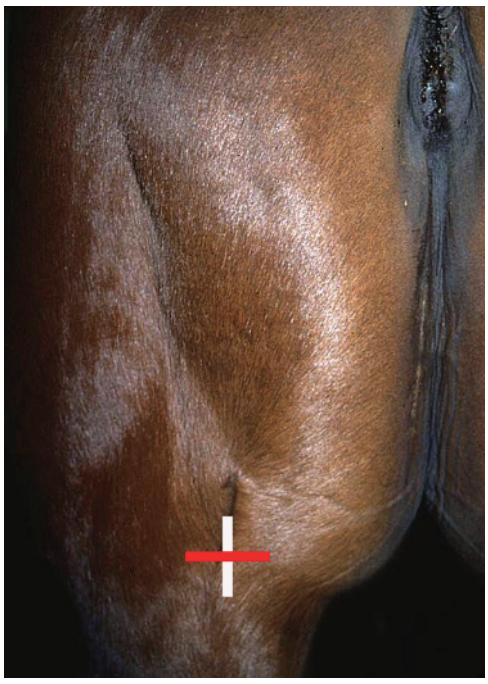

**Figure 7.40.** Caudal thigh region where the myotomy procedure is performed to correct fibrotic myopathy. The skin incision is made vertically (white line) and the fibrotic muscle is transected with a bistoury horizontally (red line).

temic and topical NSAIDs, and restricted exercise are important to reduce the inflammation and prevent further muscle injury.
- Physical therapy exercises to stretch the hamstring region as the muscles heal will help prevent adhesions and shortening of the muscle-tendon unit.
- Surgical treatment for fibrotic myopathy consists of either a semitendinosus tenotomy at the level of its insertion on the proximal medial tibia (requires general anesthesia) or a semitendinosus myotomy performed at the site of the fibrosis on the caudal aspect of the limb
- With the myotomy, the incision is made directly over the affected semitendinosus muscle, which is transected using a blunt bistoury (Figure 7.40).

### *Prognosis*

- Some immediate improvement may be evident with the myotomy, but it usually takes three to seven days for the maximum effect.
- With the standing myotomy technique, 83% of horses were able to perform at their pre-injury level, although the restrictive gait pattern did not resolve in all horses.
- Results for the tenotomy procedure are based on few cases (four of six improved) but the procedure is generally considered to be beneficial.
- The prognosis for successful surgery in horses affected by neurogenic atrophy/fibrosis of the muscles is considered poor.

## DIAPHYSEAL AND METAPHYSEAL FEMORAL FRACTURES

### *Introduction*

- Fractures of the femur are relatively common in horses, especially young horses.
- In young animals, fractures often involve the proximal or distal growth plate, and diaphyseal fractures are usually oblique and spiralling.
- Adult horses often sustain irreparable comminuted fractures of the femoral shaft.
- Treatment of femoral fractures (internal fixation or confinement) is usually only recommended in young horses weighing less than 200 to 300 kg.

### *Etiology*

- Foals frequently sustain femoral fractures during initial handling or during halter breaking.
- Occasionally a mare will step on a foal, or the foal may become trapped under a fence. In a study of 38 horses under 1 year of age, causes included a fall, severe adduction, external trauma, or being caught in a fence.
- A severe traumatic event such as a fall is often the cause in adult horses.
- Lameness or age-related osteoporosis may contribute to femoral fractures in older horses. (Figure 7.41)

### *Clinical Signs*

- The obvious sign is non-weight-bearing lameness.
- When viewed from the side, the affected limb may appear slightly shortened with the hock held higher than the opposite hindlimb, and it may be externally rotated.
- Fractures of the distal femoral metaphysis may have swelling around the stifle, mimicking a stifle injury.

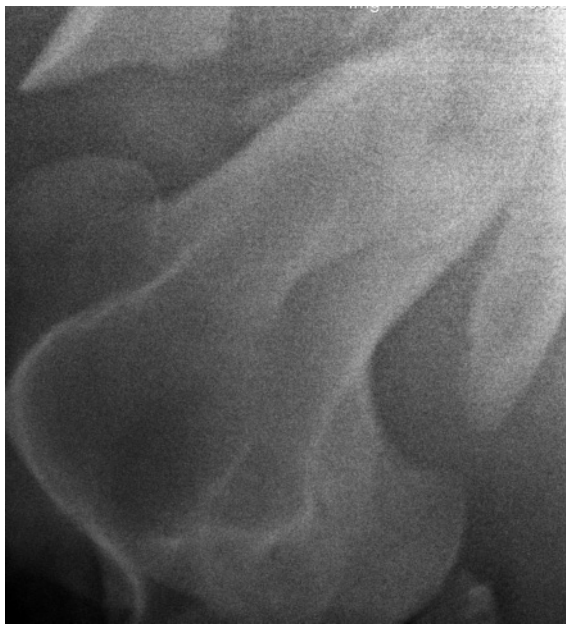

**Figure 7.41.** A lateromedial radiograph showing a comminuted fracture of the femur in a 23-year-old mare. This mare had severe lameness in the stifle of that limb since she was a yearling, and fractured it while getting up in her stall. It is likely that the bone was osteoporotic due to chronic, reduced weight-bearing from the lameness. Courtesy of Chris Kawcak.

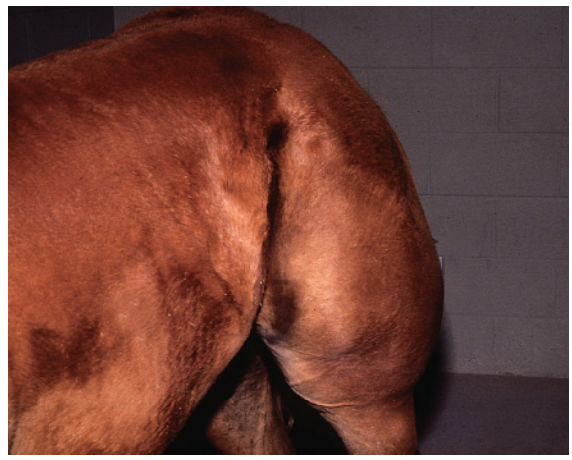

**Figure 7.42.** Cranial view of a yearling with severe swelling of the femoral and stifle region associated with a diaphyseal femoral fracture. The limb is also externally rotated.

- Obvious swelling in the mid-femoral region is usually present with diaphyseal fractures (Figure 7.42).
- Manipulation of the limb often reveals crepitus and laxity of the limb with complete diaphyseal fractures.

### *Diagnosis*

- A tentative diagnosis can usually be made based on the excessive movement of the distal limb, moderate to severe swelling, and the presence of crepitus.
- Radiographs in the standing patient can usually confirm the diagnosis. Radiographs are important to demonstrate the exact location and configuration of the fracture because these factors affect the treatment options available.
- When radiographs are not possible or advisable, ultrasound may demonstrate cortical disruption of the diaphysis and fractures of the capital physis.

### *Treatment*

- Euthanasia is indicated for most adult horses that have sustained femoral shaft fractures (Figure 7.41).
- Diaphyseal fractures have been treated with stall rest in foals weighing up to approximately 200 kg, but malunion is a risk.
- Compression plating (usually two plates) is the treatment of choice for foals with diaphyseal fractures when athletic soundness is desired.
- Intramedullary pinning using the stacked pin technique or interlocking nails have also been reported to treat diaphyseal fractures in young foals.
- Minimally displaced distal physeal fractures may heal with conservative therapy (Figure 7.43).
- Unstable distal physeal fractures can be treated with an angled blade plate, condylar buttress plate, cobra-head and dynamic compression plate, and cross-pins or Rush pins

### *Prognosis*

- Femoral fractures in horses older than yearlings carry a very poor prognosis for a successful outcome.
- Approximately 50% of foals treated surgically were successful in one report. The mean age for successfully treated foals was 2 months vs. 4 months for unsuccessfully treated foals.

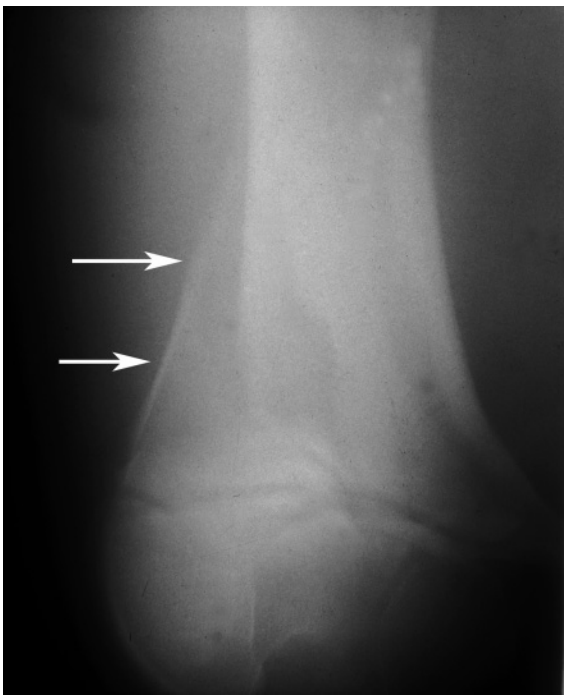

**Figure 7.43.** Lateral radiograph of a weanling with a Type II distal femoral fracture (arrows) that was successfully treated with confinement.

- Three of four foals with oblique mid-shaft femoral fractures treated by stall rest alone became sound for breeding in one study.

## CAPITAL PHYSEAL FRACTURES OF THE FEMORAL HEAD

### *Introduction*

- Fractures of the capital physis of the femoral neck occur commonly in foals less than 1 year of age.
- They are usually a Type 1 Salter-Harris physeal fracture but Types 2 and 3 are also observed.
- Another term that has been used to describe these fractures is a "slipped capital physis" (Figure 7.44).

### *Etiology*

- Trauma such as violent falls, struggles, and kicks are the cause.
- Falling on the greater trochanter is thought to cause shearing forces across the physis, resulting in displacement between the epiphysis and metaphysis.

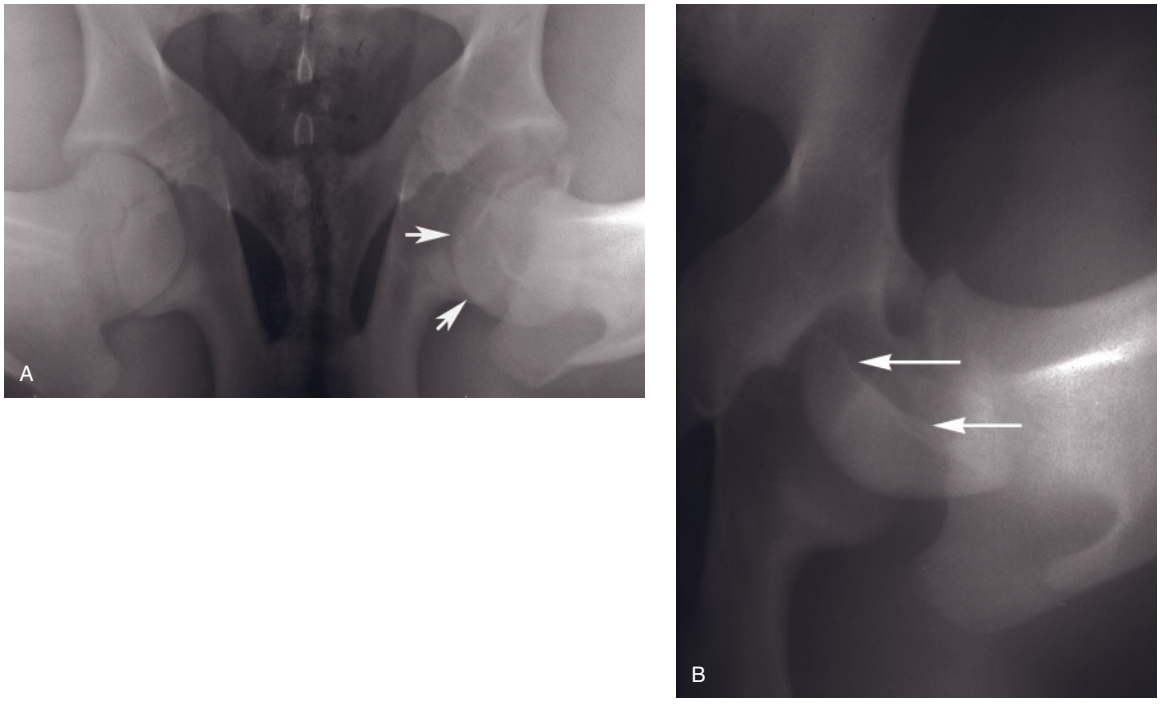


**Figure 7.44.** Recumbent radiographs of the pelvis of two different foals with capital physeal fractures (arrows) of the left proximal femur.

### Clinical Signs

- Immediate severe lameness after a violent accident is usually present but the lameness can improve within a few days.
- Some foals are able to bear some weight but often stand with a toe-out, hock-in appearance.
- Swelling and pain over the hip, pelvic asymmetry, gluteal muscle atrophy, and crepitus with hip manipulation may be present but are not consistently seen.

### Diagnosis

- Radiography of the hip, usually under general anesthesia, is necessary for an accurate diagnosis. Separation of the epiphysis and metaphysis of the femoral neck confirms the diagnosis (Figure 7.44).
- Ultrasonography of the hip in the standing horse also may confirm the diagnosis

### Treatment

- Nonsurgical management of these fractures is not recommended because malunion, avascular necrosis, and secondary hip OA can lead to debilitating lameness.
- Surgical treatment is difficult and is not recommended in foals with concurrent radiographic abnormalities.
- Surgical options include the use of cancellous or cortical bone screws, IM or Knowles pins, and an interfragmentary compression system. Small foals with a Type 1 or 2 physeal fracture are the best candidates for surgery.

### Prognosis

- The majority of foals with this condition are euthanized because of the poor prognosis with nonsurgical treatment and the questionable results with surgery.
- If the epiphysis can be reduced and maintained until healing, athletic soundness can be achieved.

## COXOFEMORAL LUXATION (DISLOCATION OF THE HIP JOINT)

### Introduction

- Luxation of the coxofemoral joint is an uncommon condition in horses compared to cattle because of the numerous ligaments and heavy musculature surrounding the joint.
- In horses, the ilium tends to fracture before the hip luxates.
- Foals, miniature horses, and ponies are most frequently affected.
- Coxofemoral luxation is unilateral and the head of the femur nearly always becomes craniodorsal to the acetabulum.

### Etiology

- Both the accessory and the ligament of the head of the femur (round) must rupture for a luxation to occur. Some type of violent trauma is nearly always the cause.
- Violent overextension and falling on the point of the stifle with the femur in a vertical position occasionally causes luxation of the coxofemoral joint.
- Luxation of the hip also may occur secondary to wearing a full-limb hindlimb cast, especially in foals.

### Clinical Signs

- A history of trauma resulting in a severe nonweight-bearing lameness is common.
- Some horses may toe-touch when walked because the affected limb is shorter than the opposite limb due to the craniodorsal position of the femur (Figure 7.45).
- The limb may "dangle" somewhat because of shortening, and the point of the hock on the affected side will be higher than that of the opposite limb. The toe and stifle turn outward and the point of the hock turns inward.
- Crepitus with limb manipulation may be present as a result of the femur rubbing on the shaft of the ilium.
- Coxofemoral luxations also may be complicated by upward fixation of the patella (Figure 7.45).

### Diagnosis

- A presumptive diagnosis can often be made based on the history and clinical signs.
- Standing or recumbent radiography confirms the diagnosis and also rules out other possible causes of the lameness such as pelvic, acetabular, and capital physeal fractures (Figure 7.46).
- Ultrasound of the pelvic region also may be able to confirm luxation of the femoral head.

### Treatment

- Many horses with this condition are euthanized because of the often unsuccessful treatment options and the poor prognosis.
- Closed reduction of the luxation is usually the best treatment option but can be very difficult

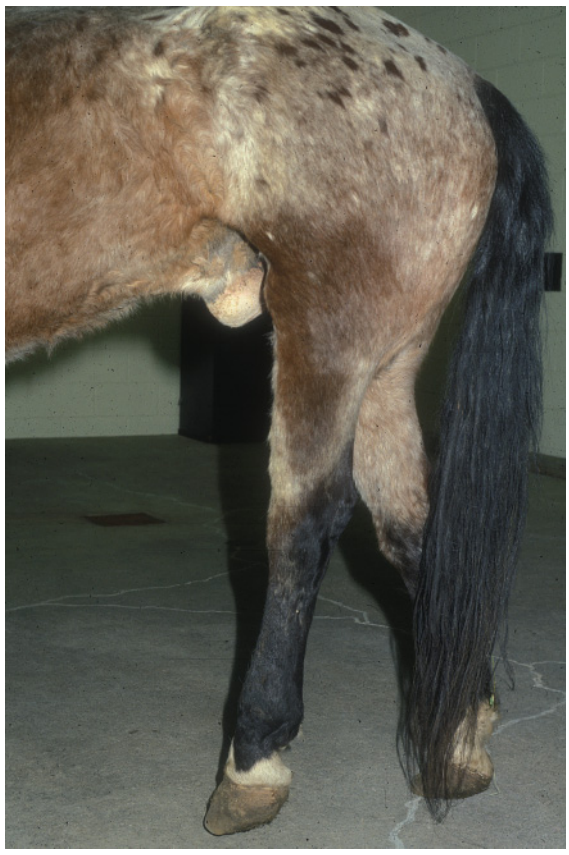

**Figure 7.45.** Horse with luxation of the hip and concurrent upward fixation of the patella. The craniodorsal positioning of the femoral head straightens the limb, contributing to upward fixation.

to perform in adult horses, and re-luxation is common. Horses with an acute luxation with no secondary fracture of the dorsal rim of the acetabulum are the best candidates for closed reduction.

- Several surgical approaches applicable for foals or small breed horses have been reported. They include open reduction alone, transposition of the greater trochanter, femoral head and neck resection, toggle pinning, or augmentation of the lateral joint capsule with synthetic sutures attached to screws.
- A combination of toggle pinning, synthetic capsular repair, and trochanteric transposition was used to successfully repair a hip luxation in an adult miniature horse.
- Some miniature horses may develop a pseudoarthrosis outside the coxofemoral joint without treatment.

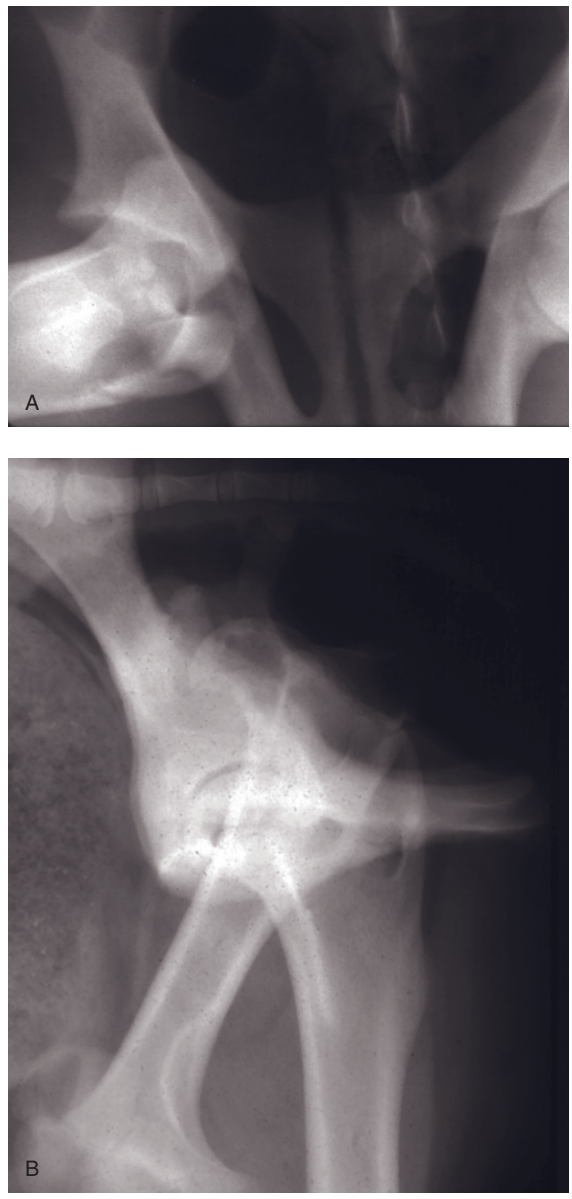


**Figure 7.46.** Ventrodorsal (A) and standing lateral (B) radiographs of the pelvis confirming luxation of the coxofemoral joint. The femoral head is outside the acetabulum in a craniodorsal location.

## *Prognosis*

- The prognosis is typically guarded to poor because successful closed reduction is not always possible.
- There is usually a better chance of maintaining permanent reduction if the femur stays in

place for approximately three months after treatment.
- Most horses can become sound enough for breeding purposes if the reduction can be maintained or a pseudoarthrosis develops.

## OA OF THE COXOFEMORAL JOINT

### *Introduction*

- OA of the coxofemoral joint is a sequel to almost any problem within the joint.
- It may occur from any type of soft tissue trauma to the hip region that does not necessarily result in an IA ligament injury or fracture.
- Hip OA is seen most frequently in older animals and should be considered in any horse with chronic hindlimb lameness.

### *Etiology*

- Any hip-related traumatic injury can lead to OA.
- Reported causes of coxofemoral joint OA include idiopathic infection, abnormal development of the coxofemoral joint, joint ill, and trauma.

### *Clinical Signs*

- Many affected horses have a significant lameness (grade 3 to 4/5) and have a low arc of foot flight and a reduced cranial phase of the stride.
- Horses with hip pain tend to move with the limb rotated externally and carry the limb abducted during advancement.
- Firm swelling over the greater trochanter and hip area may be present in chronic cases and the characteristic toe-out, hock-in stance may or may not be observed (Figure 7.47).
- Swelling over the hip region and pain on direct palpation over the hip may be observed in some horses (Figure 7.48).

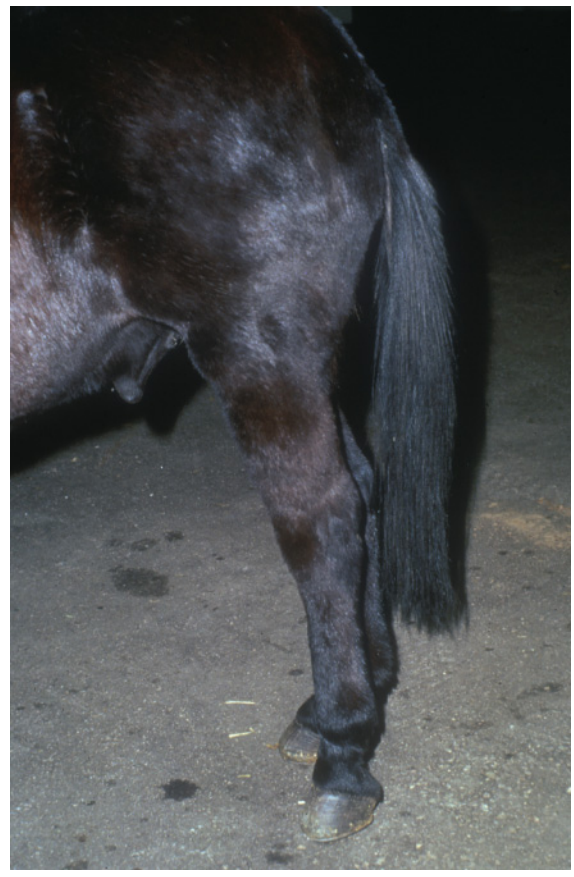

**Figure 7.47.** Lateral view of an aged pony with the typical toe-out, hock-in stance characteristic of a hip/pelvic problem. The left hindlimb is straighter than the contralateral limb and the horse is leaning to the right.

### *Diagnosis*

- Milder cases of OA can be a diagnostic challenge. Intra-articular anesthetic of the coxofemoral joint is the best method to localize the lameness to the hip.
- Nuclear scintigraphy may be helpful to localize the problem to the hip but is not very sensitive or specific for coxofemoral joint OA.
- Radiographs are often necessary for a definitive diagnosis and usually reveal evidence of bone remodeling and osteophytes production in chronic cases (Figure 7.49).
- Ultrasound also may detect evidence of OA with advanced disease.

### *Treatment*

- Treatment of hip OA is usually palliative because the lameness often progresses. Horses usually respond well to oral NSAIDs and/or IA treatment with HA and corticosteroids.
- Horses with mild or moderate hip OA may benefit from medications directed at joint healing such as IV HA, IM PSGAGs, and oral nutraceuticals.

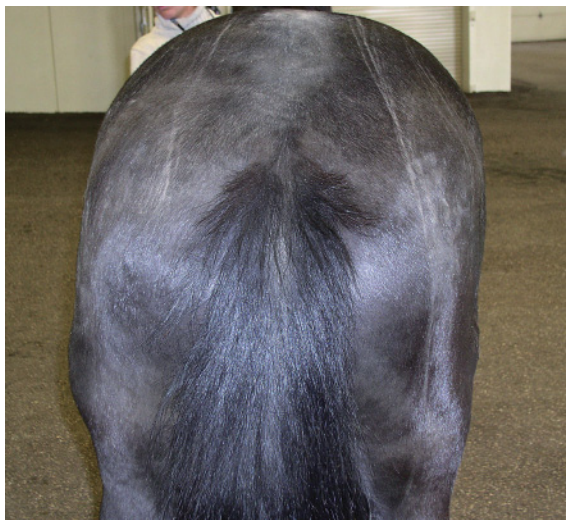

**Figure 7.48.** An older Quarter horse mare with grade 3/5 hindlimb lameness. Swelling over the left greater trochanter could be seen when compared to the opposite side, and pain was elicited with firm palpation.

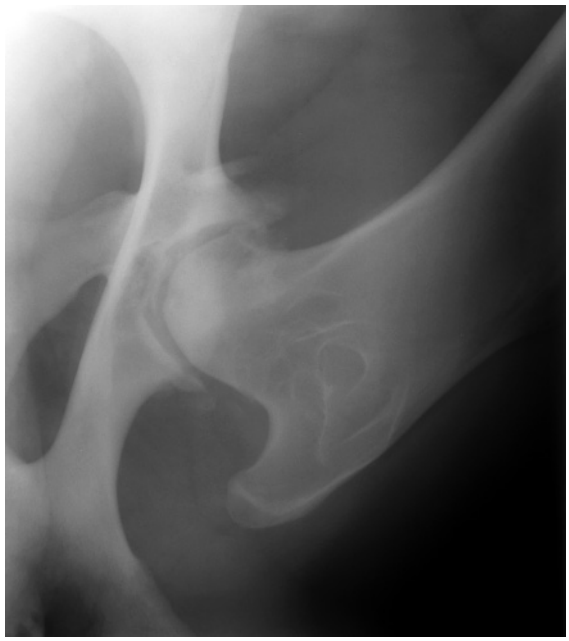

**Figure 7.49.** Ventrodorsal radiograph of the pelvis of the horse in Figure 7.48, demonstrating severe OA of the coxofemoral joint. The OA was reportedly secondary to trauma to the area from a previous fall.

- Confinement is usually not indicated because some type of controlled exercise often benefits horses with OA.

### *Prognosis*

- The prognosis for athletic use in horses with severe OA is thought to be poor. However, many of these horses can be used for breeding or maintained as pets because they tend to do well at the walk.
- Horses with mild or moderate OA may respond well to treatment and may be used at a reduced performance level.

## INFECTIOUS ARTHRITIS/PHYSITIS OF THE COXOFEMORAL JOINT

### *Introduction*

- Infection of the coxofemoral joint and the capital physis of the femur are part of the joint ill complex in foals.
- Hematogenous infections around the hip occur less frequently than at other sites in foals and can be very difficult to diagnose.

### *Etiology*

- Joint and physeal infections in foals are hematogenous in origin, and bacteria usually gain access to the circulation through the umbilicus, gastrointestinal tract, or respiratory tract.
- Affected foals usually have a history of failure of passive transfer and most infections are from Gram-negative bacteria.

### *Clinical Signs*

- Infection of the coxofemoral joint can be a diagnostic challenge because joint effusion, heat, and pain are often not found on physical examination.
- Typically these foals are less than 4 months of age and often present with a unilateral hindlimb lameness of unknown cause. These foals may have a characteristic toe-out, stifle-out, hock-in appearance.
- Pain is often elicited with deep palpation over the greater trochanter and hip region.
- It can often be confused with trauma to the limb, but the infection and therefore the lameness is often progressive, whereas a traumatic injury often improves with time.
- Young foals with severe hindlimb lameness without any definable lesions in the lower limb should be suspected of having infection of the hip joint.

### *Diagnosis*

- A complete blood count is often very helpful in differentiating trauma from a potential hip infection. High white blood cell counts and fibrinogen concentrations help confirm the presence of an infection.
- A definitive diagnosis is made by analyzing the synovial fluid for white blood cell counts of 30,000 or more cells/μL and total protein concentrations of 4 g/dL or higher.
- Physeal infections can be documented with radiographs of the hip region, where lysis around the capital physis, epiphysis, and occasionally the acetabulum is seen (Figure 7.50).

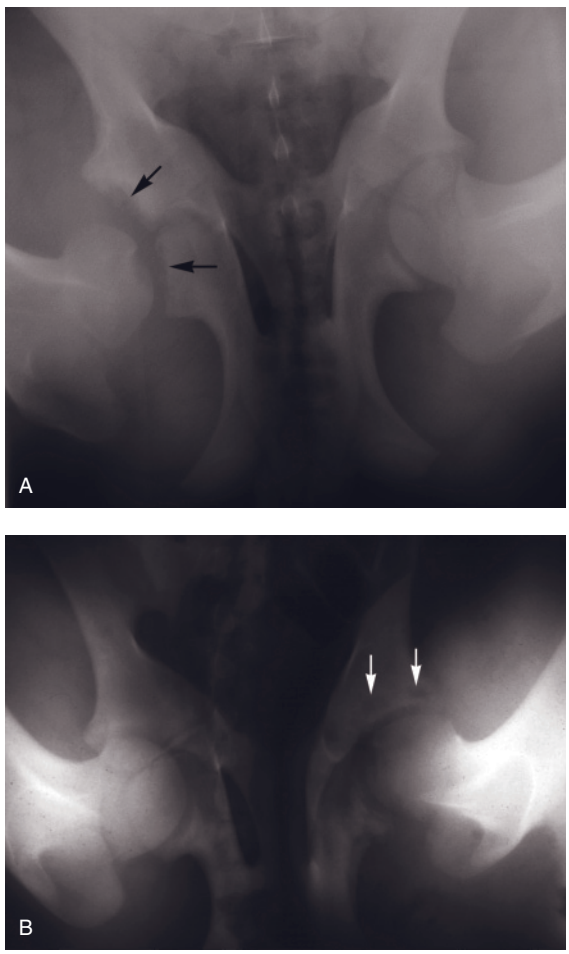


**Figure 7.50.** Ventrodorsal radiographs of the pelvis of two different 3-month old fillies with a history of increasing lameness. (A) Remodeling of the femoral head and acetabulum (arrows) consistent with a developmental abnormality. (B) Lysis of the left acetabulum (arrows) consistent with infectious arthritis and osteomyelitis.

### *Treatment*

- Treatment is similar as for any other joint/physis with a hematogenous infection. Broad-spectrum systemic antimicrobials, IA antimicrobials, and joint lavage/drainage are all important.
- Arthroscopy can be performed in small horses and foals to help lavage and debride these lesions, but cannula lavage alone can be very helpful.

### *Prognosis*

- The prognosis is usually poor because the diagnosis is seldom made before significant joint abnormalities and osteomyelitis have occurred.
- Foals with coxofemoral infectious arthritis without radiographic abnormalities should respond well to aggressive treatment and have a similar prognosis for athletic use as foals with joint ill at other locations.

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8

CHAPTER

# Common Conditions of the Axial Skeleton

## PELVIC FRACTURES

### *Introduction*

- Pelvic fractures are more common in young horses and are usually due to trauma.
- Ileal stress fractures are seen in racehorses.
- Pelvic fractures are nearly always unilateral but may involve multiple bones of the pelvis such as the ilium and pubis.
- Non-displaced fractures may be difficult to diagnose.
- Severe pelvic fractures in adult horses that involve the ilium can lacerate the iliac arteries and contribute to acute death.

### *Etiology*

- Most are single-event traumatic fractures from falling, slipping, fighting, and other types of accidents.
- Ilial fractures in racehorses are common stress-type fractures caused by repetitive overloading.

### *Clinical Signs*

- There is acute onset of lameness, which is usually severe (4 to 5/5).
- The horse may have a toe-out, hock-in limb conformation (Figure 8.1).
- There is often pain on deep palpation of the gluteal muscles and manipulation of pelvis.
- Crepitus may be audible with manipulation of the pelvis or pelvic limb.
- An alteration in height of the tuber coxae or tuber ischii may be visible.
- Palpable hematoma, fracture, or asymmetry of the pelvic canal may be found on a rectal exam.
- There may be differences in limb length; this is mostly seen with hip luxations.
- Gluteal muscle atrophy can occur quickly on the affected side with pelvic fractures.
- Chronic fractures are nearly always associated with gluteal muscle atrophy and outward rotation of the limb (Figure 3.49).

### *Diagnosis*

- The diagnosis may be made based strictly on history and clinical findings.
- Standing or recumbent radiography can be used to make the diagnosis (Figure 8.2).
- Percutaneous or transrectal ultrasonography is also useful to confirm the diagnosis.
- Scintigraphy can be used for ilial stress fractures or other types of non-displaced pelvic fractures.

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

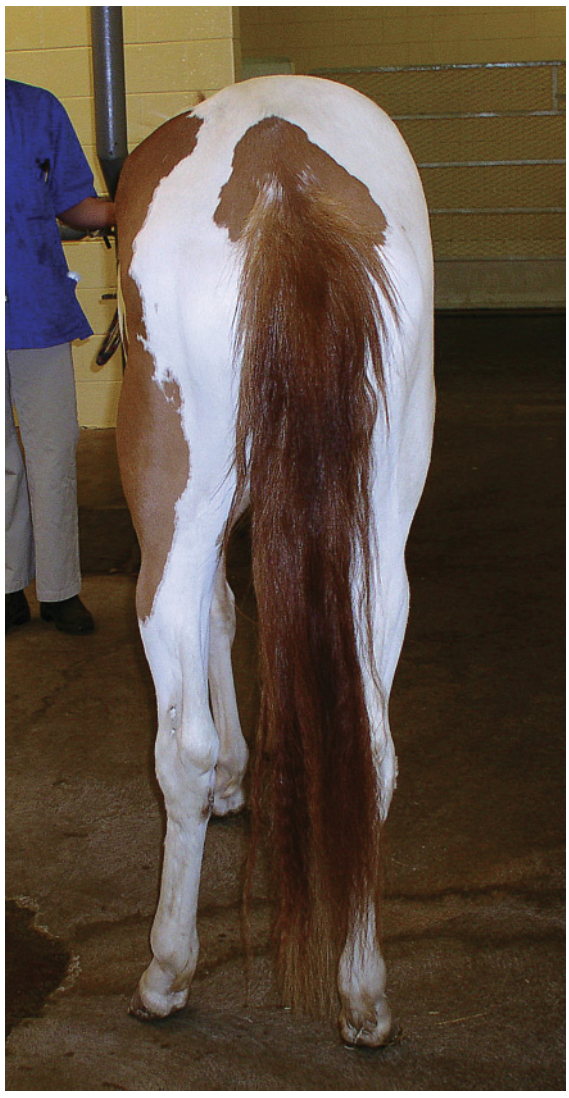


**Figure 8.1.** Young horse with asymmetry of the pelvis, muscle atrophy over the left hip, and a toe-out stance. The horse was lame at the walk and an acetabular fracture was present on radiographs.

### *Treatment*

- The treatment is usually confinement combined with variable periods of NSAIDs. Strict stall rest for at least 30 days is considered important.
- The length of confinement and prognosis depend on the type of fracture.
- Euthanasia is recommended in horses with severely comminuted and displaced fractures.

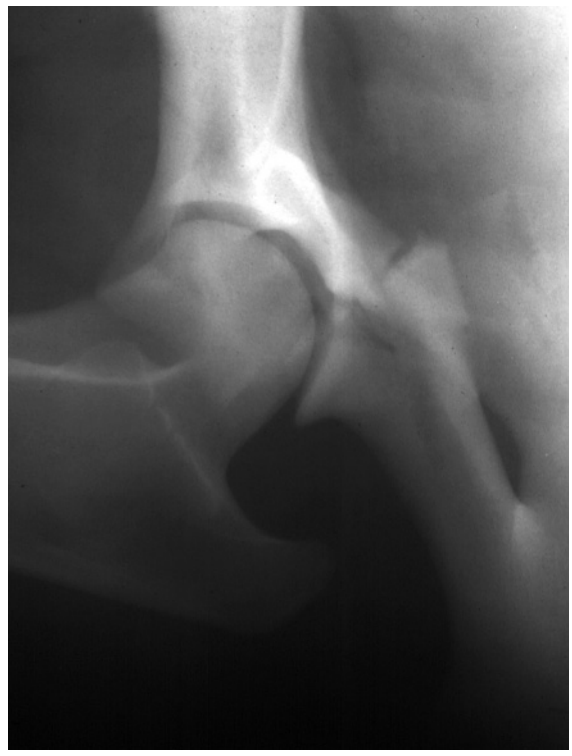


**Figure 8.2.** Ventrodorsal radiograph of the coxofemoral joint, demonstrating a minimally displaced acetabular fracture. Intra-articular fragmentation was not identified and the horse was treated with stall confinement.

### *Prognosis*

- The prognosis varies, depending on the type of fracture.
- Most minimally displaced fractures should heal with a good prognosis.
- Horses with acetabular fractures have a reduced prognosis for future performance.

## ILIAL WING FRACTURES

### *Etiology*

- Ilial wing fractures are incomplete, stress, or "fatigue" fractures associated with training and racing in young racehorses.
- They are usually unilateral but can occur bilaterally.

### *Clinical Signs*

- Young racehorse that presents with an acute onset of lameness.

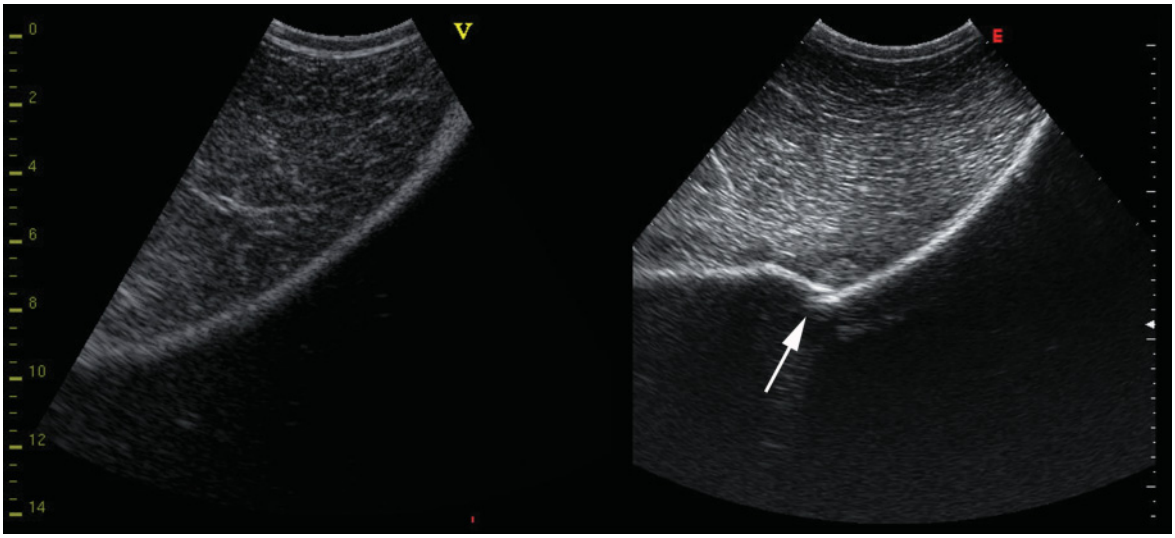


**Figure 8.3.** Ultrasonographic image of mildly displaced ilial wing fracture. At the left is the sonogram of a normal ilial wing; at the right is an ilial wing fracture (arrow). Courtesy of Mary Beth Withcomb.

- The severity of lameness varies from grade 2 to 5/5 and usually improves quickly with confinement.
- The horse may have a tendency to plait with the hindlimbs or cross over the hindlimbs at the trot.
- Gluteal muscle atrophy may occur within two weeks.

### *Diagnosis*

- Nuclear scintigraphy can be used to make the diagnosis.
- Ultrasonography may be used to detect the fracture if it is displaced (Figure 8.3).
- Radiography of pelvis under general anesthesia is contraindicated because of the risk of fracture displacement.

### *Treatment*

- Treatment consists of discontinuing training/racing.
- Usually confinement is combined with NSAIDs, depending on severity of the lameness.

### *Prognosis*

- The prognosis usually is very good for returning to racing.
- Complete, displaced fracture of the ilium is always a concern if the horse returns to training too soon.

## TUBER COXAE FRACTURES

### *Etiology*

- The cause of tuber coxae fractures is single-event trauma such as running into a door, post, wood fence, side of a building, etc.
- It may be associated with a wound.

### *Clinical Signs*

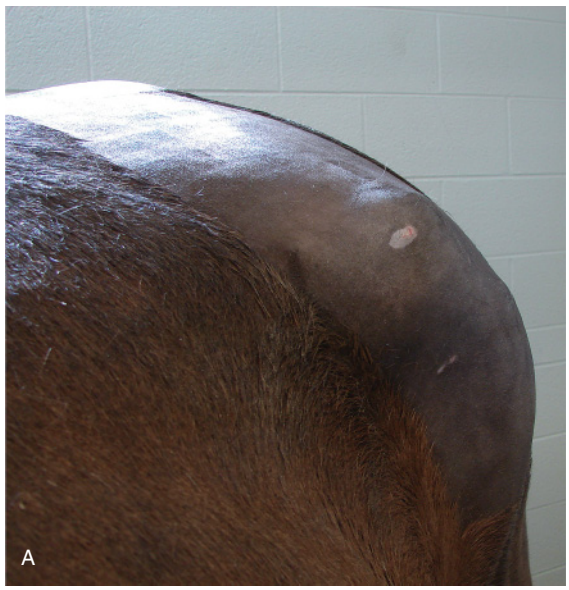
- Moderate to severe lameness that usually decreases to mild lameness in 24 to 48 hours.
- In an abnormally contoured tuber coxae, the fractured portion will move cranioventrally due to traction of the internal abdominal oblique muscle.
- The horse may have an associated wound with a draining tract with or without sequestration in chronic cases (Figure 8.4).

### *Diagnosis*

- The diagnosis is often based on clinical signs alone.
- Ultrasonography may be used in the diagnosis (Figure 8.4).
- A dorsomedial-centrolateral 50° oblique radiograph may be made of the tuber coxae in the standing horse.

### *Treatment*

- The treatment is confinement if the fracture is closed.



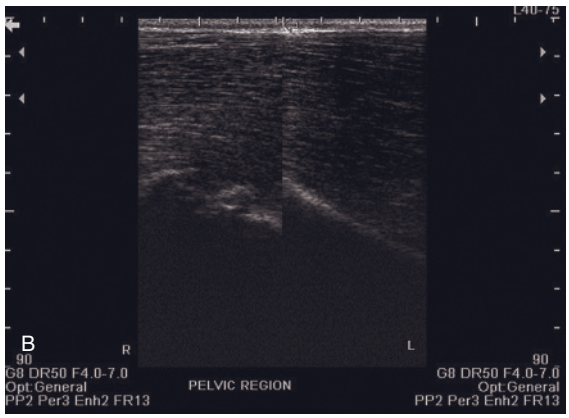




**Figure 8.4.** (A) Cranial view of the pelvis demonstrating swelling and a wound associated with the left tuber coxae. (B) Ultrasound image of the tuber coxae showing a disrupted contour of the ilial wing. Courtesy of Rob van Wessum.

- Appropriate wound management is necessary if the fracture is open.
- There is no need to remove the fracture fragment unless the wound fails to heal and a sequestrum develops.

### *Prognosis*

- The prognosis is usually very good. Up to 93% of the horses returned to athletic performance, according to a recent study.
- Surgical removal may be necessary if a draining tract and sequestrum are present.

## ACETABULAR FRACTURES

### *Etiology*

- The cause is usually single-event trauma, such as a fall.

### *Clinical Signs*

- The horse is often very lame and reluctant to move, but this depends on the duration and degree of displacement.
- Crepitation can often be heard or ausculted when the horse is rocked back and forth.
- Swelling adjacent to the acetabulum may be palpable on rectal examination.

### *Diagnosis*

- Tentative diagnosis can be made based on history and clinical findings.
- Ultrasonography (transcutaneous or transrectal) is often diagnostic if the fracture is complete (Figure 8.5).
- Radiography can be performed under anesthesia but may cause further fracture displacement. (Figure 8.2).

### *Treatment*

- The treatment is similar to other types of pelvic fractures: conservative or euthanasia.

### *Prognosis*

- The prognosis is unfavorable for return to performance if the fracture is displaced due to development of OA of the coxofemoral joint.
- The prognosis is good if the fracture is minimally displaced.

## FRACTURES OF THE SACRUM AND COCCYGEAL VERTEBRAE

### *Etiology*

- The cause is usually trauma such as a fall.
- Often no cause is known.

### *Clinical Signs*

- The clinical signs are variable; one indication is the horse's reluctance to back up.

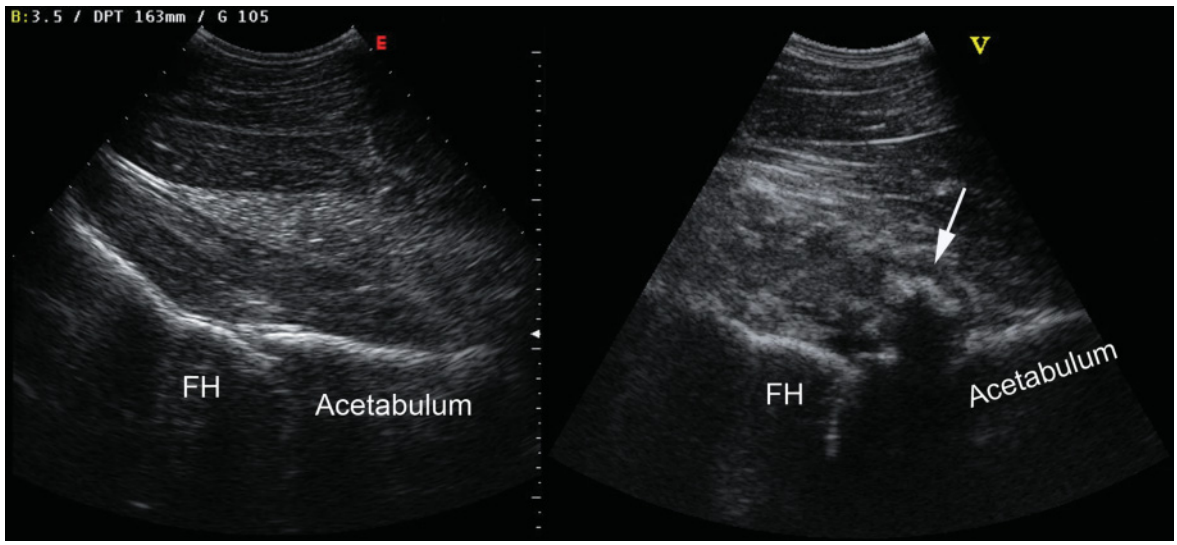



**Figure 8.5.** Ultrasonographic image of an acetabular fracture. At the left is a normal image; a fracture of the acetabulum (arrow) can be seen on the right. FH = femoral head. Courtesy of Mary Beth Whitcomb.

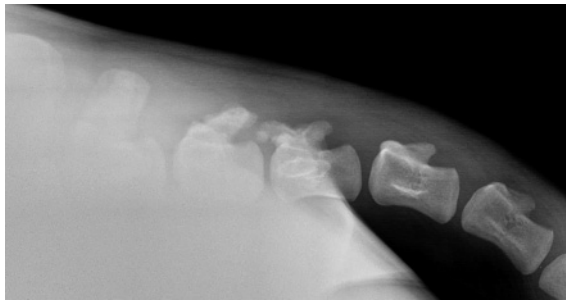


**Figure 8.6.** Radiograph of a horse with bone proliferation between the coccygeal vertebrae 1 and 2 at the dorsal aspect as the result of an avulsion fracture of the intervertebral ligament. Courtesy of Rob van Wessum.

- The normal "snake–like" locomotion pattern to the spine at the walk and canter is altered due to pain.
- Focal swelling and palpable pain are often present, with reduced mobility of the sacrum or tail.

### *Diagnosis*

- Radiography is best used to diagnose coccygeal fractures (Figure 8.6).
- Ultrasound may be able to identify the fracture.
- Scintigraphy may also be helpful locate the site of the fracture.

### *Treatment*

- Treatment consists of a reduced workload or complete rest if a displaced fracture is present.
- Caudal epidural may be administered using corticosteroids if the problem is chronic.
- Topical NSAIDs, extracorporeal shock wave therapy (ESWT), platelet-rich plasma (PRP), etc. may be used for concurrent soft tissue injuries of the sacrum.

### *Prognosis*

- The prognosis is usually very good.

## DISEASES OF THE SACROILIAC REGION

### *Introduction*

- Conditions of the sacroiliac region are recognized more and more as a cause for (low-grade) lameness or lack of performance.
- The sacroiliac (SI) joint itself and/or the soft tissue structures adjacent to the joint can cause SI problems.
- The SI joint is located where the ventral aspect of the ilium comes into close contact with the sacrum.
- The ventral aspect of the SI joint is supported in part by the ventral sacral ligaments (Figure 8.7).

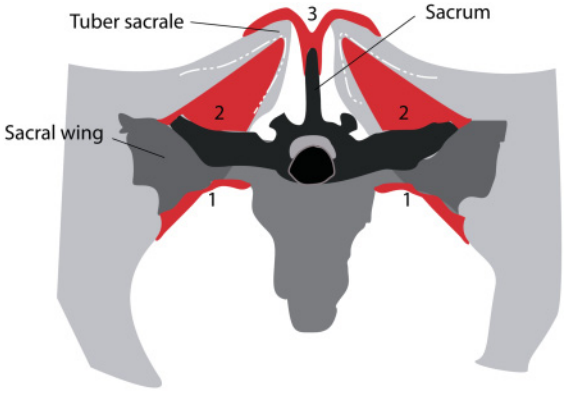



**Figure 8.7.** Schematic drawing of the sacroiliac joint region and the adjacent ligamentous structures (cranial aspect). (1) Ventral sacral ligaments. (2) Interosseus sacroiliac ligaments. (3) Dorsal sacral ligaments. Drawing by Maggie Hofmann.

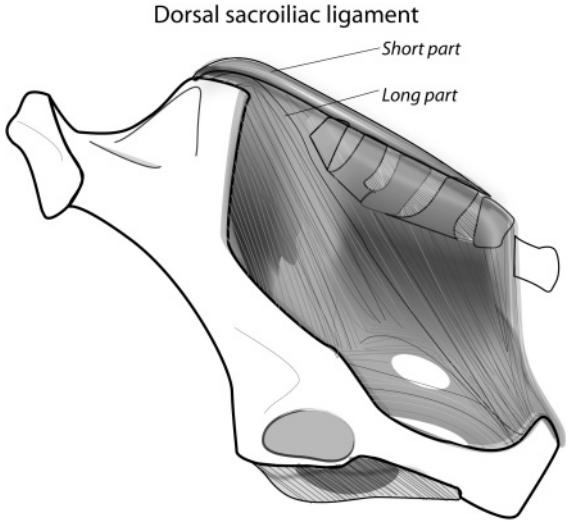



**Figure 8.8.** Schematic drawing of the dorsal sacral ligament (short part and long part). Reprinted with permission of Robinson NE, Sprayberry KA: 2009. Sacroiliac Disease. *In: Current Therapy In Equine Medicine, Sixth Edition.* St. Louis, Elsevier, 484. Drawing by Maggie Hofmann.

- The dorsal sacral ligaments originate from the tuber sacrale and include a long ligamentous branch, called the long part, that adheres to the fibrous structures of the pelvis (the sacrosciatic ligament) and a shorter portion adhering to the sacral bone and the coccygeal vertebrae, called the short part (Figure 8.8). The interosseus sacroiliac ligament also supports the SI joint.

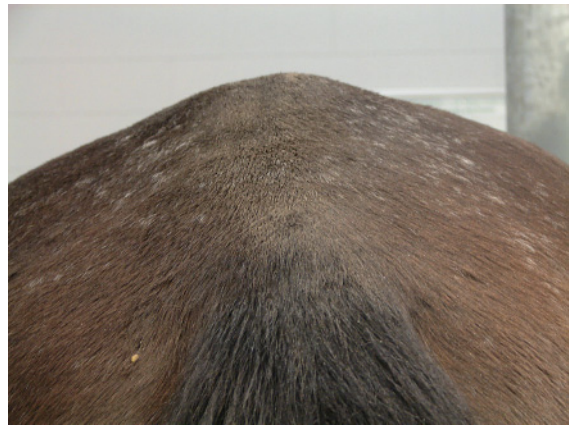


**Figure 8.9.** Caudal view of a horse with asymmetric tuber sacrales. Due to ligament damage, the right tuber sacrale is much higher than the left. Courtesy of Rob van Wessum.

## *Etiology*

- The pelvic musculature is important in providing stability and mobility to the SI region, and weakness in these muscles may contribute to injuries.
- SI conditions may involve damage to the dorsal sacral ligaments, the interosseus and ventral sacral ligaments (i.e., subluxation), or the SI joint itself (OA).
- Injuries can range from single-event trauma such as getting "cast" or repetitive injury to the area associated with performance.

## *Clinical Signs*

- Variable presenting complaints including reduced stride length in one or both hindlimbs, reduced propulsion and engagement, refusal to jump, and unwillingness to go downhill.
- Lameness or gait abnormalities may be most noticeable at the canter—a "bunny hop" in which there is less separation of footfalls of the hindlimbs.
- The horse may change leads or cross canter frequently.
- The horse may appear stiff and rigid with a lack of lateroflexion in the lumbosacral region.
- The performance of racehorses may be reduced.
- Horses display behavioral issues such as kicking, rearing, striking, and bucking, especially when asked to canter.
- Asymmetry of the tuber sacrales can be observed but is not a consistent finding (Figure 8.9).

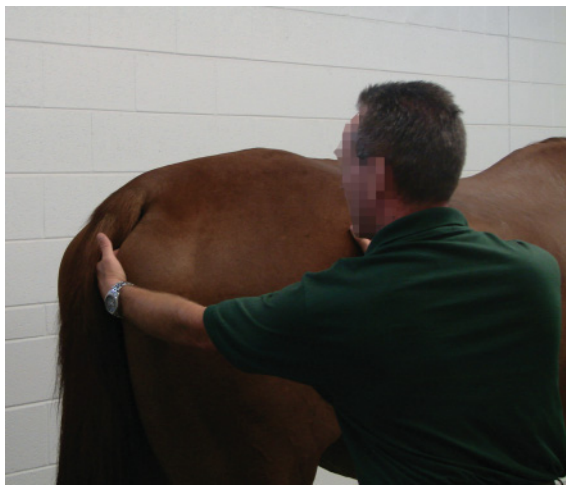


**Figure 8.10.** Pain provocation test for sacroiliac joint pain. The left hand of the examiner is on the right tuber ischium and the right hand is on the tuber coxae, creating a rocking motion in the cranial direction. Courtesy of Rob van Wessum.

- Gluteal muscle atrophy is often present.
- During flexion tests the horse may show reluctance to stand on the affected limb or lean over to the affected side so the stance limb is in midposition, reducing rotational forces to the pelvic structures.
- There may be palpable pain on deep palpation over the tuber sacrale or with pelvic manipulation (Figure 8.10).
- Severe hind limb lameness is associated with severe SI injury such as acute subluxation, dislocation, or rupture of the sacral ligaments.

### *Diagnosis*

- Scintigraphy is the best diagnostic method.
- Percutaneous ultrasound can be used on the dorsal sacral ligaments.
- Trans-rectal examination can be performed to evaluate the bony edges of the SI joint.

### *Treatment*

- Confinement and rest are indicated if an acute injury is suspected.
- A complete and intensive rehabilitation program is important in chronic injuries, with the goal of developing better muscle support of the SI region.
- Injection of the SI joint/area with corticosteroids is most useful if OA or chronic trauma to the joint is suspected.
- PRP or other regenerative therapies are most useful for injuries to the dorsal sacral ligament.

### *Prognosis*

- The prognosis varies and is difficult to predict.
- Most reports suggest a guarded prognosis for return to the previous level of performance.
- The prognosis may be improved with a structured six-month rehabilitation program with frequent re-evaluations.

## OVERRIDING/IMPINGEMENT OF DORSAL SPINOUS PROCESSES

### *Introduction*

- Overriding/impingement of the dorsal spinous processes is known as "kissing spines."
- It is a common diagnosis in horses with back pain.
- The most common location for these lesions is in the thoracic spine (between T5 and T18), but impingement of the lumbar dorsal processes also has been reported.

### *Etiology*

- Repetitive (traumatic) contact between the dorsal processes is the cause.
- Damage to the supraspinous or interspinous ligaments may lead to impingement.
- A primary injury to the ventral and ventrolateral support structures of the annulus fibrosis of the intervertebral disk may cause (asymmetrical) narrowing or collapse of the intervertebral joint.

### *Clinical Signs*

- The horse may have a variety of signs, often related to the discipline in which they perform. Show jumpers and hunters seem to be more affected than others.
- Impingement reduces the ventrodorsal mobility of the spine most often, but when pain is present, the lateral mobility may be limited due to muscle spasm.
- There are often irregularities in the size of the summits of the spinous processes of the affected thoracic or lumbar vertebrae.
- Pain with localized digital pressure of the dorsal spinous processes or the supraspinous ligament is present at the affected site(s).
- When impingement is present in the cranial part of the thoracic spine (T5-T12-T13), the horse may have a painful or violent response when putting the saddle on or when the rider mounts.

### Diagnosis

- The diagnosis is based on physical findings of irregularities and pain along the dorsal spinous processes of the affected vertebrae. It is important to differentiate between the resentment shown by some horses and distress resulting from other back conditions.
- Radiography can identify bony changes such as sclerosis, exostoses, and osteolysis.
- Ultrasonography can evaluate the contact and remodelling between adjacent spinous processes, transverse thickening of the processes, and abnormal alignment. Concomitant lesions in the supraspinous ligament and enthesiopathy on the summits of the spinous processes also can be imaged.
- Scintigraphy can identify evidence of active bone metabolism and remodelling of the spinous processes as well as adjacent structures of the spine that may be involved (intervertebral disk, facet joint, vertebral body).
- Infiltration with a local anesthetic at suspected site(s) can provide information about the likelihood that the observed impingement is the cause of pain.

### Treatment

- Conservative therapies include rest, NSAIDs, local injections of anti-inflammatory agents, acupuncture, and physiotherapy.
- Injections of corticosteroids between affected spinous processes, combined with NSAID therapy, can be very helpful in reducing or removing pain.
- Surgical removal of the affected spinous process(es) can be performed if they are determined to be contributing to the pain.
- Endoscopic resection of the spinous process and the interspinous ligament has been reported.

### Prognosis

- The prognosis is considered guarded in most reports
- When ligamentous structures are involved (supraspinous ligament, intraspinous ligament, ventral longitudinal ligament) the prognosis is less favorable than when just osseous changes are seen on radiographs.
- The prognosis is also less favorable when there are adjacent pathologic conditions in the intervertebral disk or the facet joints.
- The prognosis in horses with severe lesions seems to be better with surgery than without surgery.

## SUPRASPINOUS LIGAMENT INJURIES

### Etiology

- Supraspinous ligament injuries are most commonly found between T15 and L3.
- The specific cause is often unknown, but these injuries are often associated with repetitive-use trauma related to performance.

### Clinical Signs

- The clinical signs are similar to those of impingement of the dorsal spinous processes; when only ligament pathology is present, the signs can be mild and very difficult to detect.
- The signs are often associated with localized thickening of the ligament and palpable pain.
- The appearance of the contour of the spine can resemble kyphosis (roaching of the back) due to swelling at the site.

### Diagnosis

- Ultrasonography is the imaging method of choice. It can document ligament pathology and surrounding swelling and edema. However, a recent study found ultrasonographic changes in the supraspinous ligament in clinically normal horses as well as in horses with signs of back pain.
- Radiography may show irregular bone margins of the summits of the dorsal spinal processes, avulsion fragments, and sclerosis.
- Scintigraphy can provide additional information about the involvement of the spinous processes (Figure 8.11).
- Local infiltration of anesthetic can be used to confirm that the supraspinous ligament is the cause of pain.

### Treatment

- Conservative treatment focuses on rest, oral NSAIDs, and rehabilitation. Controlled stretching of the ligament with a lower position of the head (pasture, hay on the ground, ridden with a lower head and a longer neck) and a gradual increase in the workload are recommended.
- Local injection of anti-inflammatories (corticosteroids) can be used as treatment.
- Treatment also may include ESWT.
- Local injection of PRP, stem cells, or other types of regenerative therapies can be used.

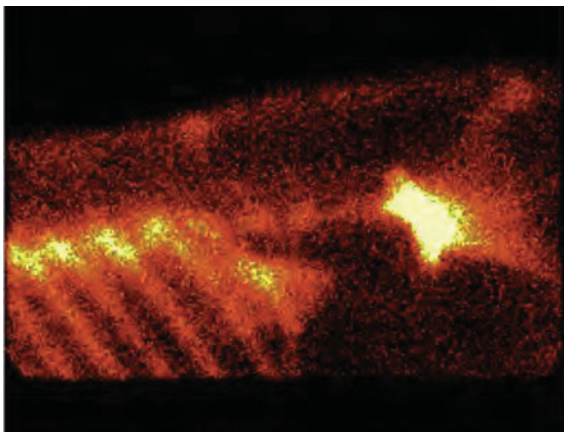


**Figure 8.11.** Scintigraphic image showing mild focal IRU at the summit of T18; it is indicative of desmitis of the supraspinous ligament. Radiography did not show any change, and ultrasonography showed mild desmitis with a roughened bone margin of the summit of the spinous process of T18. Courtesy of Rob van Wessum.

### *Prognosis*

- The prognosis is favorable when only the supraspinous ligament is involved.
- The prognosis is reduced when concurrent structures are affected (dorsal spinous processes, facet joints, or intervertebral disk).

## FRACTURES OF THE SPINOUS PROCESSES

### *Etiology*

- Fractures of the spinous processes occur primarily in the cranial thoracic spine (withers region) when horses flip over, fall backward, or run into objects with their withers.
- Sporadic fractures in the lumbar spine can be caused by excessive trauma such as falling and turning (rotational force on the spine) and at high speed (cross country, jumping, barrel racing, hunting, etc.)

### *Clinical Signs*

- The clinical signs include swelling and palpable pain in the affected region with deformation of the normal contour of the withers.
- An indentation may be observed in the withers and the withers may appear wider than normal.
- Abnormal alignment of the spinous processes may be the only finding in chronic cases.

### *Diagnosis*

- Radiography of the withers usually reveals dislocated and fractures of the thoracic spines. The spinous processes of the withers region (T3-T12) can vary in shape and have accessory centers of ossification that can be misinterpreted as a fracture.
- Radiography of the lumbar spine can be difficult to perform depending on the size of the horse.
- Ultrasonography is most helpful to identify fractures of the spinous processes in the lumbar spine.
- Scintigraphy will often show increased radiopharmaceutical uptake at the affected site but cannot be used to document a fracture.

### *Treatment*

- Conservative treatment is used to manage the pain and swelling associated with the fracture.
- Surgery is not required in most cases unless the fractured spinous process of the withers becomes infected.

### *Prognosis*

- The prognosis is usually very good for spinous process fractures of the withers, but an abnormal contour to the withers often remains.
- It may be difficult to make the saddle fit properly due to the changed shape of the withers.

## VERTEBRAL FRACTURES

### *Etiology*

- Vertebral fractures usually are traumatic in origin.
- Pathologic fractures can occur secondary to neoplasia or osteomyelitis.
- Stress fractures of the vertebrae can occur in racehorses, most often at the thoracolumbar junction and in the lumbar vertebrae.

### *Clinical Signs*

- Horses with acute fractures often have severe pain. The horse may be reluctant to move the affected part of the spine and often demonstrates increased muscle tension in the region of the fracture.
- Neurological signs may be present secondary to soft tissue swelling, hemorrhage, or fragments compressing the spinal cord or nerve roots.

- Most stress fractures cause lack of performance without well-defined signs.

### *Diagnosis*

- Scintigraphy is the imaging modality of choice because radiography of the spine in adult horses is difficult.
- Scintigraphy is nearly the only way to determine an active stress fracture of a vertebrae.

### *Treatment*

- Conservative treatment is indicated in horses with vertebral fractures without neurological signs.
- When neurological symptoms are present, treatment should be more focused on the reduction of swelling around the spinal structures; this includes systemic corticosteroids or NSAIDs, DMSO, and strict confinement.

### *Prognosis*

- The prognosis is very good for stress fractures and non-displaced vertebral fractures without neurological signs.
- The prognosis is much reduced if neurological signs are present. In general, the longer the neurological signs persist, the worse the prognosis.

## DISCOSPONDYLITIS

### *Etiology*

- Discospondylitis is an inflammatory condition involving the vertebral bodies adjacent to the symphysis between two vertebrae, and includes the intervertebral disc.
- It is usually a septic process from hematogenous spread. Bacteria isolated from adult horses with discospondylitis include *Brucella abortus*, alpha-haemolytic *Streptococcus*, coagulase negative *Staphylococcus,* and *Staphylococcus aureus*. *Rhodococcus equi* has been implicated in a foal.
- Traumatic discospondylitis has been documented. Intervertebral disc lesions including fissuration, calcification, and herniation have been described ultrasonographically and at post-mortem examination at the lumbosacral junction and the cervical spine.

### *Clinical Signs*

- Clinical signs include weight loss, back or neck pain, fever, stiffness, and ataxia
- Atrophy of the epaxial muscles in the thoracolumbar region may be evident in some horses suffering from chronic discospondylitis in this region.
- Some horses are so painful that they are reluctant to eat off the ground.
- Neurological signs may occur when there is compression of the spinal cord or nerve roots.

### *Diagnosis*

- Leucocytosis and increased fibrinogen on a complete blood count support an infectious process.
- Radiography may reveal lysis and/or proliferation of the vertebral bodies adjacent to the affected disc. The intervertebral disc space may narrow and collapse, and a smooth bony bridge may unite the affected vertebrae.
- Scintigraphy, computed tomography, and ultrasonography have been used to facilitate the diagnosis of equine discospondylitis and vertebral osteomyelitis. Scintigraphy may be useful to determine if multiple sites of bone involvement are present.

### *Treatment*

- Long-term (four to six months) antimicrobial therapy based on culture and sensitivity is used to treat septic discospondylitis. Broad-spectrum antimicrobials should be used if no causative organism is identified.
- Surgical curettage of the lesion is an option when access to the lesion is possible.

### *Prognosis*

- The prognosis is guarded in cases of septic discospondylitis.
- Favorable outcomes have been associated with early detection, nonseptic lesions, the absence of spinal cord compression, administration of long-term antimicrobial therapy, and surgical curettage.

## SPONDYLOSIS

### *Etiology*

- Spondylosis (deformans) is a degenerative condition affecting the vertebral body that results in osteophyte/enthesiophyte formation on the ventrolateral aspects of the vertebral segment between T10 and T14 (Figure 8.12).

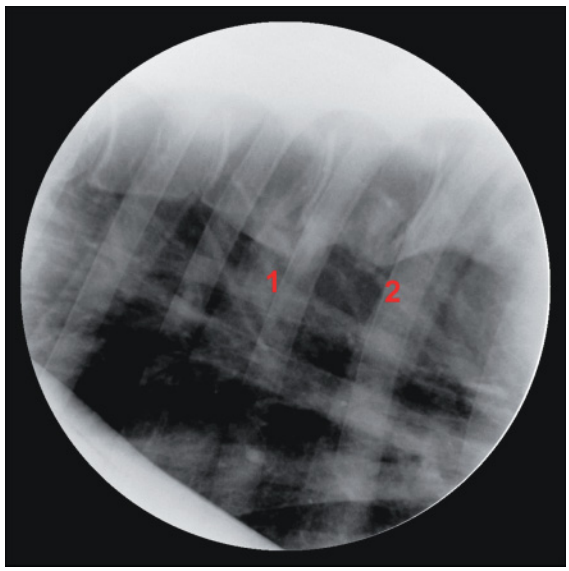



**Figure 8.12.** Radiographic image of the thoracic spine of a horse with severe spondylosis. There is proliferation of bone at the ventral aspect of the vertebral bodies. At (1) there is no complete contact between the proliferations. At (2) there seems to be complete bridging of the intervertebral disk space. Courtesy of Rob van Wessum.

- Osteophyte formation involves mechanical stress at the attachment of the most peripheral fibers of the intervertebral disc and the ventral longitudinal ligament (enthesiophytes).
- Post-mortem results of acute cases have demonstrated damage to the intervertebral disc and erosion of cartilage and hemarthrosis of the facet joints.
- Reduced mobility of the spine at the site of the osteophytes with complete ankylosis is seen as the end stage of the disease.
- The disease is more common in event horses, show jumpers, hunters, and working draft horses.

### *Clinical Signs*

- In the acute stage, severe back pain with generalized stiffness and reluctance to work is often present. These cases seem to relate to the lay term of "cold-back," in which saddling, mounting, or starting to ride initiates a violent reaction of the horse (bucking, running away, and laying down).
- The chronic stage is less painful but limits the motion of the spine, giving the horse a more stiff appearance. The horse may have the appearance of a "hollow back" or (acquired) lordosis. This may contribute to chronic back problems.

### *Diagnosis*

- Radiography and scintigraphy are the main diagnostic tools to confirm spondylosis (Figure 8.12).
- Scintigraphy can be used to demonstrate the amount of bone activity at the site(s) as an indicator for acuteness and bone activity.

### *Treatment*

- Treatment in the acute stage includes rest and NSAIDs.
- Horses with chronic disease are often treated with NSAIDs and other symptomatic treatments for chronic back pain. See Chapter 9.
- No surgical techniques have been described.

### *Prognosis*

- In most cases the prognosis is guarded to return to an athletic career.

## FACET JOINT OA AND VERTEBRAL FACET JOINT SYNDROME

### *Etiology*

- The development of OA in the thoracolumbar spine is often combined with a complex of processes in and around the vertebral facet joints known as facet joint syndrome.
- OA of the facet joints in the cervical spine is usually more isolated to the joint itself, but compression of the cervical nerve roots is more common than in the thoracolumbar region.
- The initiating incident in facet joint syndrome is damage to the facet joint, usually from trauma such as slipping, falling, flipping over backward, getting cast, etc.
- Secondary muscle spasm often occurs in an attempt to stabilize the injured vertebral facet joints. This muscle contraction can be short, lasting from hours to days, or it can last weeks or even months.
- Due to the muscle spasm, no normal sequence of contraction-relaxation occurs in the muscle, and the normal supportive function is less effective.
- Repetitive injury to the facet joint can occur.

### *Clinical Signs*

- In acute injuries, the horse may be reluctant to move and may stand with the hind feet parked out and the back lowered. Spasm and contraction of the muscles of the back and hind end is common.
- With severe pain in the thoracic or lumbar spine, signs of restlessness, pawing, looking at its back, etc. may be present.
- In chronic injuries, the locomotion of the horse can be altered and a stiff back can be noticed. At the trot, the propulsion of the hindlimbs can be reduced unilaterally or bilaterally.
- When the thoracic facet joints are involved, signs can include stiffness, reluctance to go downhill or jump, and refusal or difficulty in doing the extended trot. Lateral bending of the horse may be reduced and pain may be initiated by tightening the girth or putting the saddle on because these actions can load the thoracic facet joints.
- When lumbar facet joints are involved, the most affected gait is the canter, because at the canter the dorsoventral flexion of the lumbar spine is most prominent. In racing, dorsoventral flexion of the lumbar spine is a prominent contribution to the propulsion phase of the hindlimbs, so loss of performance may occur.

### *Diagnosis*

- Scintigraphy is the best modality for imaging the vertebral column. A recent study showed 61.5% of horses with the complaint of back pain had increased uptake of radiopharmaceutical in one or more facet joints.
- There is a high predictive value for scintigraphy in the detection of radiographic lesions and a high sensitivity for scintigraphy in the detection of back pain.
- Ultrasonography can give information about ligamentous structures (supraspinous, interspinous, and dorsal sacral ligaments) and muscle conditions. Paramedian longitudinal views of the facet joints can be used to determine possible effusion, bony proliferations at the joint margins, fractures and avulsion fragments, and ankylosis of the facet joints (Figure 8.13).
- With radiography, the thoracic vertebrae can be viewed through the lungs, facilitating radiographic imaging in the cranial part of the thorax (T1 through T15-T16). Oblique lateral views may be able to isolate the unilateral facet joints from T5-T7 to T18. Radiographic evidence of disease includes sclerosis of bone around the facet joint, narrowing of the joint space, irregular shape of the joint space and spur formation at the joint space, or complete ankylosis (Figure 8.14).
- Lumbar vertebrae are difficult to visualize with radiography. Oblique lateral views can sometimes facilitate imaging of the unilateral facet joints of the lumbar spine.
- The final diagnosis is often a summation of two or more imaging techniques.
- Local anesthesia as a tool for confirming facet joint pain is not very reliable.

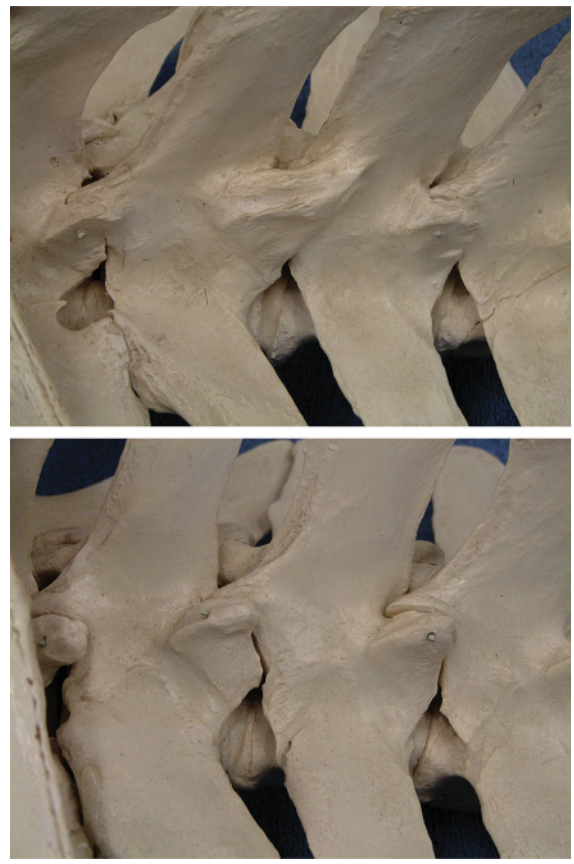


**Figure 8.13.** Anatomical specimen of the lumbar spine with the facet joints visible. In the lower image, the facet joints have smooth edges and appear to be normal. In the upper image the facet joints have more irregular edges when compared to the facet joints in the lower image. Complete ankylosis is present in one of the facet joints. Courtesy of Rob van Wessum.

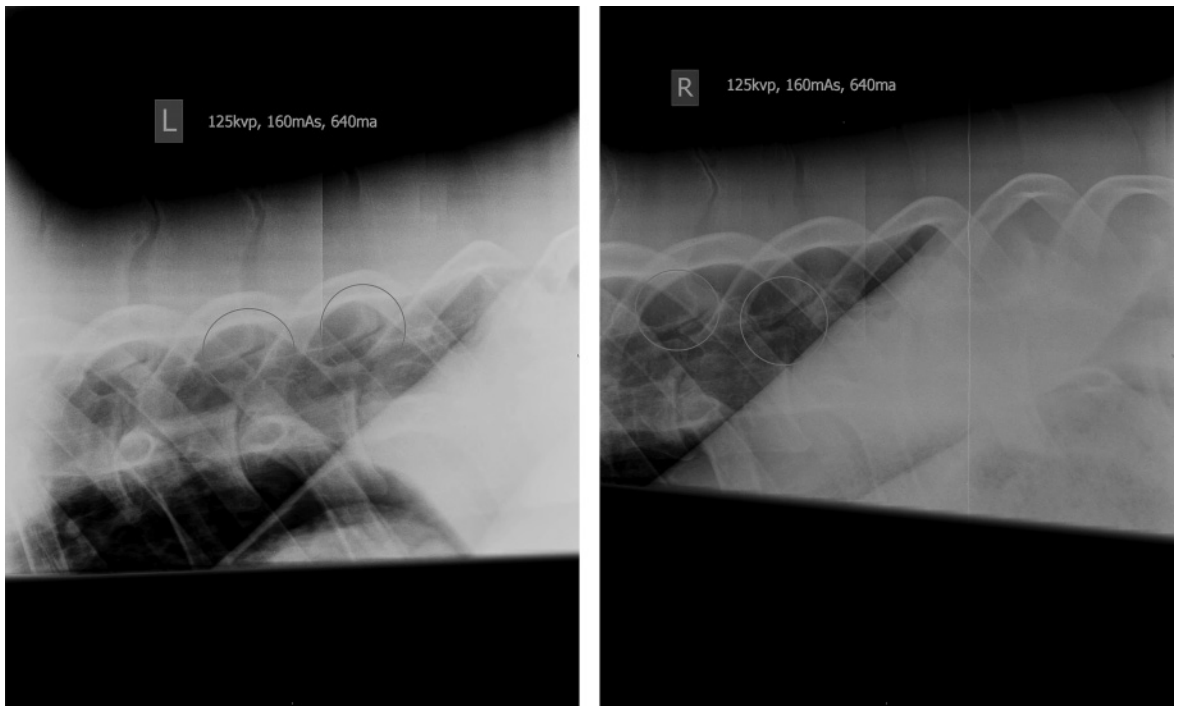



**Figure 8.14.** Radiographic image of the thoracic spine (oblique lateral view) to expose the left facet joints (circles) at the left and the right facet joints at the right. Note the more irregular joint space and some sclerosis in the right facet joints. This is indicative for OA of the right thoracic facet joints. Courtesy of Rob van Wessum.

### *Treatment*

- The initial goal of treatment is to break the inflammatory process triggering the nerves, which leads to muscle spasm, immobility of the spine, and repeated injury. Complex treatment plans that deal with several aspects of the vicious cycle are recommended.
- Treatments include local and systemic NSAIDs, muscle relaxants (methocarbamol), bisphosphonates (tiludronate), and ultrasound–guided injections of a corticosteroid into or close to the facet joint.
- A rehabilitation program is vital and should be aimed at gaining more mobility in the affected part of the spine as well as the development of better muscle support for that region.

### *Prognosis*

- The prognosis varies, depending on the location of the facet joint pathology and the use of the horse.
- The prognosis appears better in racehorses with lumbar disease than thoracic disease.
- Three-day event horses and jumpers appear to have a more guarded prognosis than dressage horses.

## NUCHAL LIGAMENT DESMOPATHY/ NUCHAL BURSITIS

### *Etiology*

- The nuchal ligament connects the dorsal processes of the cervical vertebrae with the withers and has an important function in supporting the entire neck.
- Desmopathy of the nuchal ligament is usually associated with trauma such as falling down, pulling backward when tied, head caught in fences, trailering accidents, etc.
- Training methods such as tying the horse's head to the side or between the front limbs or the "hyperflexion or Rollkur" as practiced by some dressage trainers can predispose to nuchal ligament pathology.

- The nuchal bursa, present at the proximal aspect at the 1st cervical vertebra (C1) in most horses and less frequently at the 2nd cervical vertebra (C2), can be involved in pathology of the nuchal ligament. Infection of the nuchal bursa has been reported but occurs infrequently.

### *Clinical Signs*

- Signs can be diverse and very often more visible when the horse is worked. The horse may appear stiff with very little neck movement at the walk, with no flexion of the neck depending on the diameter of the circle when worked.
- When ridden or driven, the contact with the bit through the reins or lines can feel different to the rider/driver, with one side feeling more rigid or as if the horse is pulling on one side. Dental issues can have similar signs and particular head positions can cause the horse pain, making it behave defensively or reluctant to perform certain exercises.
- General lack of performance can be one of the more indistinctive signs.

### *Diagnosis*

- With radiography, occasionally enthesiophytes can be seen at the attachment of the ligament to the bone at the poll, which may suggest desmopathy or nuchal bursitis.
- Ultrasonography may be able to detect damage to the ligament and/or the nuchal bursa.
- Endoscopy of the nuchal bursa has been reported as both a diagnostic and treatment tool.

### *Treatment*

- Therapy is similar to that of any desmopathy and may include rest, NSAIDs, extracorporeal shock wave treatment, and injection of biological products such as PRP and stem cells.
- Ultrasound-guided injection of corticosteroids into the nuchal bursa can be performed if the bursa is thought to be involved.
- Endoscopy of the nuchal bursa has been reported as an effective treatment for nuchal bursitis.

### *Prognosis*

- The prognosis is usually favorable if the horse is given time for complete recovery and rehabilitation of the nuchal ligament injury.
- Chronic injuries may lead to pain or defensive behavior (rearing, shaking the head, etc.) when the head is in a very high and upright position.

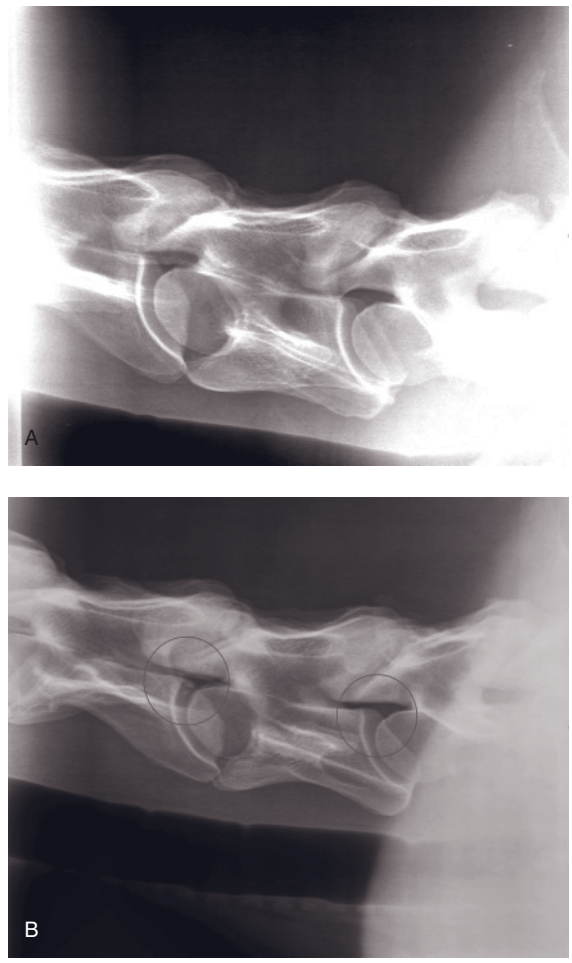



**Figure 8.15.** (A) Radiographic image of the cervical spine with C5, C6, and C7 in view. There is mild enlargement of the facet joints, indicative of OA, between C5 and C6 and between C6 and C7 with irregular joint spaces. This can be a common finding in horses without clinical signs. (B) Radiographic image of the cervical spine. Note the change at the caudal aspect of the endplate of C5 and the small fragment (circle 1) when compared to the clean appearance at C6 (circle 2). This is most likely caused by trauma to the intervertebral ligament at the ventral base of the spinal canal. Courtesy of Rob van Wessum.

- Horses with nuchal bursitis appear to have a good prognosis for complete recovery.

## CERVICAL FACET JOINT OA

### *Etiology*

- Cervical facet joint OA can be found radiographically in many older horses, even without clinical signs (Figure 8.15A).

- It can develop secondary to osteochondrosis of the articular surfaces of the cervical joints similar to many other joints in the horse. Severe developmental lesions can lead to cervical instability and malformation, causing neurological signs (Wobbler syndrome).
- Trauma to the neck can damage the cervical joints, leading to OA (Figure 8.15B). Major trauma to the neck region is often seen with trailering accidents, falling or hanging in cross ties, running into objects with the neck, falling during exercise, flipping over backward, etc.

### *Clinical Signs*

- Diverse clinical signs are present, ranging from performance-limiting lameness to severe neurological signs.
- Reduced neck mobility can limit athletic performance. The dressage horse may be reluctant to bend the neck in a lateral direction, bring the head vertical in the collection position, or stretch down. A jumping horse may lack balance during the jump due to neck stiffness, while a barrel racing horse may have difficulty turning around the barrels.
- Contraction of the muscles in the neck secondary to facet joint pain may alter the forelimb gait (shorter anterior stride, reduced protraction of the scapula) and mimic a forelimb lameness.
- Severe cervical malformation and cervical joint OA can cause compression of the spinal column and neurological deficits.
- Evidence of cervical pain and reduced range of motion may or may not be present.
- Asymmetrical enlargement of the left and right cervical vertebrae my be found on palpation.

### *Diagnosis*

- Cervical facet joint OA should be suspected when the range of motion in the neck is limited and palpable abnormalities are present.
- Radiography is the imaging technique of choice. Slight oblique views from the horizontal plane can be used to project the left or right facet joint without superimposing on top of the ipsilateral joint.
- Ultrasonography can be used to obtain information about joint space size and shape and bone proliferation at the edges of the joint.
- Ultrasound-guided intra-articular block of the facet joint(s) can be performed to verify it as the cause of the clinical signs
- Nuclear scintigraphy is very useful be document active inflammation from older inactive lesions seen on radiographs. It can be important in decision-making about treatment, i.e. whether to inject a facet joint with corticosteroids.
- Myelography documents spinal cord compression in horses with neurological signs.
- Electromyography may identify changes in muscle signals in the segmental muscles in the neck to demonstrate dennervation of these muscles as a sign of nerve root compression.

### *Treatment*

- Acute injuries are treated with topical and oral NSAIDs, muscle relaxants, and reduced activity.
- Chronic OA is often treated with ultrasound-guided injection of the facet joints with corticosteroids.
- Surgical fusion of the vertebrae can be performed in young horses with cervical vertebral malformation.

### *Prognosis*

- The prognosis is variable, depending on the severity of the clinical signs.
- It is good for horses mild OA and minor clinical signs.
- The prognosis is poor for performance in horses with severe OA or cervical malformation together with neurological abnormalities.

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**Revised from "Lameness Associated with the Axial Skeleton" in *Adams and Stashak's Lameness in Horses, Sixth Edition,* by Rob van Wessum.**

9

CHAPTER

# Therapeutic Options

## SYSTEMIC/PARENTERAL

### *Introduction*

- Systemic/parenteral medications used to treat musculoskeletal diseases in horses include IV or oral nonsteroidal anti-inflammatory drugs (NSAIDs), oral nutraceuticals, intramuscular (IM) polysulfated glycosaminoglycans (PSGAGs), IV or oral hyaluronan (HA), IV bisphosphanates, and several other miscellaneous drugs.
- Excluding NSAIDs, the debate regarding many of these drugs in the horse revolves around whether they reach high enough concentrations at the intended area.
- Many (or most) of the drugs or nutraceuticals have been intended for systemic administration in humans, and levels high enough to be efficacious in the horse are often in question.

### *Parenteral NSAIDs*

- The NSAIDs used most commonly IV are phenylbutazone and flunixin meglumine.
- Less commonly used IV NSAIDs are ketoprofen, carprofen, and firocoxib.
- Most of these NSAIDs can be delivered orally, but the IV route of delivery may be more effective and faster at yielding the desired pharmacological effect in the acute stages of disease.
- When NSAIDs are used consistently in horses, oral formulations are recommended. See "Oral/Nutritional" later in this chapter.

### *Polysulfated Glycosaminoglycans (PSGAGs)*

- Adequan® (Luitpold Pharmaceuticals, Inc.) is produced from an animal source, bovine trachea and lung, and is in a class of drugs that exhibits chondroprotective properties in cartilage.
- PSGAG is a mixture of low-molecular-weight glycosaminoglycans (GAG).
- PSGAG administration has been associated with reductions in the severity of clinical signs in both human and equine patients with OA.
- Modes of administration include IM and intra-articular (IA) routes. Elevated risks have been identified with IA administration; therefore, many practitioners still prefer to administer this drug IM.
- It has been theorized that PSGAGs inhibit a plethora of degradative enzymes that contribute to the OA process, including lysosomal elastase, cathepsins, lysosomal hydrolases, serine proteinases, neutral metalloproteinases

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

(MMP), plasminogen activators, and inducible nitric oxide.
- In a survey of more than 400 equine practitioners, IM Adequan® was one of the most commonly administered medications (that was not in the steroid category) in horses.
- Currently, the dosing recommendations are 500 mg IM every three to five days for five to seven treatments.
- Clinical indications for the systemic administration of Adequan® include the treatment of synovitis, cartilage degradation, OA, and capsulitis. Theoretically, this drug should prevent or at least minimize progression of OA if high enough levels in the joint are achieved.
- It may also be used to prevent joint inflammation/disease in sport horses in heavy work.

### *Hyaluronan*

- HA is a large, unbranched, nonsulphated glycosaminoglycan composed of repeating units of D-glucouronic acid and N-acetyl glucosamine.
- Intrasynovial injections are most likely the most efficacious route of administration; however, the desire to treat multiple joints simultaneously has resulted in its use both IV and orally.
- HA actions in the synovial joint include increasing viscosity of synovial fluid, lubricating unloaded joints, restoring the rheologic properties of synovial fluid, and most importantly, inhibiting inflammation.
- The beneficial effects of IV HA are most likely due to the effects in the synovial membrane because the plasma half-life is less than an hour and is no longer detectable after three hours. The effects noted through IV administration may be due to the fact that the synovial membrane is highly vascularized and this may allow greater exposure to synoviocytes than IA administration.
- Currently, Legend® (Bayer Animal Health) is the only licensed HA product for IV administration.
- Another product, Polyglycan® (Arthrodynamic) is licensed as a medical device and is used by practitioners both IV and intrasynovially.
- Clinical indications for IV HA are similar to those for IM PSGAGs in that IV HA reduces inflammation in one or multiple joints or prevents an inflammatory process in sport horses that are in heavy use.
- Although IA administration is the most effective way to deliver this drug to synovial cavities, often IA and IV administration are used concurrently.

### *Isoxsuprine*

- Isoxsuprine has been suggested for use in horses with navicular syndrome, sesamoiditis and laminitis.
- The drug is a β-adrenoreceptor antagonist with β-adrenoreceptor agonistic properties, which causes vasodilation. The drug is capable of decreasing blood viscosity and platelet aggregation.
- Efficacy in several clinical trials has been variable with little control of shoeing and other concurrent treatments.
- Despite reports of limited bioavailability, advocates of isoxsuprine recommend a trial dose of 0.6 mg/kg twice a day orally for 30 days.
- In horses that do not respond to the lower dose, increasing the dose to 0.9 mg/kg or 1.2 mg/kg twice daily may improve efficacy.
- Poor responses are most likely to occur in horses with major radiological abnormalities.

### *Bisphosphonates*

- Bisphosphonates have been used for decades to inhibit loss of bone mass in human patients with osteoporosis associated with age or steroid administration, and rheumatoid arthritis.
- Bisphosphonates inhibit osteoclast-mediated bone resorption, a property which clinicians feel has a role in treating conditions in which reduction in bone turnover may have beneficial effects such as OA and navicular bone edema/sclerosis.
- Currently, four studies have reported beneficial effects of reduced lameness, back soreness, and loss of bone with IV administration of tiludronate (Tildren®).
- Tiludronate is not licensed for use in horses in the United States, but veterinarians can obtain the drug with a letter of approval from the U.S. Food and Drug Administration.
- Current pharmacokinetic studies suggest the drug be administered slowly at 1 mg/kg IV rather than at 0.1 mg/kg IV once daily for 10 days. Flunixin meglumine is recommended concurrently because horses may suffer from colic secondary to administration.
- Many clinicians use the 0.1 mg/kg dose locally in the distal limb as an IV regional perfusion to minimize the systemic side effects.

### *Tetracyclines*

- Tetracycline antibiotics have historically been used to treat rheumatoid arthritis and OA in people.
- Clinical effects reported include improvement in joint pain and reductions in cartilage erosion.
- Horses in which tetracycline have been used empirically to treat Lyme disease have realized an improvement in lameness despite the fact that titers were negative.
- The mechanism of action, at least in part, is due to the ability of tetracyclines to reduce MMP activity.
- The suggested dose is 5 mg/kg once or twice daily orally.

### *Robaxin (Methocarbamol)*

- Muscle relaxants are occasionally used in horses to treat various pathologies of muscle disease.
- Methocarbamol appears to produce variable results but may be beneficial in some horses that are prone to rhabdomyolysis or sore backs.
- The dose administered ranges from 6 to 22 mg/kg but usually is 10 mg/kg orally twice daily for five to 10 days.

## TOPICAL/LOCAL

### *Introduction*

- Local and topical therapies are commonly used to treat equine musculoskeletal diseases both acutely, when inflammation is most pronounced, and chronically, when ongoing inflammation results in soreness and/or lameness.
- Local modes of therapy to control associated edema and release of inflammatory mediators are often used concurrently with systemic NSAIDs.
- Effective use of topical therapy is thought to reduce the need for systemic NSAID therapy.

### *Topical NSAIDs*

- A topical formulation of diclofenac liposomal cream (Surpass®, Boehinger Ingelheim Vetmedica, Inc.) is now approved for treatment of horses with OA.
- Surpass® comes as a cream, which is applied locally to areas of inflammation.
- Studies have reported that the drug readily penetrates skin and significantly attenuates carrageenan-induced local production of PGE.
- Application of 7.3 grams of Surpass® twice daily (label dose) to the skin showed significant improvement in lameness in a model of carpal OA.
- Surpass® can be used under a bandage and there are currently no reported adverse effects.
- Although the dose is 7.3 grams BID, some clinicians use this drug once daily or every other day for inflammation that is less acute.
- It can be used for OA, periostitis, tendinitis, and various musculoskeletal inflammatory conditions.
- Concurrent use of this product with systemic NSAIDs is common and seems to be very effective for a variety of inflammatory conditions.

### *Topical First Aid (Cold Therapy and Bandaging)*

- Cold compression in the early stages of inflammation retards the inflammatory processes of exudation and diapedesis as well as reduces edema.
- The application of cold therapy as a primary treatment for most acute joint or tendon injuries is commonly practiced and is extremely beneficial.
- Following an initial "cooling out" phase, usually 48 to 72 hours, warm hydrotherapy is often used to relieve pain and tension in tissues as well as stimulate the vasodilatory effect to aid with fluid resorption and the stimulation of phagocytic cells.
- Cold therapy can be applied using ice boots, cold water hosing/hydrotherapy, or medical devices that provide cold therapy and compression (Game Ready®).
- Bandaging and pressure wraps are also commonly used to decrease edema formation and generalized swelling.
- Bandage support also may assist healing as stimulation of mechanoreceptors occurs, and this may decrease pain sensation.
- The duration to use pressure wraps is unknown, but the general practice is to apply them as long as ongoing inflammation occurs.

### *Dimethyl Sulfoxide (DMSO)*

- The chemical solvent DMSO has been used alone or mixed with corticosteroids to treat soft tissue swelling and inflammation resulting

from acute trauma. Its main benefit is considered to be reduction of edema.
- DMSO has been shown to possess superoxide dismutase activity and inactivate superoxide radicals, and enhance penetration of percutaneous steroids when it is mixed with DMSO.
- The combination of furacin, DMSO, and glycerin is often used as a sweat to reduce inflammation and edema.
- If applied under a bandage it should be used with caution due to its ability to cause skin irritation.

### *Extracorporeal Shock Wave Therapy (ESWT)*

- Extracorporeal shock waves are acoustic waves generated outside the body, characterized by transient high peak pressures, followed by negative pressure and then return to zero pressure.
- Pressures reached by current equipment range from 10 to 100 MPa with a rapid rise time of 30 to 120 ns and a short pulse duration (5 μs). Other variables include energy level, pulse frequency, and depth of penetration.
- It has been used to treat select musculoskeletal conditions in horses such as suspensory ligament desmitis, OA of the low-motion joints of the tarsus, and dorsal metacarpal disease.
- The exact mechanism of action that extracorporeal shock wave therapy has on specific tissues is not known. Suggested effects on bone include microfracture of the cortical bone, medullary hemorrhage, subperiosteal hemorrhage, and stimulation of osteogenesis.
- Analgesic properties attributed to ESWT are thought to be due to decreases in nerve conduction properties.
- Use of ESWT at race tracks has stimulated debate due to its abilities to reduce or eliminate pain from an injury that may become catastrophic if the horse continues to race.
- ESWT also has been shown to increase microcracks on the dorsal surface of the MC/MT.
- Musculoskeletal conditions in horses that have been treated with ESWT include the bucked-shin complex, tibial stress fractures, proximal sesamoid bone fractures, incomplete proximal phalangeal fractures, subchondral bone pain, insertional desmopathies (most notably proximal ligament suspensory desmitis), impinging dorsal spinous processes, OA of the distal hock joints, navicular disease, and SDF tendinitis.
- The most common clinical entity that is treated with shock wave therapy is proximal suspensory ligament desmitis. Studies have reported a 70% to 80% success rate of return to work six months following focused or radial shock wave therapy.

### *Counterirritation*

- Topical blistering has long been part of veterinary medicine but is used less frequently today.
- Topical blisters historically were made of iodine or mercuric iodide and were mainly used on splints, sore shins, or curbs. The technique involved application to the skin and light exercise.

## INTRASYNOVIAL

### *Introduction*

- Intrasynovial therapies are used to diminish the inflammatory response in synovium, cartilage, tendon (or tendon sheath), or meniscus.
- An effective therapy should halt progression of degradation of these structures and restore the normal intrasynovial environment.
- Intrasynovial therapies, specifically corticosteroids, are used frequently to minimize or control pain associated with synovitis and OA.
- New therapies such as disease modifying agents of osteoarthritis drugs (DMOAD) are being used to prevent, retard, or reverse morphologic cartilaginous lesions of OA. While claims of these properties exist, evidence-based clinical studies are still in their infancy as to the true nature of their abilities to reverse OA.

### *Corticosteroids*

- Corticosteroids (CS) are the most potent anti-inflammatory drugs available that decrease the catabolic effects of joint disease.
- Their use has been highly controversial due to the negative effects associated with overuse and high dosages used in the past.
- Clinical studies suggest judicious use of corticosteroids may be beneficial and can result in long-lasting pain relief and control of inflammation.
- Corticosteroids have powerful inhibitory effects on inflammation through stabilization of cellular lysosomal membranes, reduction of vascular permeability and leukocyte adherence to vessel walls (margination), inhibition of platelet aggregation, and leukocyte diapedesis.
- Corticosteroids are also considered to be disease modifying by reducing degradative

enzymes such as matrix metalloproteinases (MMPs) and other related proteinases.
- Detrimental effects of corticosteroids include decreased chondrocyte size, loss of GAGs and decreased GAG synthesis, inhibition of proteoglycan synthesis, and chondrocyte necrosis.
- At high concentrations, CS inhibit production of important components of cartilage such as proteoglycans, collagen, and hyaluronic acid.
- Detrimental effects are less noted, and more cartilage-sparing effects have been observed with low-doses of corticosteroids.
- Currently, the most commonly used formulations of CS are triamcinolone acetonide (TA), methyl-prednisolone acetate (MPA), and betamethasone acetate (BA; Table 9.1). Less commonly used CS include isoflupredone acetate and flumethasone.
- Doses vary depending on the volume of synovial fluid in the joint, severity of inflammation and the number of other joints that require treatment. However, lower dosages of CS are currently being used than in the past (Table 9.1).
- In general, TA is used more commonly in high-motion joints and MPA in low-motion joints.
- TA injected into one joint can affect other joints in the horse. This remote effect of TA has not been observed with other CS.
- Complications of using CS include post-injection flare and infection, and laminitis (all of which are rare). Combining CS with amikacin is commonly performed to minimize the risk of infection.

### *Hyaluronan (HA)*

- HA is locally synthesized by chondrocytes, is the backbone of the proteoglycan aggregate in the extracellular matrix, and is secreted by the Type B synoviocytes of the synovial membrane.
- HA serves various important functions in the joint such as providing viscoelasticity to the joint fluid and boundary lubrication of the IA soft tissues.
- HA also may influence the composition of synovial fluid through steric hindrance of active plasma components and leukocytes from the joint cavity.
- Furthermore, HA appears to modulate the chemotactic response within the synovial membrane by reducing cell migration and decreasing rates of diffusion and flow of solutes.
- Although the exact mechanism of action is unclear, studies suggest that exogenously administered HA may replace the actions of depleted or depolymerized endogenous HA in the synovial fluid, which restores viscoelasticity, steric hindrance, and lubrication of the articular soft tissues.
- HA also may possess both anti-inflammatory and analgesic properties within the joint, and increases synthesis of high-molecular-weight HA by the synoviocytes.
- Many clinical studies in horses and people have supported HA administration in joint disease.
- Greater clinical efficacy is thought to occur with higher-molecular-weight products (Table 9.2).

### *Corticosteroid and HA Combinations*

- The combination of HA and steroids to treat inflammation intrasynovially is commonplace.
- Synergistic effects have been reported in human patients and similar effects have been seen *in vitro* in horses.

**Table 9.1.** Commonly used intra-articular corticosteroids.

| Corticosteroid | Trade name | Manufacturer | Drug concentration(mg/ml) | Dose (mg) | Duration of action |
|---|---|---|---|---|---|
| Triamcinolone acetonide | Kenalog® | Bristol-Myers Squibb | 10 | 6 to 12 | Medium |
| Methylprednisolone acetate | Depo Medrol® | Pfizer | 40 or 20 | 40 to 100 | Long |
| Betamethasone acetate | Celestone Soluspan® | Schering-Plough | 6 | 3 to 18 | Medium to long |

**Table 9.2.** Commonly used hyaluronan preparations.

| Trade name | Concentration (mg/mL) | Manufacturer | Molecular weight (Daltons)[a] | How packaged | Current recommended dose (mg)/joint |
|---|---|---|---|---|---|
| Hylartin-V | 10 | Pharmacia and Upjohn | $3.5 \times 10^6$ | 2-ml syringe | 20 |
| Hyvisc | 11 | Boehringer Ingelheim | $2.1 \times 10^6$ | 2-ml vial | 20 |
| HY-50 | 17 | Bexco Pharma | | 3-ml syringe | 51 |
| Equron | 5 | Solvay | $1.5–2 \times 10^6$ | 2-ml syringe | 10 |
| Equiflex | 5 | Chesapeake Biological | $1 \times 10^6$ | 5-ml vial | 10 |
| Synacid™ | 10 | Schering—Plough | $0.15–0.20 \times 10^6$ | 5-ml vial | 50 |
| Hyalovet | 10 | Fort Dodge/Vetrepharm | $4–7 \times 10$ | 2-ml syringe | 20 |
| Legend®[b] | 10 | Bayer | $3 \times 10^5$ | 4-ml vial | 40 mg (IV) |

[a]Manufacturer's reported molecular weight.
[b]Marketed for IV and IA use.

- The combination of CS and HA is thought to permit using a smaller dose of corticosteroid but this has not been substantiated.
- The combination of HA and CS is also thought to provide a longer clinical effect than either injection alone, especially in high motion joints.

### *Polysulphated Glycosaminoglycans (PSGAGs)*

- PSGAG is principally composed of chondroitin sulfate and is a semisynthetic preparation from bovine trachea.
- PSGAG is reported to have chondroprotective and anti-inflammatory properties as well as provide induction of articular cartilage matrix synthesis and minimization of matrix degradation.
- The exact mechanism of action of PSGAGs remains unclear but studies have revealed a significant ability of the drug to decrease lameness, modify OA through reducing bone remodeling, promote synthesis of endogenous HA, and inhibit mediators of inflammation, specifically PGE2 production.
- In one clinical study that compared PSGAGs to IA CS (Depo Medrol®, 40 mg), a regimen of weekly IA PSGAGs for three treatments had significantly improved clinical results (67%) compared to one treatment of CS (46%) in terms of returning horses back to athleticism.
- Currently, the recommended frequency of IA administration of PSGAG is three to five injections weekly.
- PSGAG may be most efficacious in joints with known or suspected articular cartilage pathology.
- IA administration is commonly combined with 125 to 250 mg of amikacin to reduce the risk of infection; this practice appears to be effective.

### *Polyglycan®*

- Polyglycan® is a patented formulation comprised of HA, chondroitin sulfate, and N-acetyl-D-glucosamine, but is not yet licensed for use as an IA medication.
- Currently, this product is used extensively in equine practice to treat horses with joint disease.
- In one controlled study in which Polyglycan® was administered IA at days 0, 7, 14, and 28, significant improvements were observed in lameness, bony proliferation, and the severity of full-thickness articular cartilage erosions seen grossly.

### *Autologous Conditioned Serum (ACS)*

- ACS (also called interleukin receptor antagonist protein; IRAP) has been used in practice to treat intrasynovial inflammation, especially in cases that are refractory to IA corticosteroids.
- Due to cost, this treatment method is used less frequently; however, clinical impressions have been favorable.
- IRAP, a substance that inhibits IL-1 activity, decreases the progression of joint disease and is believed to be present in high amounts in ACS.
- Use of this medication requires incubation of equine serum with beads coated with chromium sulfate to obtain the ACS solution.
- ACS injected into the middle carpal joints on days 14, 21, 28, and 35 following induction of OA through a chip fragment model revealed clinical improvement in lameness, and reduced gross cartilage fibrillation and synovial membrane pathology.
- The ACS is usually administered IA weekly for three to four treatments, depending on the quantity of ACS obtained.

### *Bone-Marrow- or Fat-Derived Mesenchymal Stem Cell Therapy*

- The use of bone-marrow- or fat-derived mesenchymal stem cells (MSC) has grown in popularity in equine practice in the last decade.
- It is believed that these cells possess anti-inflammatory properties and also may contribute to healing of musculoskeletal tissues by becoming incorporated into the repair tissue.
- Although more is known regarding the efficacy of MSCs in tendon injuries, early results suggest these cells also may have a place as an intrasynovial therapy.

### *Combining Medication with Anesthetic*

- Occasionally an intrasynovial structure is injected with the medication and anesthetic as a combination of treatment and diagnostic aid.
- A recent study found that the combination of TA and mepivacaine did not alter the potency or duration of action of TA.
- Clinicians should be aware that intrasynovial medications may diffuse between synovial structures. Two recent studies found that intrasynovial corticosteroids diffused between the coffin joint and navicular bursa (when medication was injected into the coffin joint) and between the TMT and the DIT joint (when medication was injected into the TMT joint).

## INTRALESIONAL

### *Introduction*

- Therapies that are injected intralesionally are usually directed at healing tendon or ligament.
- They are most often intended to augment the healing processes locally by providing the necessary components of healing to the tissue being treated.
- They may act locally to reduce inflammation and/or signal the cellular and molecular components of the injured and surrounding tissue to begin the reparative processes
- Intralesional approaches for tendon injuries are directed at maximizing the chances for a more physiologically functioning tendon.
- The ultimate goal of intralesional therapy is to maximize the chances for a tendon or ligament to repair with adequate strength and elasticity for a return to a similar level of performance with minimal risk for re-injury.

### *Hyaluronan (HA)*

- HA has been investigated for tendon injuries and adhesion prevention throughout the last two decades.
- Its use is predicated on its abilities to decrease inflammation and prevent adhesion formation through its mechanical characteristics.
- Equivocal success has been reported in horses with tendinitis treated with intralesional HA.
- One study revealed fewer adhesions within the digital flexor tendon sheath treated with HA compared to methylcellulose.
- A clinical study reported no difference in outcome or re-injury rate in performance horses when horses were injected with intralesional HA, PSGAG, or nothing following diagnosis of SDF tendinitis.
- Because of these equivocal results, HA is currently rarely used as an intralesional treatment for tendinitis.

### *Polysulfated Glycosaminoglycans (PSGAG)*

- The intralesional use of PSGAG is based on its known abilities to inhibit many of the enzymes associated with connective tissue degradation.

- PSGAG also possesses a dose-related effect on fibroblast and tenocyte metabolism, causing elevated production of collagen, noncollagen proteins, and sulfated glycosaminoglycans.
- One clinical report of using PSGAG IM had a higher rate of horses (National Hunt and Point-to-Point) return to work with no difference in re-injury rate compared to conservatively managed horses.
- The only clinical study in horses in which SDFT were injected with intralesional PSGAG (and IM PSGAG at the same time) did not find any difference in re-injury rate when horses were compared to patients injected with intralesional HA or conservative management.
- Similar to HA, PSGAG is not used frequently as an intralesional treatment for tendinitis because of the perceived lack of efficacy.

### *Mesenchymal Stem Cell Therapy*

- MSCs have been used to treat a variety of conditions such as subchondral bone cysts, cartilage and fracture repair, and tendon injuries.
- By far, the most frequent use of MSCs has been in the treatment of tendon injuries.
- The mechanism of action of how these cells influence their "trophic" activity is unknown.
- Studies suggest that MSCs secrete bioactive molecules that:
  1. Inhibit apoptosis and limit the field of damage or injury
  2. Inhibit fibrosis or scarring at sites of injury
  3. Stimulate angiogenesis and bring in a new blood supply
  4. Stimulate the mitosis of tissue-specific and tissue-intrinsic progenitor cells
- Transplantation of MSCs into various injured skeletal tissues has been shown to promote healing, and the use of autologous cells has the benefit of not inciting an immune response.
- Current theories on how transplanted MSCs act in tendon when injected intrathecally are that they either differentiate into cells capable of synthesizing tendon matrix or they secrete important factors that induce adjacent cells to synthesize tendon matrix.
- The two current techniques for MSC transplantation use cells derived from fat or cultured bone marrow aspirates.
- The fat-derived stem cells do not involve a culture step and have the advantage of lower cost and speed of preparation (cells are returned to the practitioner with 48 hours). However, the cell mixture is believed to be heterogeneous with regard to cell type.
- The bone-marrow-derived MSC technique involves aspirating bone marrow from the sternum or tuber coxae, transferring to a laboratory for culture and expansion, and then implanting the cell population (approximately 10 to $50 \times 10^{6}$) under ultrasound guidance.
- The optimal time of injection is still unclear, but greater cell viability is thought to occur if the injection is performed two to three weeks following injury.
- Most believe that the repair tissue maximally benefits when MSCs are injected in the initial phase of healing, therefore at least by four to five weeks following injury.
- The primary benefit of MSCs is thought to be a better quality repair tissue that reduces the re-injury rate. One study in racehorses reported an 18% re-injury rate in treated horses compared to a 56% re-injury rate for horses receiving no intralesional therapy.

### *Platelet-Rich Plasma (PRP)*

- The reasoning behind the use of PRP comes from the knowledge that growth factors are released from platelet α granules including platelet-derived growth factor (PDGF), transforming growth factor-β (TGF-β), fibroblastic growth factor (FGF), vascular endothelial growth factor (VEGF), insulin-like growth factor-I (IGF-I), and epidermal growth factor (EGF).
- Many animal models have demonstrated positive effects on tissue healing of these growth factors, both alone and in combination.
- Current studies have reported beneficial effects of PRP on equine ligament and tendon, both *in vitro* and *in vivo*.
- Optimal timing of injection, as with MSCs, is still unclear, but early in the healing process (two to four weeks following injury) is most likely ideal so that the growth factors present in the PRP can contribute to the cellular environment and enhance matrix components integral to healthy repair tissue.

### *Corticosteroids*

- Perilesional corticosteroids are occasionally used in acute or chronic cases to treat tendinitis/desmitis as an anti-inflammatory.
- Use of corticosteroids should probably be reserved for tendon or ligaments in which

minimal or no structural damage is seen ultrasonographically.
- Numerous other uses have been described for local injection of corticosteroids in areas of inflammation such as over splint bones, muscle/back soreness, and various soft tissue inflammatory conditions. The choice and amount of steroid seem to be empirical and dependent on the clinician.

## ORAL/NUTRITIONAL

### *Nonsteroidal Anti-inflammatory Drugs (NSAIDs)*

#### Introduction

- NSAIDs are a large group of drugs with differing degrees of analgesic, anti-inflammatory, and antipyretic properties (Table 9.3).
- In a recent survey conducted by Merial of 1,400 horse owners and trainers, 96% of respondents stated that they use NSAIDs and 82% said they administer them without consulting a veterinarian.
- Oral formulations come in many varieties such as pills, paste, granules, and powder, allowing the consumer to choose the best formulation for each individual.
- Although used frequently, NSAIDs have a wide range of side effects, mainly involving the gastrointestinal, renal, and cardiovascular systems. These side effects can result from multiple doses and vary among the different drugs.
- NSAIDs act by inhibiting cyclooxygenase (COX) enzymes that convert arachadonic acid into prostaglandins and thromboxanes.
- There are two well-known isoenzymes of the COX enzymes: COX-1 and COX-2. COX-1

**Table 9.3.** Commonly used NSAIDs and their modes of action, formulations, and doses for equine musculoskeletal disorders.

| Name of NSAID | Primary inhibitory action | Available formulations | Recommended dose |
|---|---|---|---|
| Phenylbutazone | Cox-1 and -2 | Powder<br>Tablets<br>Paste<br>Injectable solution | 2.2 to 4.4 mg/kg SID or BID |
| Flunixin meglumine | Cox-1 and -2 | Paste<br>Granules<br>Injectable solution | 1.1 mg/kg SID |
| Acetylsalicylic acid | Cox-1 > Cox-2 | Gel<br>Powder<br>Granules<br>Paste<br>Tablets | 25 to 35 mg/kg SID or BID |
| Meclofenamic acid | Cox-1 and -2 | Granules | 2.2 mg/kg SID for 5 to 7 days |
| Naproxen | Cox-1 and -2 | Granules<br>Tablets<br>Suspension | 10 mg/kg SID or BID |
| Firocoxib | Cox-2 | Paste and IV | 0.1 mg/kg SID and 0.09 mg/kg IV |
| Carprofen | Cox-2 > Cox-1 | Tablets<br>Injectable solution | 0.7 mg/kg SID |
| Vedaprofen | Cox-2 > Cox-1 | Gel<br>Injectable solution | 2 mg/kg loading dose followed by 1 mg/kg q 12 hours |
| Meloxicam | Cox-2 > Cox-1 | Suspension<br>Injectable solution | 0.6 mg/kg SID for max of 14 days |

is responsible for producing prostaglandins involved in normal physiological functions such as gastric and renal function and hemostasis. The COX-2 isoenzyme is considered to be an important inducible mediator of inflammation in several organs; it is also primarily responsible for the inflammatory pathway.
- The variation in efficacy and toxicity of the different NSAIDs is closely related to inhibition of the different COX isoenzymes (Table 9.3).
- Some NSAIDS are more potent inhibitors of COX-1 than COX-2, some equally inhibit both, and others inhibit COX-2 more than COX-1.
- In general, the anti-inflammatory and analgesic properties of NSAIDs are believed to be mainly due to inhibition of the inducible COX-2, whereas the adverse effects seem to be caused by inhibition of the constitutive COX-1.

### Phenylbutazone (PBZ; Butazolidin®, Butatron™, Bizolin®, Phenylbute™, Phenylzone®, Equiphen®, Butequine®, Superiorbute®, Equizone 100™)

- Oral PBZ is an inexpensive NSAID that has potent pain relief, antipyretic, and anti-inflammatory properties.
- It provides pronounced analgesia and reduction in inflammation in many common lameness problems including chronic OA.
- PBZ is usually very good at minimizing pain and inflammation associated with orthopedic surgical procedures when it is administered prior to surgery.
- The plasma half-life after oral administration has been reported to be between 6.2 and 6.7 hours. However, absorption can be influenced by the drug's formulation and its route of administration.
- Oral PBZ is considered relatively nontoxic at repeated doses of 2.2 mg/kg twice a day or less.
- The gastrointestinal tract is the most commonly affected site of toxicity following oral administration, contributing to ulcers (oral, esophageal, gastric, cecal, and right dorsal colon) and a protein-losing enteropathy.
- Studies have shown a better clinical improvement in lameness when using a combination of NSAIDs than PBZ alone. However, several toxic effects have been reported with combinations of NSAIDs in horses sensitive to NSAIDs.

### Flunixin Meglumine (Banamine®)

- Oral flunixin meglumine can be used to treat musculoskeletal disorders in horses, but because of its cost compared to PBZ, oral formulations are used infrequently.
- The combination of flunixin with other NSAIDs has been used to enhance the analgesic properties of these drugs in performance horses.
- After oral administration the drug is rapidly absorbed with a peak in plasma levels within 30 minutes, and the plasma half-life is approximately 1.6 hours.
- This rapid absorption of flunixin may be important in minimizing potential ulcerogenicity.
- The onset of action after oral administration occurs after two hours, with the greatest effect obtained between two and 16 hours; some activity may persist for up to 30 hours.
- Similar to phenylbutazone, recent feeding delays absorption.
- Oral administration of flunixin is relatively safe. However, high doses for long periods can cause gastrointestinal intolerance, hypoproteinemia, and hematological abnormalities.

### Acetylsalicylic Acid (Aspirin)

- Acetylsalicylic acid is a weak acid that reduces platelet aggregation and has analgesic, anti-inflammatory, and antipyretic properties.
- It is only available in oral forms and has a limited clinical use in the horse.
- Aspirin has been reported to decrease platelet numbers and prolong bleeding time in horses with doses between 12 mg/kg and 24 mg/kg. These effects can occur after a single dose and can last 48 hours.
- Due to its effect on platelets, aspirin has been used for the treatment of navicular syndrome and laminitis. However, its therapeutic benefits for these diseases are not well defined.

### Meclofenamic Acid (Arquel)

- Meclofenamic acid (MA) is another oral NSAID used to treat lameness and chronic musculoskeletal conditions such as navicular disease, OA, and laminitis in horses.
- Compared with other NSAIDs, MA has a slow onset of action of 36 to 96 hours for full effect.
- It is not clear whether feeding time dramatically affects absorption. One study demonstrated that fasted and non-fasted ponies had similar oral absorption, whereas another study

demonstrated that plasma levels could be delayed by feeding.
- High doses produce clinical signs of toxicity similar to those of PBZ (at a dose 13 to 18 mg/kg).

### Naproxen (Equiproxen®, Naprosyn)

- Naproxen is an NSAID with analgesic and antipyretic properties that has been used primarily to treat myositis and soft tissue problems.
- In a study using an equine myositis model, oral naproxen was reported to be superior to oral PBZ, with faster relief of inflammatory swelling and associated lameness.
- Naproxen has a wide margin of safety. Oral administration of 3× the recommended dose for 42 days did not cause signs of toxicity.

### Firocoxib (Equioxx®)

- Firocoxib is a new coxib class of NSAID that reduces inflammation, pain, and fever, and decreases the risks of toxicities of other traditional NSAIDs.
- Firocoxib has been approved by the FDA for controlling pain and inflammation associated with OA in horses.
- The bioavailability after oral administration in horses is 79%, with a time to peak concentration of 3.9 hours and elimination half-life of 30 hours. It is well distributed in the body, including synovial fluid, liver, fat, kidney, and muscle.
- In a study done in horses with chronic OA and navicular syndrome, no significant differences in clinical improvement were found between horses treated 14 days with firocoxib compared to PBZ.
- Compared with other NSAIDs, firocoxib is relatively safe, with no clinical and biochemical signs of NSAID toxicity reported using the recommended dose.
- Toxicity signs were reported when the drug was used at 3 to 5× the recommended dose for 30 to 92 days.
- The main downfall to firocoxib is that it is more expensive than PBZ.

### Carprofen (Zenecarp, Rimadyl®)

- Carprofen is a propionic acid NSAID approved in Europe for oral use in horses.
- Its mechanism of action in horses is still unclear; however, it is described as being a more effective analgesic than anti-inflammatory agent.
- Carprofen in horses was well tolerated when given at twice the oral dose for 14 consecutive days.
- The benefit of using this NSAID in horses compared to other available NSAIDs needs further investigation.

### Vedaprofen (Quadrisol®)

- Vedaprofen is approved in Europe for oral use in horses. It is recommended for musculoskeletal disorders and soft tissue lesions.
- Side effects of this drug are those that are associated with the use of NSAIDs in general.
- The main toxic effect is ulcer formation in the gastrointestinal tract, making it contraindicated for use in foals under 6 months of age.

## *Nutraceuticals*

### Introduction

- Management of OA in horses has historically focused on symptom relief by using NSAIDs or IA steroids.
- Because NSAIDs and IA steroids have failed to provide complete symptom relief and have been associated with several side effects, alternative therapies have been proposed to help alleviate the side effects or incomplete relief of symptoms of conventional therapies.
- Nutraceutical/supplement administration is one of the most commonly used alternative therapies in horses.
- There are a multitude of nutraceutical products available with a variety of differing ingredients. Quality control of the products is not performed, and therefore the actual content of the ingredients can be quite variable.
- Even though these substances are neither nutrients nor pharmaceuticals, they are generally used in an attempt to lower the dose of other drugs that are more problematic and provide medical benefits that prevent or treat the disease.

### Glucosamine (GLN)

- Glucosamine is an aminosaccharide essential for normal growth and repair of articular cartilage.
- Glucosamine compounds have been used as nutraceuticals in horses due to the possible role they have in stimulating chondrocyte

metabolism and reducing inflammation in the articular cartilage.
- Exogenous glucosamine can be produced synthetically or derived from marine exoskeletons or beef carcasses.
- There are three commercially available forms of exogenous GLN: hydrochloride (HCl), sulfate, and N-acetyl-D-glucosamine. There are potential differences in efficacy among the forms.
- Several studies have generally identified that GLN induces the production of new cartilage while protecting cartilage that is already present.
- GLN stimulates synthesis of proteoglycans (PG) and collagen while inhibiting PG degradation, and may protect against some of the negative effects of steroids on cartilage.
- High levels of GLN used for *in vitro* experiments that were effective have never been achieved in experimental models in humans and animals.
- The most consistent information that can be extrapolated from human studies regarding GLN is that it appears to take four to eight weeks before it begins to work, and that GLN likely achieves its best effects when used preventatively prior to the advancement of the disease process.

### Chondroitin Sulfate (CS)

- CS is a long-chain polysaccharide that constitutes about 80% of all glycosaminoglycans in articular cartilage.
- CS is often derived from shark and bovine cartilage and it is rather expensive to synthesize and extract.
- The species or tissue of origin of CS can determine differences in the concentrations, pharmacokinetics profile, molecular composition and weight, metabolic fate, and therapeutic results. Because of these differences, it cannot be assumed that all CS products have the same clinical effect.
- In horses, CS is orally absorbed; however, the molecular weight and source can have a direct influence on its permeability across the gastrointestinal tract and its bioavailability.
- Reports using radiolabelled CS have demonstrated that it achieves high concentrations in plasma, articular cartilage, and synovial fluid.
- Exogenous CS has been shown to reduce cartilage degradation and profound anti-inflammatory effects on several tissues involved with joint metabolism.
- In a synovitis model in horses, CS was found to be less effective than PSGAG administered IM (Adequan®, Luitpold Pharmaceuticals, Inc.) for relief of lameness, stride length, and carpal flexion.
- Another study using the same model suggested that CS had therapeutic value irrespective of the route of administration (oral or IM). However, the time of onset of clinical improvement was slower with oral administration of CS.

### Glucosamine and Chondroitin Sulfate (GLN-CS)

- Many equine nutraceuticals contain a combination of GLN and CS; this combination may be synergistic.
- It has been shown that the combination improves collagen synthesis in tenocytes and ligament cells, and it may be important for use in accessory joint structures.
- Oral administration of GLN-CS at doses greater than those recommended in horses is associated with a good safety profile, with no alterations in hematological or clotting profiles.
- GLN-CS has demonstrated equivocal success in research models, showing no clinical benefit in one study and improvement in another.
- In studies of clinical cases treated with this compound, horses showed improved lameness, flexion, and stride length, while navicular horses showed significant improvement in soundness compared to placebo controls.

### Hyaluronic Acid (HA)

- Hyaluronic acid is a natural component of articular cartilage that is responsible for the viscoelastic and lubricating properties of synovial fluid. It plays an important physiologic role in nutrition of the articular cartilage.
- Many oral formulations of HA have become available for horses. There are several anecdotal reports about the use of these products in horses but scientific evidence of oral absorption, bioavailability, distribution, and controlled evaluation of efficacy of oral HA products in the horse are lacking.
- One controlled double-blinded study in yearling Thoroughbreds with OCD lesions of the tarsus demonstrated that the mean effusion

score for the treated horses (100 mg orally once daily) was significantly lower than for the placebo group.

### Methylsulfonylmethane (MSM)

- Methylsulfonylmethane is a normal oxidative metabolite product of industrial-grade DMSO that is naturally found in small amounts in fruit, alfalfa, and corn. It is very soluble in water.
- MSM can be found as a product by itself or in combination with GLN and/or CS.
- MSM has been used as a nutraceutical because of its analgesic, anti-inflammatory, and antioxidant properties.
- Very little is known about oral administration of MSM safety and toxicity or its clinical use to manage OA in horses.
- Horses receiving oral MSM and vitamins demonstrated that MSM could exert some protective effect on oxidative and inflammatory exercise-induced injury related to jumping.
- In another uncontrolled non-peer reviewed study, MSM administration was associated with improved performance in Standardbred racehorses in training.

### Avocado and Soybean Unsaponifiable Extracts (ASU)

- The unsaponifiable portions of avocado and soybean oils are extracted via hydrolysis to make up fractions of one-third avocado oil and two-thirds soybean oil.
- It appears that this mixture has synergistic properties, but the active ingredient is still unknown.
- *In vitro* studies have suggested that ASU extracts may have a positive effect on both the inflammatory cascade and structural components of the cartilage matrix.
- One controlled study that used horses with induced OA in the middle carpal joint failed to demonstrate any significant clinical effects.
- ASU has been combined with GLN-CS in some products.

### Fatty Acids

- Polyunsaturated fatty acids (PUFAs) are essential fatty acids that are found in fish and plants.
- The two principal essential fatty acids are linoleic acid and α-linolenic acid. In the body they are desaturated and elongated to produce analogs of arachadonic acid (n-6 fatty acid).
- When horses in a study received fish oil in their diet, there was an increase in the concentration of eicosapentaenoic acid (EPA; Omega-3 fatty acids) and docosahexaenoic acid (DHA; Omega-3 fatty acids) in their serum, compared to horses that received corn oil.
- Studies have demonstrated that n-3 fatty acid supplementation can reduce or inhibit the inflammatory and matrix degradative response elicited by chondrocytes during OA progression.
- Cetyl myristoleate (CM) is another fatty acid that is used in equine nutraceuticals.
- CM is an ester, Omega-5 fatty acid that may act by inhibition of the 5-lipo-oxygenase pathway, which is responsible for the metabolism of leukotrienes (potent inflammatory mediators from the arachadonic acid cascade).
- One equine product containing CM demonstrated lower lameness scores compared to placebo horses in a double-blinded OA clinical trial.

### Collagen Hydrolysate (CH)

- Collagen hydrolysate is a food ingredient that has been used in humans to improve joint comfort and function.
- CH is derived from bovine or porcine skin and bones.
- Orally administered CH has been shown to be absorbed intestinally and accumulate in cartilage.
- In contrast with other nutraceuticals, no direct analgesic and anti-inflammatory effects have been found after using CH.
- The theory behind CH is that it provides amino acids specific to the collagen network, playing an important role in the structure and function of cartilage by directly stimulating chondrocytes to synthesize collagenous matrix.
- There are no published reports on the safety, absorption, metabolism, or clinical use of CH in the horse.

## CORRECTIVE TRIMMING AND SHOEING

### *Introduction*

- The distal limb has sophisticated mechanisms to dampen shockwaves and spread out the duration of loading and unloading.
- Extreme exercise or changes to the foot or ground surface that enhance the accelerations

within the distal limb or shorten the duration of loading increase the likelihood of injury.
- Both shock waves and large loads associated with weight-bearing are potentially injurious.
- Attaching steel shoes with nails is known to increase the magnitude and frequency of impact shock waves.
- The goals of therapeutic shoeing are almost always designed to strategically address the way structures in the distal limb are stressed to prevent, palliate, or heal injury to the structures that are at risk or diseased.
- Prevention of injury is aimed at normalizing the stresses in the distal limb, and treatment is designed to protect a structure from stress as it heals.
- The hoof capsule differs from other musculoskeletal structures because it is constantly growing and being worn away, and because of its remarkable viscoelastic properties.
- In response to prolonged application of abnormal stresses, the hoof capsule becomes distorted, and prolonged altered load-bearing changes both wear and growth patterns.

### *Examination of the Limb for Therapeutic Shoeing*

- Examination of the horse and its limbs should appropriately identify both predisposing factors and disease.
- When the disease process is identified, a specific strategy may be formulated that may involve a combination of therapeutic shoeing, medication, and/or surgery.
- Frequently the clinician is expected to develop a strategy to treat a lameness originating from the digit that cannot be ascribed to a particular structure or process, compensate for a conformational abnormality, correct an apparent imbalance in the foot, or more than one of the above. This necessitates careful examination of the morphology of the foot, the relationship between the foot and the rest of the limb, and the way the foot interacts with the ground.
- Radiography is a useful adjunct to assess the relationship between the phalanges and the relationship between the phalanges and the hoof capsule.
- Examination of the hoof capsule morphology is useful to indicate where the wall is unduly stressed. With the foot on the ground, the following findings suggest too much stress in the wall in one portion of the foot: compression of growth rings (Figure 9.1), flares at the quarter or toe, underrunning of the heels, and proximal and/or abaxial displacement of the coronary band.

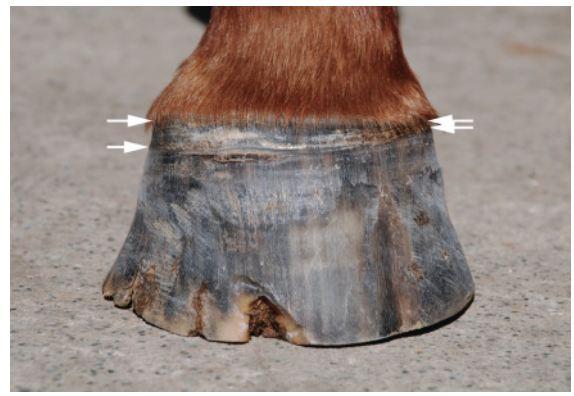

**Figure 9.1.** Photograph of a horse with growth rings that diverge markedly from medial to lateral. This horse has medial distal displacement of the distal phalanx. Courtesy of Andy Parks.

- With the foot held up, the ground surface should be examined for mediolateral symmetry including position of the heels and irregularities in the outline of the capsule.
- The ground surface should be evaluated for appropriate width in relation to length, and position of the widest point of the foot in relation to the center of the ground surface.
- The normal pattern of landing at a walk or trot is either lateral heel/quarter first or both heels simultaneously.
- Toe-first landing and medial first landing are both indicative of an abnormality in gait. Excessive lateral landing, especially if seen at the trot, is also considered abnormal.
- Radiography for assessment of conformation and balance of the distal limb requires lateral and standing dorsopalmar radiographs.
- In an ideal foot, the dorsal wall is parallel to the dorsal surface of the distal phalanx, the sole depth is appropriate for the size of horse (optimally greater than 15 mm), the center of rotation of the DIP joint is slightly palmar to the center of the weight-bearing surface of the foot, and the average angle that the solar margin of the distal phalanx makes with the ground is 6° (Figure 5.37).
- The most important observations on a DP view are the angle of the articular surface of the distal phalanx to the ground and the symmetry of the articulations. Additionally, the

height of the coronet should be correlated with the position of the distal phalanx, and any flares identified.

### *The Trim*

- The foundation for any corrective shoeing is the trim. If the trim is not appropriate, whatever shoe is used will be less than optimal.
- There are only three basic variables that can be altered with trimming: depth of the sole, angle of the dorsal wall, and mediolateral symmetry.
- The length of the wall at the toe is predicated on the depth of the sole; the wall is either level or slightly longer than the adjacent sole.
- The length of the heels is then predicated on the length of the toe and the angle of the hoof-pastern axis.
- Distortions in the hoof capsule may necessitate additional measures.

### *Shoeing*

- The basic principles that can be implemented to improve the function of the equine foot include moving the center of pressure, shock absorption, easing movement about the distal joints in the limb, altering the distribution of force, and altering the motion of the limb during the flight phase of the stride (Table 9.4).
- A wide range of tools can be applied to achieve these principles, and all too often the focus is on the method rather than underlying principles.
- The same type of shoeing does not work for every disease or for every horse, and willingness to use trial and error will increase the likelihood of success.

#### Shock Absorption

- High-frequency and high-energy shock waves are generated during the impact phase of the

**Table 9.4.** Principles for therapeutic trimming and shoeing.

| Modification | Goal of modification | Examples of how to achieve goal |
|---|---|---|
| Move the center of pressure | Moves the ground reaction force in relation to center of foot/center of rotation of the DIP joint to move load of weight-bearing from one side of the foot to the other | Wedges<br>Extensions<br>Alter width of web unevenly, either medial vs. lateral or dorsal vs. palmar |
| Shock absorption | Dampen vibrations associated with deceleration and acceleration | Aluminum shoes instead of steel<br>Synthetic polymer shoes<br>Viscoelastic pads |
| Ease movement about the DIP joint | Shorten arm of extensor moment about the DIP joint at break-over | Roll the toe<br>Rocker the toe<br>Square the toe<br>Set the shoe back |
| Change distribution of force | Increase ground contact of part or all of sole, bars, and frog to reduce load on wall | Heart-bar shoes<br>Custom pad (e.g., heart-bar pad)<br>Pour-in pads |
| Motion of limb during flight | To prevent interference or improve esthetics of gait | Forging: Speed up break-over of forelimb and retard break-over of hindlimb.<br>Brushing in hindlimbs: Widen gait by squaring toe, adding traction or trailer to lateral branch, with or without lowering medial wall |

stride and to a lesser extent during the breakover phase.
- Numerous manipulations of the foot are known to influence the magnitude of shock waves including the application of a shoe, the method of shoe attachment, the material of the shoe, pads interposed between the shoe and hoof, traction devices on the shoe, and the surface of the ground.
- Most interventions that involve shoeing increase the shock waves of impact. Therefore, the natural damping mechanisms within the digit almost always function better in a healthy horse if it is barefooted.
- Studies have determined that a steel shoe reduces the natural damping mechanisms within the foot and foot expansion. Aluminum and synthetic shoes may impede the natural damping mechanisms less than steel.
- Rigid pads and pads that become compressed with use are unlikely to improve shock absorption, but pads that undergo viscoelastic compression and relaxation with the phases of the stride may offer significant benefit. However, there is little information available on the benefit of commercially available pads.
- Shoe modifications can be used to either increase (calks, toe grabs, rim shoes) or decrease (wide web flat shoes or half round shoes) traction, which will alter the impact vibrations within the foot. Too much traction increases the horizontal impact vibrations, and too little predisposes to slipping. There are few data to assist a clinician on the choice of these devices to optimize traction.
- The ground surface also affects the impact shock waves that occur within the foot but the surface is usually dictated by the nature of the work.

## Weight-Bearing and the Center of Pressure

- The center of pressure determines the relative load-bearing of the different parts of the foot
- At rest, the center of pressure is at or near the center of the ground surface of the foot.
- The center of pressure can be moved in numerous ways. The simplest that works on all surfaces is to increase the relative length of one side of the foot to the other, and the center of pressure will move to the elevated side.
- Increasing the length of the heel or toe moves the center of pressure toward or away from the center of rotation of the DIP joint (Figure 9.2); in doing so, it shortens or lengthens the arm of the extensor moment about the joint.
- Shoe extensions increase the length of the lever arm, thus increasing the leverage that the

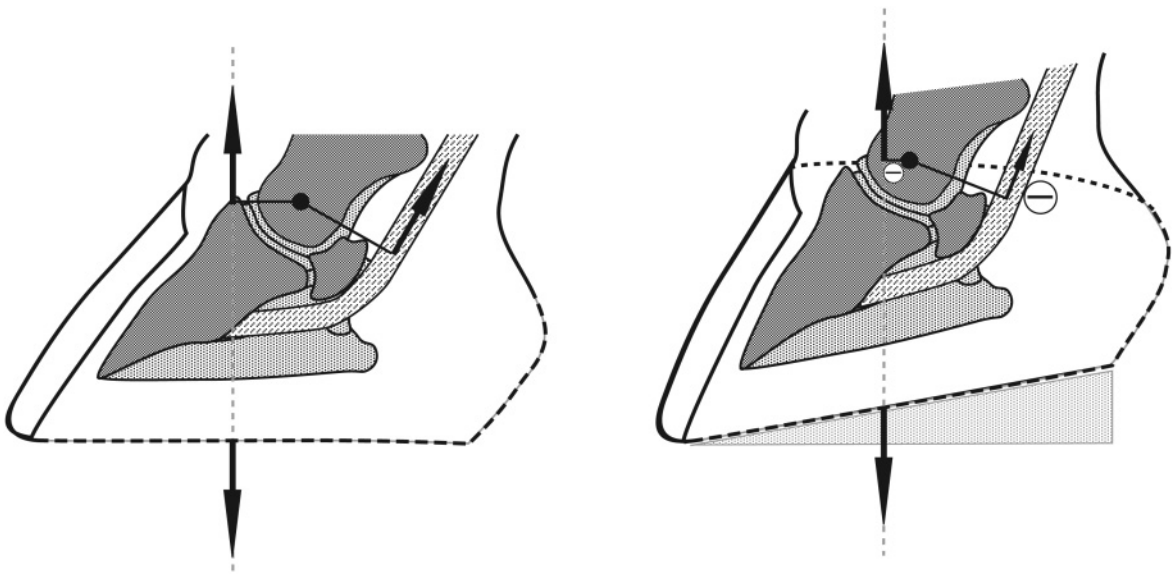

**Figure 9.2.** Schematic illustration demonstrating the effect of a heel wedge on moments about the distal interphalangeal joint. Elevating the heel shortens the moment arm of the extensor moment; as the flexor moment arm remains approximately the same, the force in the deep digital flexor tendon is decreased. The center of pressure remains approximately in the center of the foot. Vectors represented by arrows are for illustrative purposes and do not represent real values. Courtesy of Andy Parks.

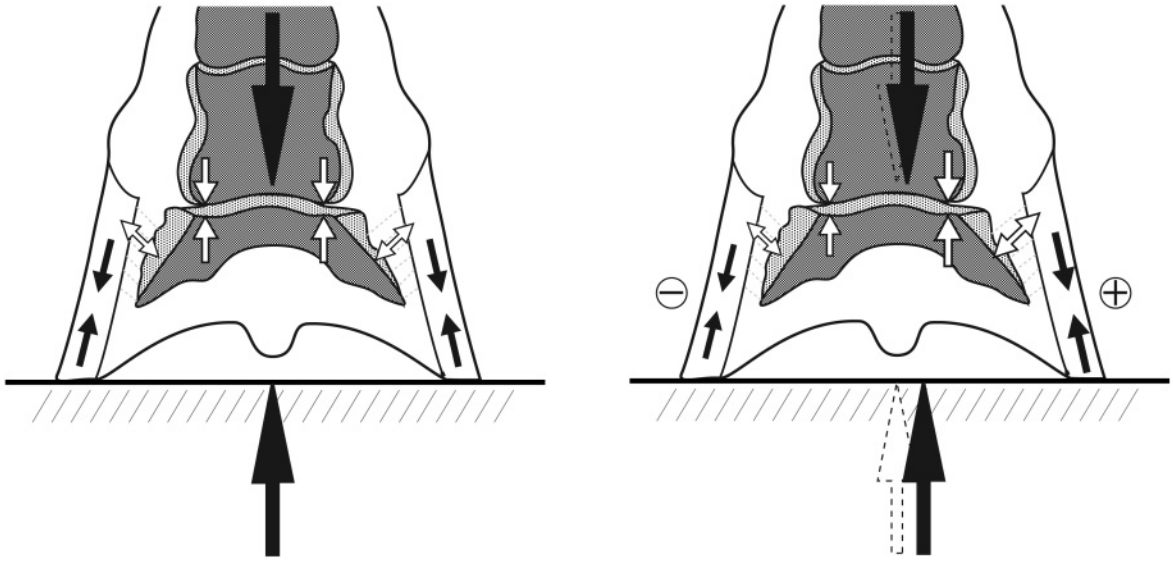

**Figure 9.3.** Schematic illustration to demonstrate the effects of moving the center of pressure in the frontal plane on the structures in the foot. As more weight is borne by one side of the foot, the bones and joints become more compressed, the tension in the collateral ligament is reduced, and the tension in the lamellae is increased. These changes are reversed in the opposite side of the foot. Vectors represented by arrows are for illustrative purposes and do not represent real values. Courtesy of Andy Parks.

ground can exert about the center of rotation; for example, an egg-bar shoe moves the center of pressure away from the toe.

- A decrease in the width of the web increases the descent of the shoe into the ground surface, and an increase in web width decreases it. Moving the center of pressure in this manner to one side of the foot decreases the stress in the ipsilateral collateral ligament and the contralateral lamellae and articular surfaces (Figure 9.3).
- Moving the center of pressure toward the heels reduces tensile stress in the DDFT, compressive stress on the navicular bone, and tensile stress in the dorsal lamellae (Figure 9.4).

### Ease of Movement

- It is assumed that moving the point of break-over more palmar improves the function of the foot and relieves lameness associated with the palmar aspect of the foot. However, studies have been unable to demonstrate a decrease in the duration of the break-over or a change in the flight of the foot.
- Moving the point of break-over does not change the peak moment about the DIP joint or the peak force on the navicular bone, even though it decreases the length of the moment arm of the ground reaction force about the DIP joint at break-over (Figure 9.5).
- The most likely explanation for the clinical improvement is that the foot breaks over (unrolls) more smoothly, thereby decreasing the stresses in the underlying tissues.
- The medial and lateral abaxial branches of the shoe also can be beveled to improve the ease of motion during turning.

### Distribution of Force

- The main rationale for distributing part of the load of weight-bearing over more of the sole and frog is to reduce load-bearing by the lamellae and wall (i.e., recruiting the entire ground surface in horses with laminitis).
- The distribution of force on the foot can be changed by increasing the size of the web of the shoe so that it has greater contact with the ground and the foot (Figure 9.6).
- A variety of materials, such as polyurethane or silicone putty, may be placed inside the contour of the shoe to fill the space between the branches of the shoe and the ground surface of the foot to accomplish the same thing.

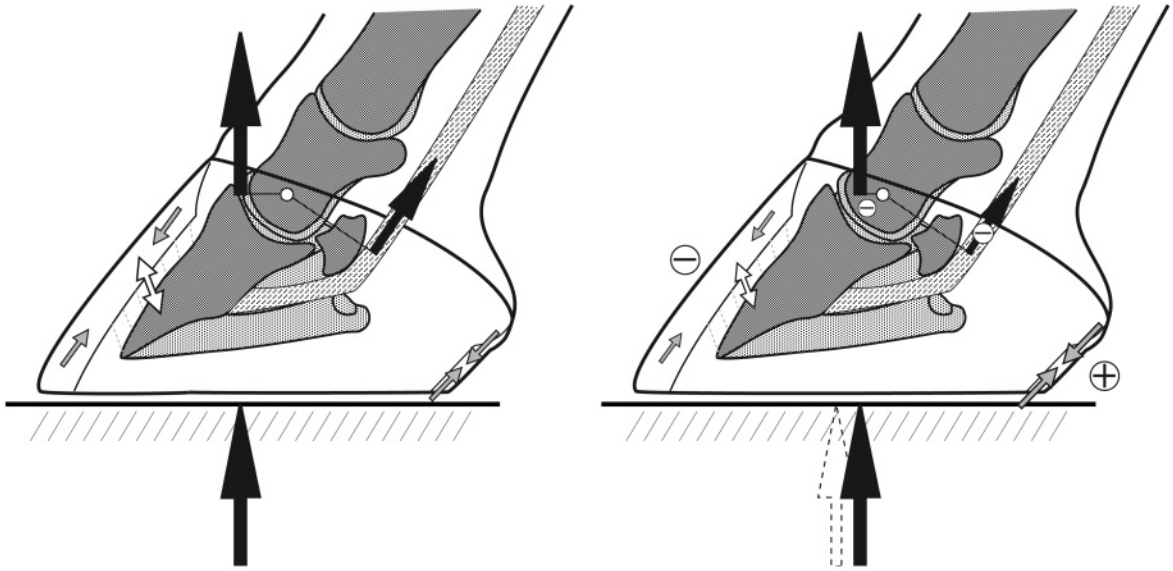

**Figure 9.4.** Schematic illustration to demonstrate the effect of moving the center of pressure in a palmar direction. The arm of the extensor moment becomes shorter, and because the arm of the flexor moment remains approximately the same, the force in the deep digital flexor tendon is decreased. Vectors represented by arrows are for illustrative purposes and do not represent real values. Courtesy of Andy Parks.

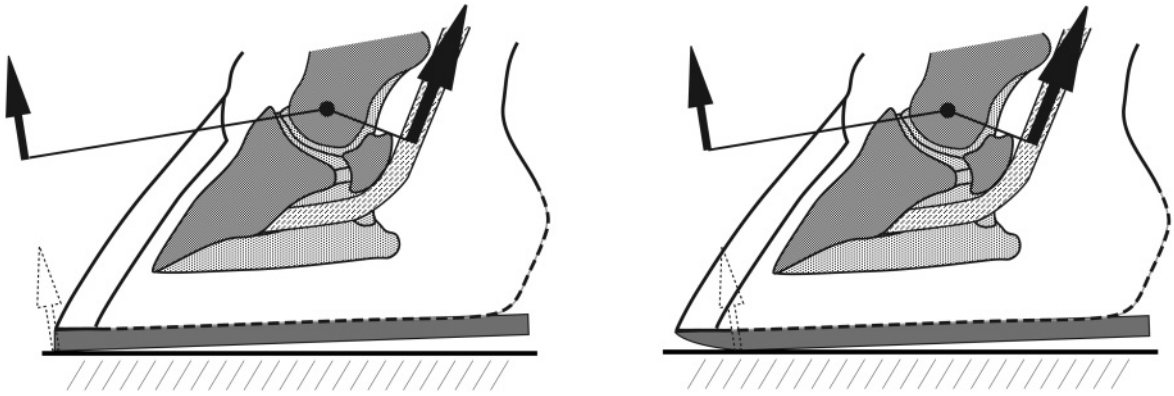

**Figure 9.5.** Schematic illustration to demonstrate the effect of moving the point of break-over in a palmar direction. At the moment the heel leaves the ground, the arm of the extensor moment is shorter in shoes with rolled toes compared to shoes without rolled toes. Vectors represented by arrows are for illustrative purposes and do not represent real values. Courtesy of Andy Parks.

- A heel plate in conjunction with a composite also helps distribute the forces on the foot.
- This assumes that the wall and lamellae bear most of the weight, but the role of the sole and wall in weight-bearing is poorly understood. Recent studies suggest that when a horse is standing on a deformable surface such as sand or turf, the load is transmitted through the sole to the wall, and presumably to the lamellae.

## Motion of Limb During Flight

- The way the foot lands influences the position of the foot during the stance phase, and the position of the foot during the stance phase influences the way the foot leaves the ground.
- Redirection of the path of flight of the foot is primarily directed at preventing interference injuries, but might also be directed at improv-

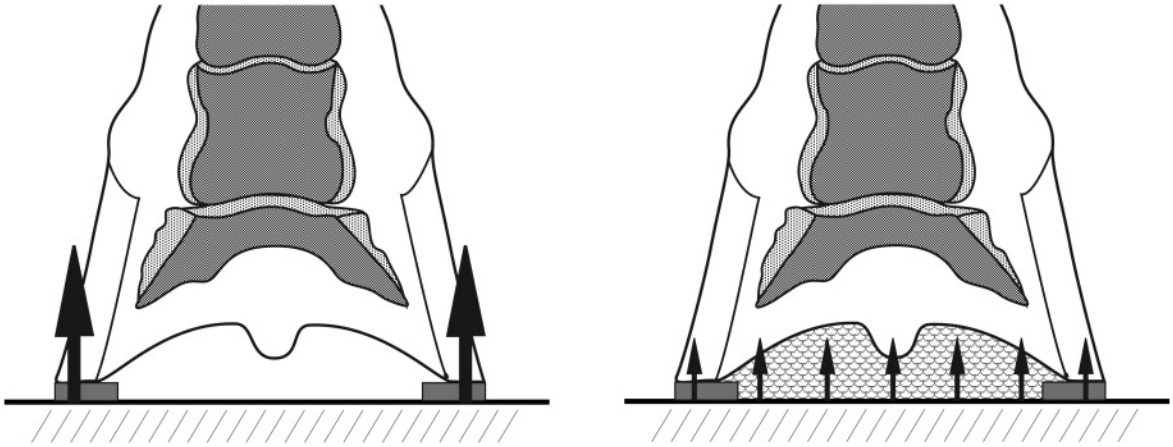

**Figure 9.6.** Schematic illustration to demonstrate the effect of distribution of pressure. It is unknown how the redistribution of force on the ground surface of the foot affects the transmission of load to the distal phalanx. Vectors represented by arrows are for illustrative purposes and do not represent real values. Courtesy of Andy Parks.

ing the appearance of flight path for esthetic reasons.

- Any manipulation of the foot to change the flight path that causes the structures to move outside of their normal plane of motion or increases their range of motion potentially predisposes these structures to injury.
- Interference is more common in shod horses and it is thought that conformation, imbalance, and fatigue are the main precipitating factors.
- The best starting point for preventing interference is to ensure the foot is balanced and use the lightest appropriately sized shoe compatible with the level of work.
- The general principle to treat interference between contralateral limbs is to widen the gait. This is generally achieved by a combination of squaring the toe, increasing traction laterally, and using a lateral trailer on the hind foot, plus or minus lowering the medial side of the foot.
- When the above approaches are ineffective and interference persists, then the limb being interfered with should be protected with boots, and the shoe on the interfering limb may be safed.

### *Protection and Support*

- Protection and support are the goals of therapeutic shoeing.
- "Protection" in relation to shoeing indicates reduction or prevention of pressure on part of the ground surface of the foot.
- Protection is usually provided by eliminating pressure in a given area by redistributing the forces on the foot or by placing a shielding device superficial to the area. This may involve using a wider web shoe, a strategically placed bar, or a treatment plate across the entire ground surface of the foot.
- "Support" is more ambiguous but can be used in relation to maintaining an anatomical relationship; for example, after the DDFT has been severed, a heel extension may be used to maintain the foot flat on the ground.
- A structure also can be considered "supported" when the stress on it is reduced; for example, in the case of a strained DDFT, elevating or extending the heels decreases the stress in the tendon.
- It is common to hear that a modification to the shoe is "supporting" the heels, usually by extending or elevating them. In reality, extending or elevating the heels is maintaining the alignment of the foot-pastern axis and "supporting" the DDFT, but increasing the load on the heels, thereby increasing the propensity that they will deform and decreasing their growth.
- Most actions used to provide "support" of the foot involve movement of the center of pressure.

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**Revised from "Principles of Therapy for Lameness" in *Adams and Stashak's Lameness in Horses, Sixth Edition*, by Laurie R. Goodrich, Troy N. Trumble, Nicolas Ernst, and Andrew Parks.**

10

CHAPTER

# Musculoskeletal Emergencies

## SEVERE UNILATERAL LAMENESS

### *Introduction*

- Non-weight-bearing lameness of a single limb is often due to synovial infection, hoof abscess, or a fracture/luxation.
- Soft tissue injuries typically cause less severe swelling and lameness than fractures, and improve more quickly over time.
- Horses with infections usually worsen over time vs. those with soft tissue injuries that improve with time.

### *Etiology*

- Infection (hoof abscess, synovial infection, etc.) is a cause of severe unilateral lameness.
- Fractures anywhere in the limb also cause non-weight-bearing lameness in a single limb.
- Penetrating injuries (e.g., nail in the foot) are a cause.

### *Clinical Signs*

- Acute onset of non-weight-bearing lameness is the typical clinical sign of severe unilateral lameness.
- Limb deviation, abnormal posture, crepitus with manipulation, and pain on palpation are often present in horses with fractures (Figures 6.74 and 10.1).
- Horses with foot abscesses may have no visible swelling but will have extreme pain with hoof testers.
- Synovial infections often have synovial effusion, peri-synovial edema, heat, and pain on palpation.
- Purulent drainage from synovial cavities is often present with penetrating injuries (Figure 10.2).

### *Diagnosis*

- Horses with suspected fractures should have the limb radiographed (or ultrasound performed) as soon as possible.
- Synoviocentesis with evaluation of the fluid for white blood cell count and total protein remains the most expedient method to document synovial infection.
- Radiography (including contrast radiography) and ultrasound can be used to document osteomyelitis and other soft tissue injuries within the synovial cavities.
- Hoof testers are usually very helpful to document the location of a hoof abscess.
- Perineural anesthesia should not be used in horses with suspected fractures, but can be

*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.

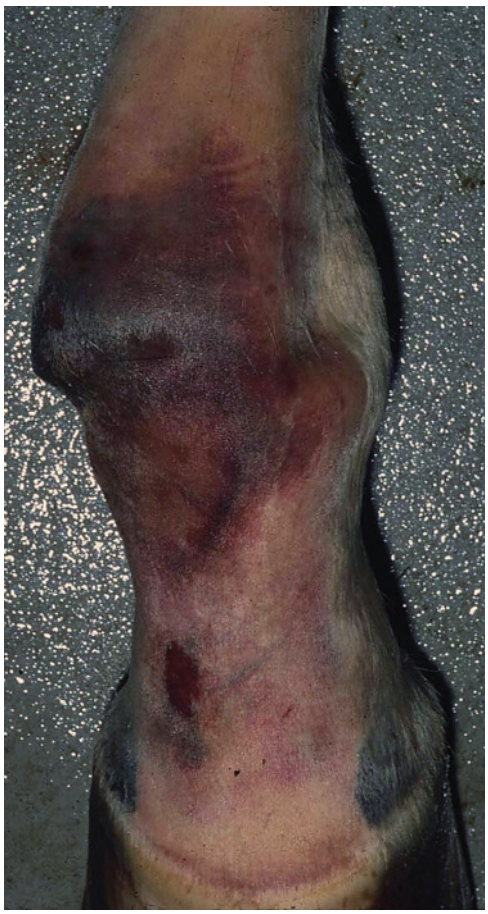

**Figure 10.1.** Horse with a closed luxation of the fetlock joint with deviation of the distal limb and extensive soft tissue bruising.

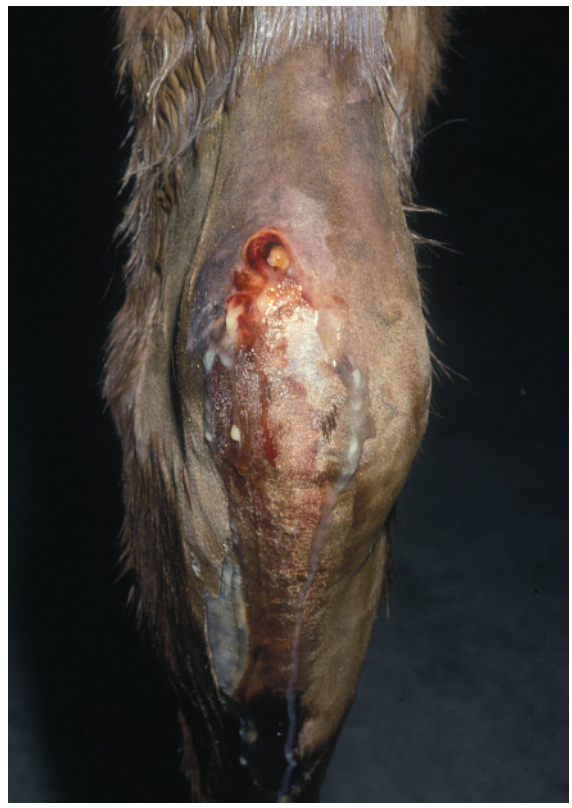

**Figure 10.2.** Horse with a puncture wound and infection within the calcaneal bursa. Purulent drainage is exiting the wound and the horse was non-weight-bearing.

helpful to localize the lameness to the foot in cases of suspected sole abscesses.

### *Treatment*

- Fractured limbs should be immobilized to reduce anxiety and pain and for transport if surgical treatment is warranted. Some fractures of the upper forelimb and hindlimb cannot be adequately immobilized. See fracture immobilization, below.
- Hoof abscesses are usually treated by drainage, soaking, and foot protection until the hoof defect has cornified.
- Synovial infections are best treated with arthroscopic or endoscopic lavage; parenteral, regional, and intrasynovial antimicrobials; and NSAIDs. See synovial infections, below, for further details.

### *Prognosis*

- The prognosis is variable, depending on the type and location of the fracture and the severity and location of the infection.
- Most horses with foot abscesses have a very good prognosis. Other conditions within the foot such as keratomas, chronic laminitis, osteomyelitis, etc. should be suspected in horses with recurring abscesses.
- Most horses with synovial infections that are treated aggressively have a good prognosis.

## SEVERELY SWOLLEN LIMB

### *Introduction*

- Numerous conditions can result in limb swelling.

- The severity of lameness is very important in the initial assessment of the horse.
- Horses with fractures, luxations, and synovial infections usually have severe lameness together with localized swelling at the affected site.
- Less severe lameness with generalized limb swelling is often seen with cellulitis, lymphangitis, traumatic wounds, or hematomas.
- Horses with traumatic soft tissue injuries usually have localized swelling, can bear weight on the limb, and respond quickly to first aid treatment.
- Young horses with osteochondritis dissecans (OCD) often have localized joint effusion with only mild to no lameness.

### *Etiology*

- The cause is usually traumatic or infectious if lameness is moderate to severe.
- The cause is most likely developmental if lameness is mild or absent.

### *Clinical Signs*

- The clinical signs include localized or diffuse swelling of the limb with or without edema, pain, and heat
- Joint effusion alone is often seen in horses with OCD or acute cases of synovial infection.
- Diffuse, painful swellings are often associated with cellulitis, lymphangitis, or small puncture wounds.
- Effusion together with edema around the synovial cavity are often present with synovial infections (Figure 10.3).
- Crepitus may be felt or heard during manipulation of complete long bone fractures.
- The severity of lameness can be variable, but most acute cases are often lame at the walk or non-weight-bearing.
- Diffuse swelling with serum oozing through the skin may be present with lymphangitis and cellulitis.

### *Diagnosis*

- Often, a tentative diagnosis can be made based on physical findings and the severity of the lameness.
- Radiography is used to document a fracture/luxation or OCD within an effusive joint.
- Ultrasonography is very helpful to evaluate soft tissue swelling from any cause.

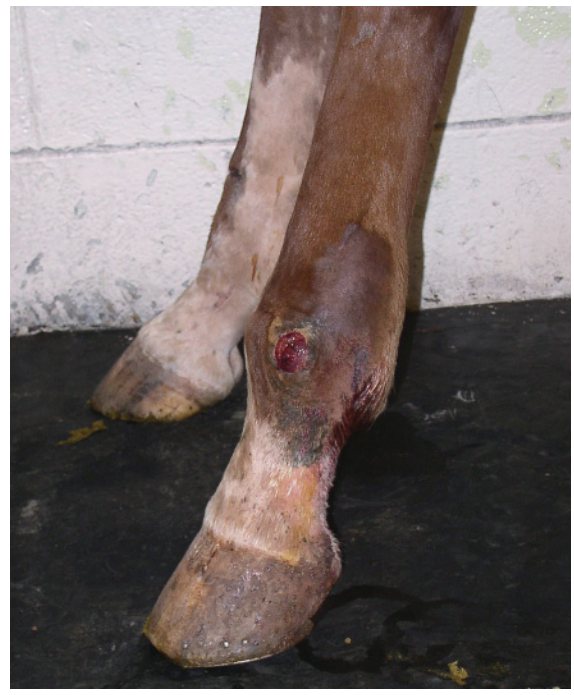

**Figure 10.3.** This wound on the dorsal aspect of the fetlock healed by second intention and trapped infection within the fetlock joint. Joint effusion, soft tissue swelling, and periarticular edema were present.

- Bacterial culturing should be performed in horses with suspected diffuse or localized infection.

### *Treatment*

- The definitive treatment depends on the cause, but initial first aid treatment of soft tissue swelling is often similar and may consist of hydrotherapy, topical and systemic NSAIDs, bandaging, and systemic antimicrobials if infection is suspected.
- Fractures should be immobilized if possible and the horses sent to a hospital for treatment.
- Horses with synovial infections should be treated with synovial lavage (preferably with the arthroscope), and systemic, regional, and intrasynovial antimicrobials.

### *Prognosis*

- The prognosis usually is very good for horses with cellulitis, except for horses with lymphangitis.

- Horses with soft tissue injuries usually respond well to first aid treatment, but the prognosis depends on the structure that is damaged (i.e., muscle vs. tendon).
- Horses with fractures/luxations and synovial infections have a worse prognosis than horses with cellulitis.
- Horses with OCD usually have a very good prognosis.

## LONG BONE FRACTURES/LUXATIONS

### *Introduction*

- Complete long bone fractures usually result in a severe non-weight-bearing lameness.
- Horses are not readily ambulatory on three limbs and often become very anxious when they are unable to place weight on a fractured limb.
- Fractures of the phalanges and MC/MT occur most commonly.
- The most common sites for joint luxations include the fetlock, pastern, tarsus (PIT or TMT joints), carpus, stifle, and hip.

### *Etiology*

- Repetitive overloading of the bone with eventual complete loss of the structural support is a cause of fractures.
- Single-event traumatic injury such as an awkward step, fall, kick, or strenuous event also is a cause.

### *Clinical Signs*

- Acute onset of severe non-weight-bearing lameness is a characteristic sign.
- Limb deviation, abnormal limb posture, crepitus with manipulation, swelling, and pain on palpation are often present.
- Abnormal rotation or angulation of the joint often can be identified with complete joint luxations. Pain is obvious with manipulation.
- Both fractures and luxations can be open or closed.

### *Diagnosis*

- The diagnosis is usually confirmed with radiography.
- Stress radiographs are helpful to identify joint luxations and loss of collateral ligament support (Figure 10.4).

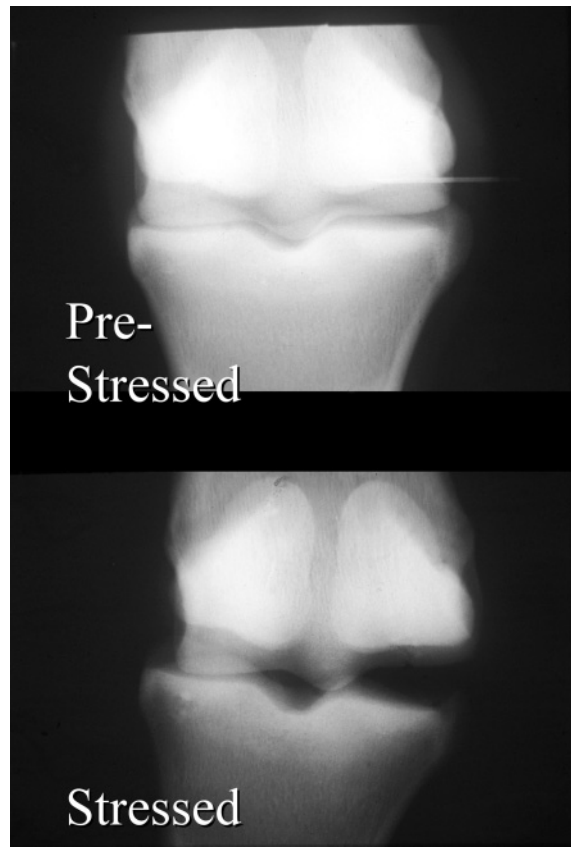


**Figure 10.4.** Pre-stressed and stressed radiographs of a collateral ligament injury demonstrating an increased gap at the medial edge of the joint. Courtesy of Alicia Bertone.

- Ultrasound can be used to identify fractures of the upper limb such as the pelvis and scapula.

### *Initial Treatment (Fracture Immobilization)*

- First aid measures should be directed toward minimizing further damage to the fractured limb and maintaining it in a position and condition that will facilitate repair (Table 10.1).
- The goals of first aid fracture management are to prevent damage to neural and vascular elements of the limb, prevent skin penetration of the fracture fragments or minimize further contamination of an existing wound, relieve anxiety of the animal by stabilizing the fractured limb, and minimize further damage to the fractured bone ends and surrounding soft tissue.

**Table 10.1.** Contents of fracture first aid kit for horses.

| Material | Purpose |
|---|---|
| Cotton, rolled and/or sheet | Padding under splint-cast combination or Robert Jones bandage |
| Gauze, sterile and nonsterile | Applying dressing to wounds if present and Robert Jones bandage |
| Razor or portable clippers | Removing hair from around wound |
| Antibiotic ointment | Topical dressing for open wounds |
| Support wrap (Vetrap, Elastikon, etc.) | Robert-Jones bandage or bandage under a splint-cast combination |
| White tape | Secure splints or boards to bandage |
| PVC splints (varying lengths) | Splint-cast combination |
| Fiberglass cast material | Splint-cast combination |
| Board splints (varying lengths) or aluminum rod | Splinting of radial and tibial fractures |
| Drugs (antibiotics, sedatives, NSAIDs) | Tranquilization, pain relief, and treatment of open fractures |
| "Leg saver" Kimzey splint (optional) | Immobilization of distal limb fractures |

**PVC = polyvinyl chloride, NSAIDs = nonsteroidal anti-inflammatory drugs.**

- Fractures of the upper forelimb and hindlimb in horses are nearly impossible to stabilize with external splints.
- Immobilization of fractures involving the metacarpus/metatarsus, radius, and tibia is critical to prevent the development of an open fracture during transport.

## *Methods of Immobilization Based on Location (Figure 10.5)*

### Phalanges and Distal Metacarpus

- The phalanges and distal metacarpus are the most common locations for fractures to occur.
- The splinting techniques should attempt to counteract the bending force at the fetlock.
- A cotton bandage combined with a dorsally placed polyvinylchloride (PVC) splint or a splint-cast combination with the limb maintained in a straight line from the carpus to the hoof provides optimal immobilization (Figure 10.6).
- Over-padding should be avoided because fracture fragments can move within a large bulky bandage.
- A half-limb fiberglass cast with the limb in a similar position also can be used, but is more difficult to apply than a PVC splint.

### Midforelimb (Mid-metacarpus to Distal Radius)

- Fractures in this location are stabilized best with a Robert-Jones type bandage combined with full-limb PVC splints applied caudally and laterally.
- The splints should extend from the elbow to the ground at 90° to each other and be tightly secured to the bandage using nonelastic white tape.
- Lightweight, rigid material such as wood, aluminum, or flat steel may be used effectively for splinting if PVC is not available.

### Middle and Proximal Radius

- Preventing abduction of the distal limb is the goal when immobilizing fractures in this location. Limb abduction allows the proximal fracture fragment to penetrate the skin on the medial aspect of the radius.

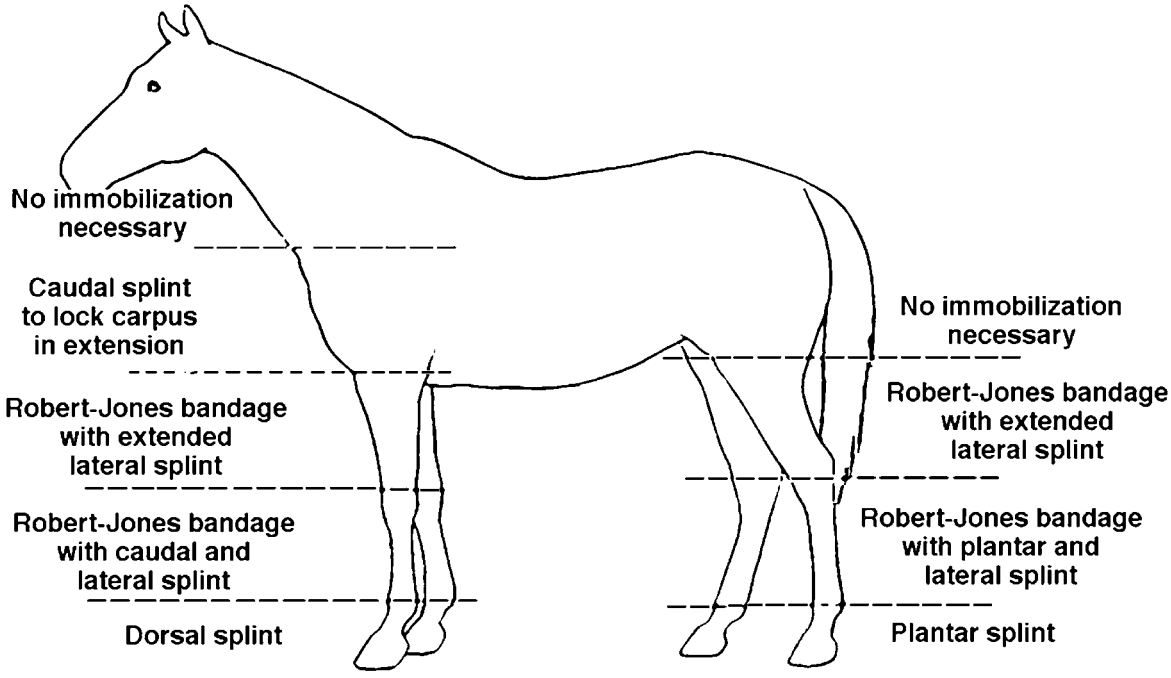


**Figure 10.5.** Functional divisions of the horse's limbs that can be used as a guide for appropriate application of external support to immobilize fractures for transport.

- Limb abduction is best achieved by applying a Robert Jones bandage (including the caudal PVC splint) similar to that used with mid-forelimb fractures, but the lateral splint is extended up the lateral aspect of the shoulder, scapula, and chest.
- A wide board (15 to 20 cm) or metal rod appears to work better than PVC for this lateral splint. The upper extension of the lateral splint should lie against the shoulder and scapula so that it will prevent abduction of the distal limb during ambulation (Figure 10.7).

### Proximal to the Elbow

- Complete fractures of these bones disable the triceps muscle apparatus, making it impossible for the horse to fix the elbow in extension for weight-bearing.
- A full-limb cotton bandage with a full-limb caudally applied PVC splint will keep the carpus extended and help restore triceps muscle function (Figure 6.72A).
- Not all fractures in these locations require stabilization as the risk of skin penetration is extremely low, and foals may not have the upper forelimb strength to move the limb with a splint in place.

### Phalanges and Distal Metatarsus

- Fractures in this location can be managed similarly to those in the forelimb except that the PVC splint is best placed on the plantar surface of the limb when using the splint-cast combination.
- Splints applied to the dorsal surface of the hindlimb over a bandage tend to break more readily than in the forelimb.
- The Kimzey splint may be used to stabilize fractures in this location, similar to that in the forelimb (Figure 6.34).

### Middle and Proximal Metatarsus

- A Robert-Jones bandage with PVC splints applied laterally and caudally using the calcaneus as a caudal extension of the metatarsus provides adequate support for fractures in this location.
- The bandage should be less extensive than in the forelimb because it will be difficult to secure the splints to the limb if the bandage is too bulky.

**Figure 10.6.** A bandage and PVC splint applied to the distal limb of a horse to immobilize a fractured phalanx. The splint was placed dorsally and secured with white nonelastic tape. Courtesy of Chris Ray.

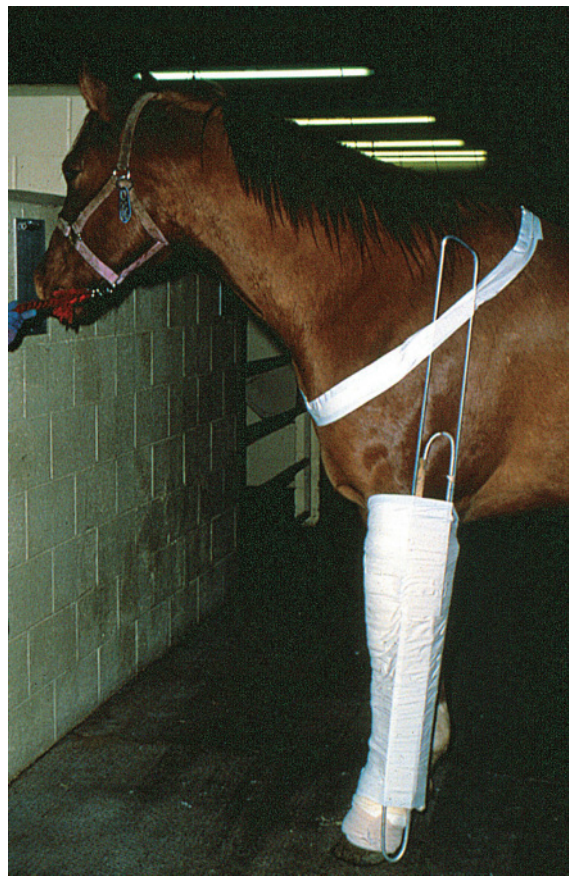

**Figure 10.7** A full-limb bandage with a caudal splint is placed and secured. A loop of aluminum rod has been shaped to extend from the ground up over the shoulder and scapula to help immobilize a radial fracture. Courtesy of Chris Ray.

### Tarsus and Tibia

- Fractures in this location are difficult to stabilize because of the reciprocal apparatus and its effect on joint motion in the tarsus and stifle.
- The main principal for stabilization is similar to that of the radius, which involves preventing abduction of the distal aspect of the limb.
- A single laterally placed splint that is bent to follow the angulation of the limb and extends proximally above the stifle joint works well to prevent abduction. The splint is applied over a full-limb Robert-Jones bandage, and is best made of lightweight metal such as aluminum that can be bent into the correct position (Figure 10.8).
- An alternative to the metal splint is a wide board (15 to 20 cm) that extends from the ground to the ilium and is applied to the lateral aspect of the bandage.

#### *Definitive Treatment*

- Treatment options for horses with fractures/luxations range from stall confinement (with or without splinting) to euthanasia.
- Fractures of the phalanges, MC/MT, and ulna are the most amenable to some type of internal fixation.

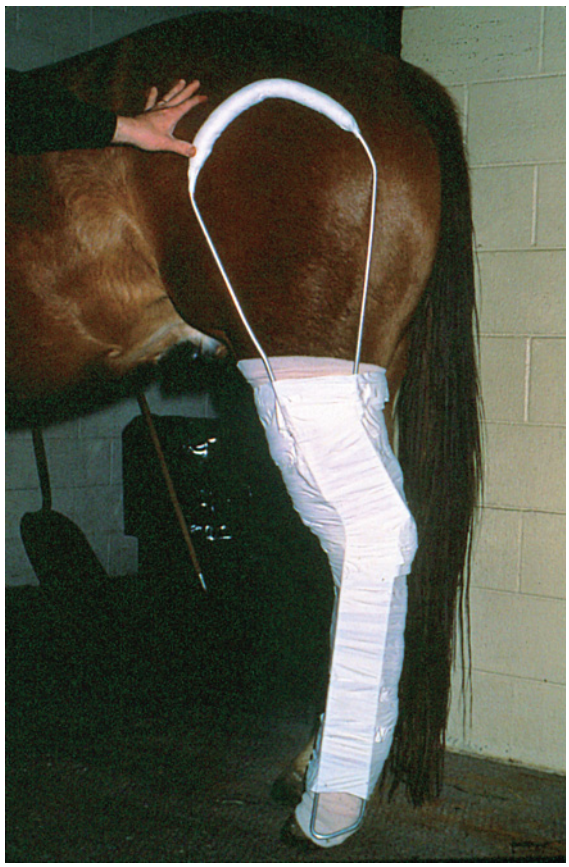

**Figure 10.8.** An aluminum rod has been shaped to extend from the ground up over the stifle and hip in this horse with a tibial fracture. Similar to radial fractures in the forelimb, the splint prevents abduction of the limb distal to the fracture, further stabilizing fractures of the tibia. A wide board could be used instead of the aluminum rod. Courtesy of Chris Ray.

- Casting alone may be used for some fractures of the phalanges, MC/MT, and distal radius, and for luxations of the fetlock, carpus, and tarsus.
- In general, the smaller the horse, the more likely that any type of treatment will be successful.

### *Prognosis*

- The prognosis is extremely variable, depending on age, size, and temperament of the horse, and the type and location of the fracture.
- In general, the prognosis for open fractures is much worse than for closed fractures, and worse for adults than for foals.
- Comminuted long bone fractures in adults have a poor prognosis and are usually not treated.

## SYNOVIAL INFECTIONS

### *Introduction*

- Synovial infections are often associated with penetrating injuries to the navicular bursa, coffin and pastern joints, digital flexor tendon sheath, fetlock joint, hock joint, calcaneal bursa, tarsal sheath, and carpal joints.
- Any wound affecting a synovial structure greater than 24 hours old should be considered to have an established synovial infection.
- Synovial infections without external trauma are often associated with previous treatments (i.e., injections) or hematogenous spread (joint–ill in foals).

### *Etiology*

- Trauma associated with a wound is a common cause of synovial infection.
- The cause may also be iatrogenic from previous injections or surgery.
- Hematogenous synovial infections occur most commonly in foals, and can be associated with failure of passive transfer.

### *Clinical Signs*

- Adult horses with infected synovial structures usually present because of severe lameness.
- Synovial effusion is usually present together with concurrent soft tissue edema, swelling, and heat
- Those with concurrent open wounds often have a yellowish to clear, sticky fluid consistent with synovial fluid exiting the wound (Figure 10.2).
- The severity of lameness in foals is less consistent than in adults and multiple synovial cavities can be involved.

### *Diagnosis*

- Wound involvement of synovial cavities can be documented by probing with a finger or teat cannula (Figure 10.9), injecting sterile fluid into the synovial structure at a site remote from the wound and observing for fluid exiting the wound, contrast radiography (Figure 5.32), or documentation of air within the synovial space using radiographs or ultrasound.
- The presence of abundant fibrin, severe synovial effusion, and thickening of the synovial

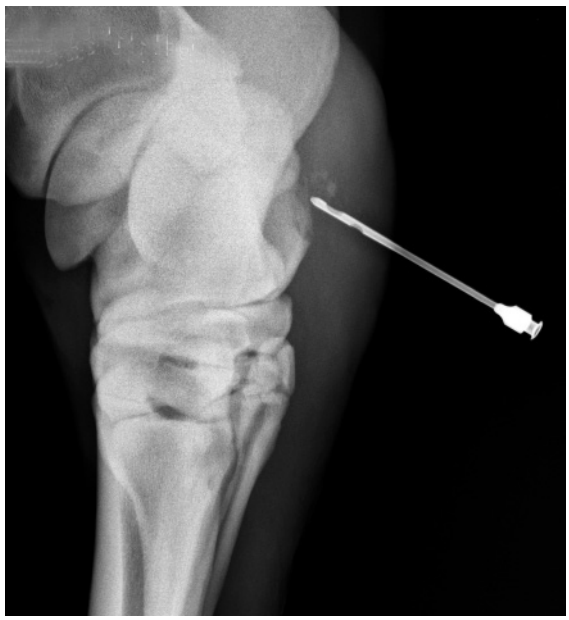

**Figure 10.9.** This relatively minor wound on the medial aspect of the tarsus was associated with fractures of the sustentaculum tali and a probe went directly to the bone and entered the tarsal sheath.

lining on ultrasound also is suggestive of infection.

- Synovial fluid white blood cell counts greater than 30,000 cells/μl and total protein concentrations greater than 3.5 to 4 g/dl are highly consistent with infection.
- Culturing the synovial fluid, fibrin, or synovial membrane is the most definitive method to document infection, but not all cultures are positive.
- Foals with joint-ill with fibrinogen values of 900 mg/dl or greater should be suspected of having concurrent osteomyelitis.

### *Treatment*

- Systemic and intrasynovial antimicrobials are the cornerstone of treatment (Tables 10.2, 10.3). IV or IM administration is recommended for a minimum of five days, with oral antimicrobials continued for two to three weeks based on culture results.
- IV regional or intraosseous perfusion also may be used to increase the concentration of the antimicrobials to the site of the infections

**Table 10.2.** Systemic antimicrobials used to treat horses with synovial wounds and infections.

| Antimicrobial | Dosage | Combinations and indications |
|---|---|---|
| Penicillin | 22,000 to 44,000 IU/kg q 6 to 12 hours IV or IM | Combined with aminoglycoside, ceftiofur, or enrofloxacin (Gram-positive infections) |
| Ampicillin | 22 mg/kg IV q 8 hours | Combined with aminoglycoside |
| Cefazolin | 10 mg/kg q 8 hours IV or IM | Can be used alone; usually combined with gentamicin or amikacin |
| Gentamicin | 6.6 mg/kg q 24 hours IV or IM | Combined with penicillin, cephalosporin, or ampicillin (Gram-negative infections) |
| Amikacin | 7 mg/kg q 12 hours or 14 mg/kg q 24 hours IV or IM | Combined with penicillin, cephalosporin, or ampicillin (Gram-negative infections) |
| Ceftiofur | 2.2 mg/kg q 12 hours | Can be used alone or combined with penicillin or aminoglycoside (staphylococcus infections) |
| Enrofloxacin | 7 mg/kg q 24 hours IV or PO | Not recommended for foals (resistant Gram- negative infections; poor for anaerobes) |
| Doxycycline | 10 mg/kg PO q 12 hours | Used as follow-up to parenteral antimicrobials |
| Trimethoprim-sulfonamides | 30 mg/kg q 12 hours PO or 3 to 5 mg/kg q 12 hours PO (based on trimethoprim) | Used as follow-up to parenteral antimicrobials |
| Vancomycin | 7.5 mg/kg IV q 8 hours | Methicillin-resistant staphylococcal and enterococcal infections |

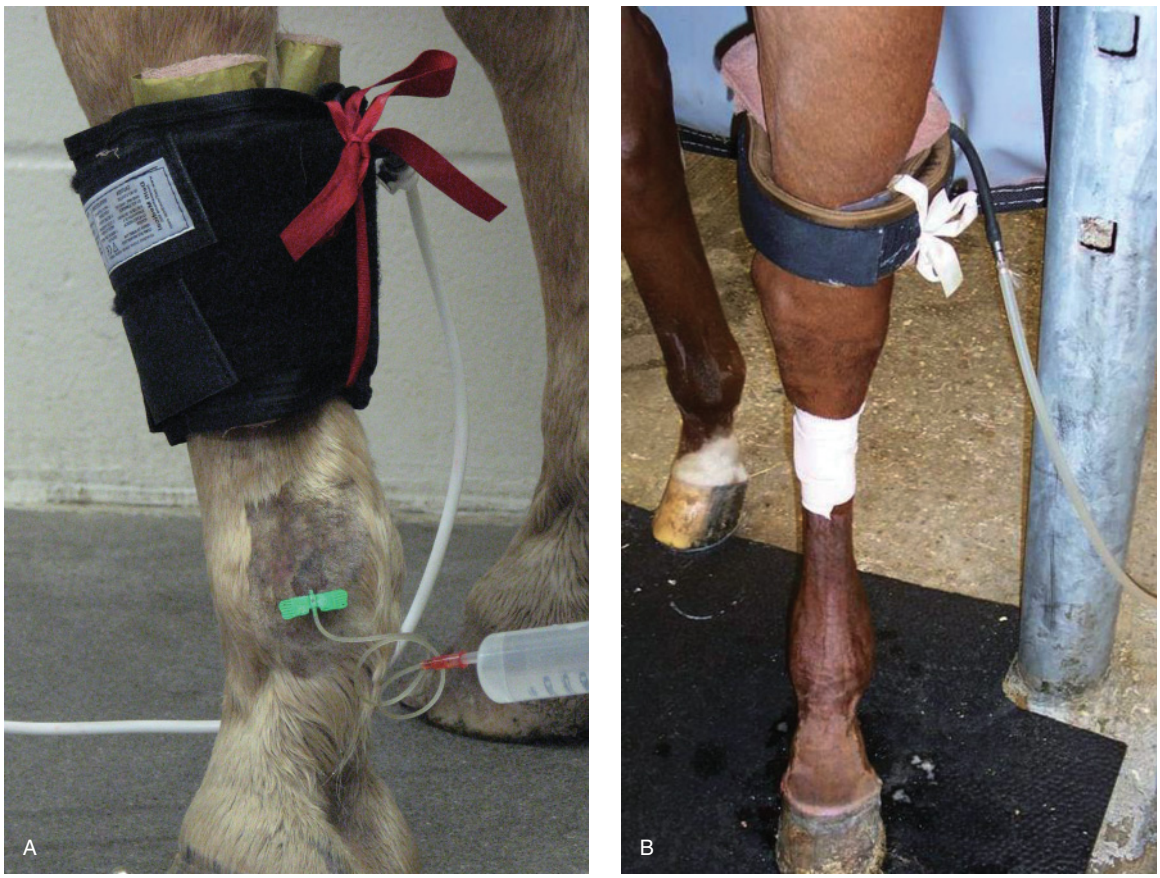


**Figure 10.10.** Intravenous regional limb perfusions using a pneumatic tourniquet. The tourniquet is placed above the site of the lesion and a vessel is selected to infuse the antimicrobials, the palmar vein in (A) and the saphenous in (B). Rolls of gauze can be applied over the vessels for extra pressure; these can be seen under the tourniquet in both (A and B). Courtesy of Jeremy Hubert.

(Figure 10.10). Amikacin is commonly used for this purpose and IV perfusion is preferred over intraosseous perfusion (Table 10.3).
- Synovial lavage/drainage is recommended. Methods include through and through lavage, arthroscopic/endoscopic exploration, arthrotomy, or placing a drain within the synovial cavity.
- Arthroscopic/endoscopic lavage of infected synovial cavities is preferred because it facilitates removal of foreign material and fibrin, debridement of bone or tendinous lesions if present, and assessment of cartilage damage.

### *Prognosis*

- In general, the quicker synovial involvement can be identified and treated, the better the prognosis.
- The absence of secondary bone or tendinous injuries should improve the prognosis regardless of the location of the injury.
- In one study of horses treated with arthroscopy/endoscopy, 90% of the horses survived and 81% of the horses returned to performance.
- Horses with wounds involving the navicular bursa and coffin joint remain a difficult challenge to return to performance.
- About 60% of foals with hematogenous synovial infections alone survive compared to 35–40% with concurrent osteomyelitis.

## TENDON LACERATIONS

### *Introduction*

- The distal limb is prone to deep lacerations that may involve tendons because of the

**Table 10.3.** Concentration-dependent antimicrobials used for local delivery (intrasynovial, regional perfusion, impregnated beads, etc.) in horses with synovial infections.

| Antimicrobial | Dosage | Methods of delivery |
|---|---|---|
| Cefazolin | 1 g | All modes of local delivery |
| Ceftazidime/ceftriaxone | 2 g (100 mg/hour) | Intrasynovial continuous infusion |
| Penicillin/ampicillin | One-fourth to one-half systemic dose | Intrasynovial or regional perfusion |
| Gentamicin | 0.5 to 1 g<br>1.8 mg/kg/day | All modes of local delivery<br>Intrasynovial continuous infusion |
| Amikacin | 0.5 to 1 g<br>5.5 mg/kg/day or<br>2 or 3 g (100 mg/hour) | All modes of local delivery<br>Intrasynovial continuous infusion |
| Ceftiofur | 150 mg to 1 g | Intrasynovial or regional perfusion |
| Ticarcillin plus clavulanate | 300 to 400 mg | All modes of local delivery |
| Tobramycin | 0.5 to 2 g | PMMA or other impregnated beads |
| Vancomycin | 300 mg | Regional perfusion<br>PMMA or other impregnated beads |
| Enrofloxacin | 500 mg | All modes of local delivery |

horse's flight response, kicking defense, and high speeds.
- Tendon lacerations occur more commonly in the hindlimbs than in the forelimbs.
- Most tendon lacerations/wounds occur in the mid-MC/MT region and involve the extensor tendons most frequently (common digital extensor tendon in the forelimb and long digital extensor tendon in the hindlimb) or the flexor tendons (SDFT and/or DDFT).
- Extensor tendon injuries are often avulsion type trauma with loss of skin over the dorsal aspect of the cannon bone (Figure 10.11).
- Laceration of both flexor tendons in the MC/MT region is a medical emergency similar to a fracture.

## *Etiology*

- These injuries may result from external trauma from sharp objects.
- Horses may jump sharp objects, fences, or often pull excessively if their lower limb is trapped, inciting significant tendon injury.
- Tendon rupture may occur from excessive exercise but this is less common than external trauma.

## *Clinical Signs*

- Characteristic gait deficits occur following complete tendon lacerations.
- If the extensor tendons are severed, the foot may move more freely and the horse may intermittently knuckle at the fetlock due to failure to extend the digit during limb placement (Figure 10.12).
- Horses are often not lame on weight-bearing with extensor tendon injuries but may have difficulty advancing the limb.
- Complete severance of both the DDFT and SDFT will result in hyperextension of the fetlock and the toe coming off the ground during limb placement (Figure 10.13A).
- Laceration of just the SDFT will cause the fetlock to drop with normal foot placement (Figure 10.13B).
- If the suspensory ligament is also severed, the fetlock will drop almost to the ground.
- Severe lameness accompanies flexor tendon lacerations.
- Palmar/plantar lacerations around the fetlock region may involve the digital flexor tendon sheath.

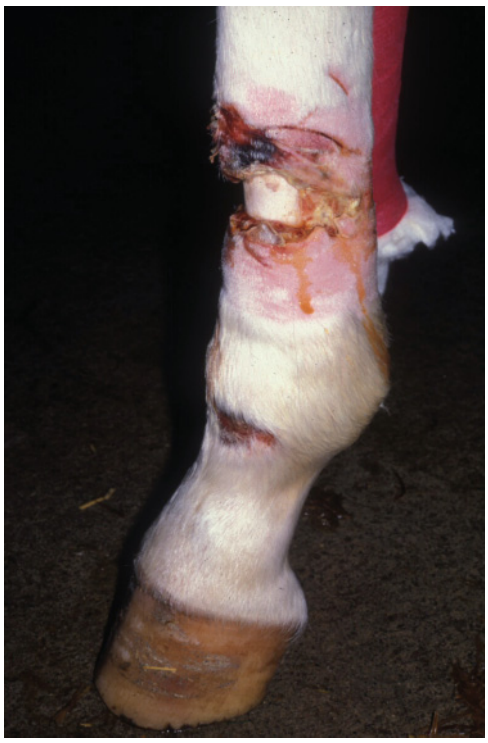

**Figure 10.11.** The dorsal metatarsus is a common location for extensor tendon lacerations, and many injuries also involve the metatarsus.

- Immobilization of limbs with lacerated DDFT and SDFT to support the fetlock and prevent further injury should be performed similar to those with fractures.

### *Diagnosis*

- Often, a tentative diagnosis can be made based on the location of the wound and the characteristic gait abnormality.
- Digital palpation of the wound can often reveal the extent of the laceration to the tendons (Figure 10.14).
- Radiography should be performed to detect secondary bone abnormalities such as fractured splint bones.
- Ultrasound examination may be used to define partial tears and possibly locate foreign material.

### *Treatment*

- Routine first aid management of all wounds is recommended initially.

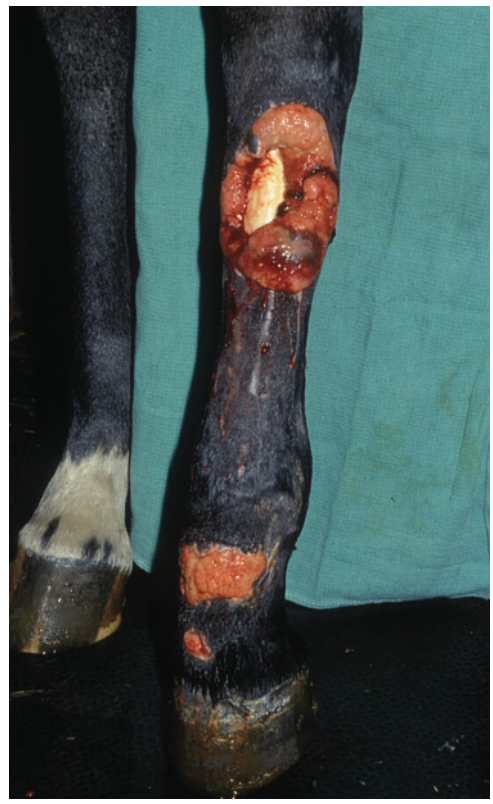

**Figure 10.12.** Avulsion injuries on the dorsal aspect of the metacarpus/metatarsus, resulting in dorsal knuckling of the fetlock. Note the wound on the dorsal fetlock and the sequestrum in the proximal MTIII.

- Most extensor tendon lacerations cannot be closed due to the severity of trauma and are treated with second intention healing. However, suturing the extensor tendons can be performed if the injury is acute and soft tissue trauma is minimal.
- Weight-bearing can be permitted for extensor tendon lacerations but external coaptation may be needed to prevent dorsal knuckling of the fetlock (Figure 10.12).
- Removal of sequestra from the dorsal cannon bone may be necessary in severe avulsion injuries (Figure 10.12).
- The optimal treatment of flexor tendon lacerations is wound debridement and closure, tendon apposition with suture, and cast application for a minimum of four weeks (Figure 10.14).
- Double locking loop or the three-loop pulley suture patterns are recommended for repair of the flexor tendons.
- If the tendon ends cannot be re-apposed, wound debridement and closure can still be

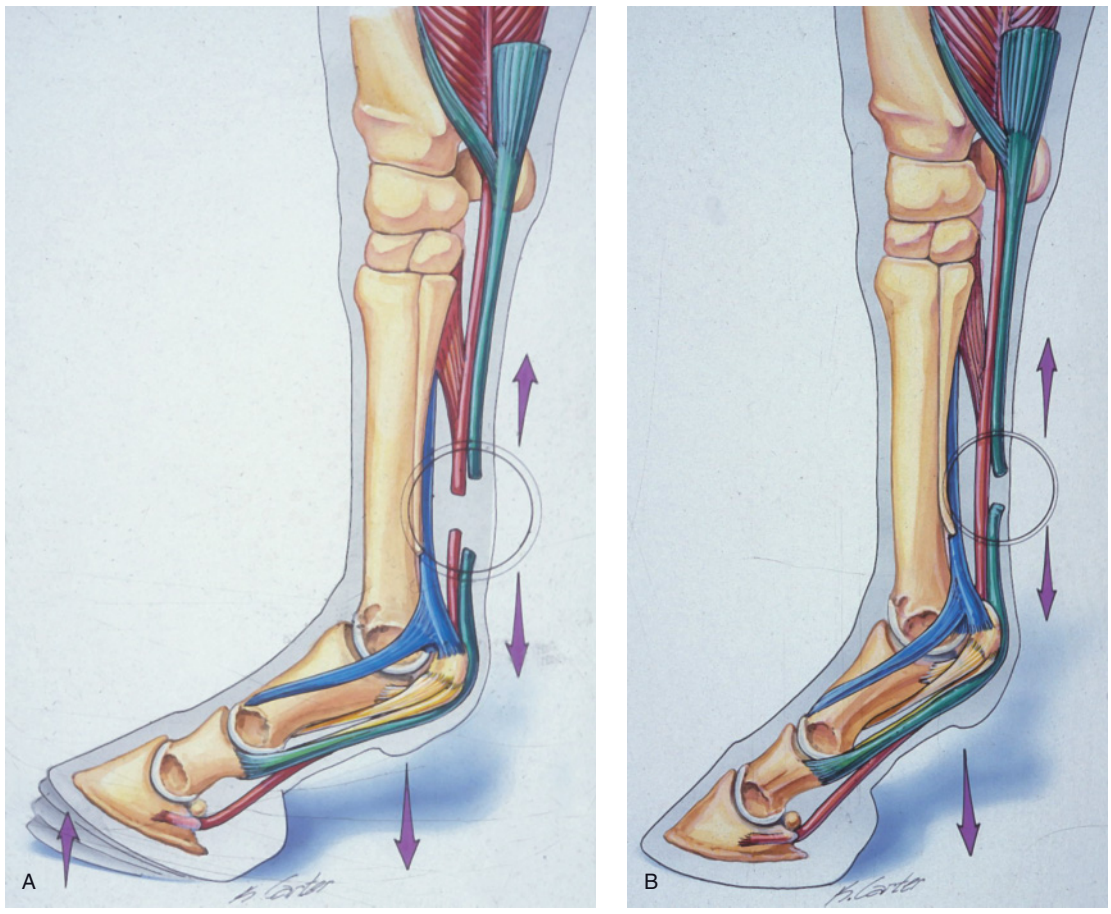


**Figure 10.13.** Illustration of the biomechanical effects of lacerations of both the SDFT and DDFT (A) and just the SDFT (B) in horses with flexor tendon lacerations.

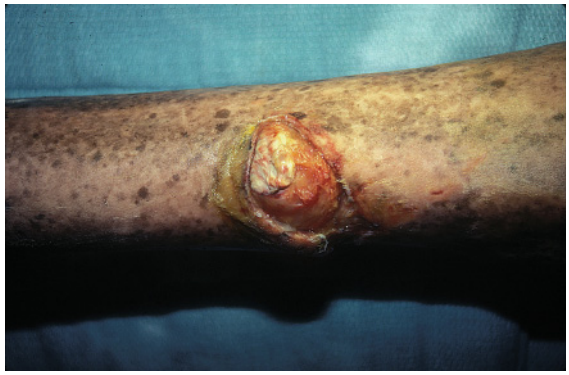

**Figure 10.14.** Typical location and appearance of a laceration of the flexor tendons in the mid-metatarsal region. The SDFT can be seen outside the wound and digital palpation revealed a lacerated DDFT as well.

performed because the tendons will heal with gap healing provided they are adequately immobilized with a half-limb cast.

- If the digital sheath is concurrently involved with the laceration, tenoscopic exploratory of the sheath or lavage of the sheath should be performed. Closure of the tendon laceration and sheath are indicated in most cases unless gross contamination is present.

## *Prognosis*

- Horses with extensor tendon lacerations have a good to excellent prognosis (more than 70%) for return to full function with conservative management.
- Horses with flexor tendon lacerations have a good prognosis for pasture or breeding

soundness and a guarded to fair prognosis for athletic soundness.
- Complications include restrictive fibrosis, infection, delayed healing, and chronic lameness.

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# INDEX




*Manual of Equine Lameness*, First Edition. Edited by Gary Baxter.