Part I: Operative Orthopedics of the Fetlock Joint of the Horse: Traumatic and Developmental Diseases of the Equine Fetlock Joint

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The invitation to present the Frank J. Milne State-of-the-Art Lecture is a special degree of flattery for one’s career. The flattery comes with a degree of responsibility to present the current state of practice and, to a certain extent, challenge doctrine and lay out theories of practice that result from one’s years of practice in a specialty area. The lecture should, therefore, enjoy the possibility of moving the “state of the art” forward. In areas where the author of this manuscript has a view of pathology that varies from the current concepts, the views will be presented as theories based on years of observation and, where possible, controlled studies. The object is to lay them out for examination and challenge or refutation by future practitioners of this specialty. I want to thank the members of the American Association of Equine Practitioners for this opportunity. Author’s address: Rood and Riddle Equine Hospital, PO Box 12070, Lexington, Kentucky 40580; e-mail: lbramlage@roodandriddle.com. © 2009 AAEP.

“An orthopedic surgeon must be able to think like a bone, and feel like a joint.”—Anonymous

1. Introduction

The fetlock joint is, arguably, the joint that makes a horse a horse. Its unique anatomy and physiology allow the high-speed, medium-distance activity that has lead to the unique place for the horse in society, historically and currently. Its evolution allowed the horse to become a single-digit quadruped. The fetlock is a joint, a shock absorber, an energy storage system, and a stabilizer of the distal limb. It is constructed like a suspension bridge with structural members incapable of supporting its loads until the appropriate ligament tenses and supports the bone. It is the most fascinating of the complex of joints that allows a horse to move at high speeds and over rough terrain with little conscious concern. Because of its complexity, it is vulnerable to a variety of traumatic and developmental problems that are the veterinarian’s purview.

This paper will discuss the traumatic and developmental diseases of the fetlock joint and concentrate on the physical causes and surgical treatment
of diseases of the fetlock joint. Space and time limitations will preclude details of surgical technique for most procedures. Concepts will be presented and surgical techniques will be referenced where possible.

2. Defining the Problem

Short of a fracture needing the reconstruction of an articular surface, the majority of clinical problems in the fetlock joint present a risk for secondary degenerative arthritis as their sequelae. To reach the point of a decision to treat the fetlock joint surgically, we must be able to understand the concept of progressive degeneration, to assess the current status of the joint, and to understand when the surgeon can make a difference.

Primary degenerative arthritis is rare in the horse. What we see in the horse is degenerative arthritis secondary to an insult of traumatic or developmental origin. The overwhelming volume of literature in recent years has concentrated on the biology of the degenerative arthritis cycle. This paper will concentrate on debris in the joint, the resultant physical damage, and the biologic responses that it initiates.

The physical debris liberated into the joint as part of the original insult and by the joint’s attempt to heal mediates most of the ongoing damage after a traumatic or developmental osteochondral fragment. If the cause is developmental or traumatic, the secondary reaction of the joint is remarkably similar. So, it follows that the treatment will be similar as well. When one examines the reaction of the joint to a traumatic disease such as an osteochondral fragment or to a developmental abnormality such as an osteochondritis dessicans (OCD), the place where the two inciting causes are similar is in the shedding of debris into the joint.

Debris is physical (particulate) and biologic (inflammatory cytokines/mediators of inflammation that are liberated by the chondrocytes and synoviocytes, and inflammatory cells recruited to the joint by response to the physical insult). As the debris is liberated the joint responds to the insult. Injection of cartilage particles experimentally can create synovitis and mechanical injury to cartilage, but bone debris is likely more important and more damaging. Removal of the inciting lesion and its associated debris allows the joint to return normal if the secondary arthritis has not reached the critical threshold of self-perpetuating degeneration.

Degenerative joint disease (DJD) has sometimes erroneously become a “catch all” for any change seen on radiographs. Most joint inflammation seen in the equine athlete is not degenerative, especially in young horses; it is the response to traumatic or developmental insult. The response to trauma or a developmental OCD lesion is often reversible early in the course by removing the inciting cause, and therefore, it is not degenerative.

Degenerative arthritis certainly will occur if the inciting cause is left unattended and the destruction progresses to the point that it will proceed unabated even if the cause is removed. But, fortunately, lameness and decreased performance usually occur in horses in strenuous activities before the joint reaches this state, providing the opportunity for diagnosis and treatment of the primary disease before it becomes irreversible.

The clinical signs can be subtle, especially if the level of the horse’s activity is moderate. In the author’s experience, the attending veterinarian for high-level equine athletes has an advantage in that horses in heavy work will show more significant signs than horses in light activity, although the joint’s response is the same in total. High-level activity elevates the rate of debris shedding and magnifies the joint response, but hypothetically, the amount of debris shedding seems to be a direct product of the severity of the problem and the activity level of the horse. Horses with small problems or low levels of activity may shed debris at a rate that causes a subtle joint response, creating lameness that remains subclinical. But low-level clinical signs are indications for concern and eventually, can cause acute significant lameness, because, in the author’s opinion, when debris shedding approaches a critical mass, the clinical damage will be similar.

The need for treatment is obvious with acute damage such as a dorso-medial proximal phalanx (P-I) chip fracture of the fetlock joint in a racehorse, because the debris shedding is acute, the damage to the joint is rapid, and it causes lameness early in the course of the disease. But, a show horse or a pleasure horse with a similar lesion may show only mild signs, such as effusion and pain on flexion, but no lameness. Although the debris shedding may be slower in less strenuous activity, clinical observation suggests slower shedding causes lameness and causes joint damage after a comparable amount of debris is shed.

The need for treatment in the racehorse is obvious when it cannot perform because of lameness. The need for treatment for a similar injury in the horse of less strenuous activity may be less clear when debris shedding causes only the synovial effusion, but the horse can still perform. The permanent damage accumulating in the joint is less obvious, because the rate of damage is slow but the end effect is no less severe. After the same amount of debris is liberated, although it takes a much longer time, the end result is equally severe.

The determination of the need for treatment in the pleasure or working horse with subclinical disease is more difficult. Not all horses’ careers are valuable enough to warrant surgical intervention for all conditions. Simply the fact that the horse can perform is not an indication that the fragment is innocuous, especially if the horse requires ongoing intra-articular therapy to mitigate the signs. Often a fragment will be tolerable for a prolonged period of
time, especially with intra-articular medication. If the horse is easily replaceable or its use can be easily changed, then the decision may be to tolerate the problem. But, if it is unacceptable to risk having the horse’s disease progress and potentially shorten the athletic career, then fragment removal is the best treatment. So, each diagnosis of potential damaging traumatic fragmentation or developmental bone malformation must be evaluated in light of the long-term effect on the horse’s career and performance level. The decision to remove or to tolerate the presence of a fragment needs to be an active one and not simply a matter of assuming that minimal clinical lameness means no harm.

Economic realities must figure into the evaluation. Early surgical removal requires an upfront expense but with the good prospect of permanently solving the disease.\(^5\)–\(^11\) Forgoing the surgical treatment of the fragment and treating it medically, especially with symptom-modifying medications, will be progressively expensive over time and will ameliorate the signs but not stop the progression of the disease. Eventually, the debris shedding can damage the hyaline cartilage to the degree that it will disable the joint. This can end or denigrate the horse’s career after a few years, necessitating replacement of the horse before the end of the horse’s natural career span. Medication may be able to relieve the clinical signs or interrupt the biologic processes of joint inflammation by negating the biologic debris, (inflammatory cytokines/mediators of inflammation that are liberated by the chondrocytes and inflammatory cells recruited to the joint by response to the physical insult) but it will not stop the physical debris shedding, which continually takes its toll on the hyaline cartilage. Owners and some veterinarians mistakenly assume that the mitigation of clinical signs is a cure, especially in high-class performance horses that can afford frequent intra-articular therapy. They assume that frequent joint injections are “simple maintenance.” Then, when the “maintenance” no longer “maintains” the joint, they are confused about the lack of response and consider surgery as a treatment. The worst choice that can be made is to medically treat a horse with a surgically resolvable disease, allow the joint to suffer irreversible damage, and then attempt to surgically remove the cause. This provides the worst of both possibilities: the most possible expense and the shortest possible career. The irreversible change in a joint is the loss of the structural aspect of the hyaline cartilage, primarily the collagen. The chondrocyte population is labile but has some reproductive capability if the insult is not overwhelming.\(^3\)–\(^12\) The physiologic component of the cartilage maintained by the chondrocytes, the proteoglycan, is replaceable; in fact, it is in a constant state of flux, normally in equilibrium, with production equaling destruction.\(^3\) In inflammatory conditions, the production may be slowed or the destruction may be accelerated, tilting the balance to a deficit in proteoglycan balance, but simple proteoglycan depletion is reversible.

The lubricating function of the proteoglycan protects the collagen in the normal joint. In the diseased joint, the proteoglycan becomes depleted, and the collagen becomes exposed and vulnerable. So, any measure that preserves proteoglycan function, its lubrication of the cartilage surface, and therefore, its protection of the collagen is likely worthwhile to consider for the prevention of collagen degeneration and degenerative joint disease/degenerative arthritis. This is where the disease-modifying medications have an important potential for benefit and again caution should be exercised with use of medication that is only symptom-modifying.\(^13\) This is also where surgery can shine if the cause is removable and the rest of the joint is still normal.

Extensive or prolonged loss of proteoglycan combined with use of the joint makes the cartilage collagen architecture so vulnerable to wear that it allows permanent damage in short order in the form of destruction of the collagen “backbone” of cartilage.\(^6\)–\(^14\) Some medical treatment is aimed principally modifying the proteoglycan balance in the joint.\(^13\) No treatment, medical or surgical, has yet shown the ability to produce quality collagen replacement of the type of collagen architecture that is present in hyaline cartilage.\(^5\)–\(^12\)–\(^14\)

The subchondral bone architecture is very important in its support of the collagen in the correct configuration for joint function.\(^15\)–\(^16\) The bone determines the anatomy and keeps the cartilage in the correct location to articulate with its opposing joint surface. The calcified cartilage on the surface of the bone also serves as the anchoring point for the collagen of the cartilage. Bone can be replaced, but it is very difficult, nearly impossible, to replace the calcified cartilage/subchondral bone contour and its perfectly adapted anatomy of the normal joint.\(^17\)

Therefore, the collagen’s role in normal joint function requires the anatomy of the bone to be preserved.

Collagen can be created and proteoglycan can be replaced, but the body is unable to recreate the arching configuration of the collagen architecture that anchors hyaline cartilage to the bone.\(^15\)–\(^17\)–\(^18\) It is this architecture that is required to reach the functionality of hyaline cartilage, and it is this architecture that is missing from fibro-cartilage that is formed after hyaline cartilage loss. “Hyaline-like” replacement fibro-cartilage has proteoglycan content and collagen but not the collagen architecture that it takes to stand up to exercise in the horse. Therefore, it is at this point impossible to recreate normalcy in a damaged joint surface in an adult horse by any means. The lack of a solution for degeneration places a premium on prevention.

In degenerative arthritis, the collagen is worn from the joint surface into the middle and deep layers of cartilage because of failure of the lubrica-
tion/protection mechanism and the physical effects of the debris. The superficial layer of the cartilage containing the tops of the arches of collagen is lost first (Fig. 1). This exposes the collagen, initiating the process we recognize as fibrillation of the cartilage. With fibrillation, the low-friction joint surface is converted into a higher friction joint surface, lubrication becomes more difficult, proteoglycan loss accelerates, and physical wear overwhelms the cartilage’s resistance to wear, resulting in progressive loss of joint function. This process can be accelerated or mitigated with exogenous joint therapy, systemic or local. Texts and manuscripts are voluminous on this subject in the literature.19

Hyaline cartilage is resistant to repair, because it has no blood supply, and it must be replaced by fibro-cartilage; however, fibro-cartilage does not have the collagen arches that provide the resistance to joint loading and wear unique to hyaline cartilage. Fibro-cartilage has an irregular haphazard collagen arrangement with cross linking of the fibrocartilage. Further, the anchorage to the bone is believed to be different than native cartilage. Fibrocartilage can be compared to covering a joint surface defect with a scar, which it does effectively, but may not participate in joint function to a similar extent as native cartilage. Clinically in the horse and in some experimental studies, fibro-cartilage functions well to cover a defect in the joint surface that penetrates the subchondral bone, but does not resist wear effectively and under heavy loading, especially in shear, fails and detaches from the bone it covers.17,18,20

Bone can heal but has difficulty restoring joint-surface architecture. Unless bone healing restores the innate joint anatomy perfectly, the repaired joint surface is suboptimal for weight bearing, because the fibro-cartilage covering the injured bone does not reach perfect articulation with the opposing joint surface.

Taken together, the repair process for bone and cartilage of an injured joint surface is poor and achieves success as a scar but not as a functional replacement.21 This can result in prevention of inflammation in some cases, but the scar cartilage and deranged bone anatomy is not completely thought to restore normal joint function to the injured joint.

3. What Is Surgically Possible?

Surgical reconstruction of a joint surface whenever instability is present and preservation of as much articular surface as possible is a prime indication for surgical treatment. But, when a joint is injured, most treatments consist of removing the damaged area to negate the ongoing effects of the injury. The joint resumes function using the preserved normal hyaline cartilage and to some extent the repaired surface. One cannot rely on a significant area of repaired joint surface to function for high level athletic activity.

The most theoretically promising approaches are surgical techniques such as microfracture resurfacing, and cartilage augmentation and implantation systems.22–24 Unfortunately they have yet to achieve routine clinical success in the horse.23,24 Any injury over 1 cm in diameter, even in a person, has difficulty resisting the loading asked of a repaired joint surface, even with the added benefit of microfracturing (Fig. 2).25–27 In the author’s opinion, microfracturing injured joint surfaces on the dorsal condyles of the cannon bone or on the articu-
lar surface of the sesamoid bones has not aided in recovery from the damage to those areas that badly need a treatment solution. These are such heavily loaded articular surfaces that asking fibro-cartilage to perform functionally is not likely feasible.6

Injuries that violate the subchondral bone plate can recreate a superior fibro-cartilage, because the fibro-cartilage can anchor on the exposed cancellous bone effectively.15 However, a deficit in the bone rarely fills to the degree that the bone becomes congruent for weight bearing; so, the cartilage is anchored well and does not fail, because it is in a protected environment below the normal articular surface and overlying cancellous bone. But, it does not participate in the function of the joint; therefore, it is of a high quality histologically but is of little use functionally to the horse.

Injuries that remain weight bearing, even if microfractured, seldom maintain the fibro-cartilage in the face of serious weight-bearing exercise, because the collagen anchorage is insufficient to resist the forces of weight application and is quickly sheared from the subchondral bone. Removing the subchondral bone plate totally to expose cancellous bone helps the collagen anchor to the bone; however, it does not achieve function, because the normal architecture is destroyed to the point that joint function cannot be preserved. Forage of damaged subchondral bone with multiple drill holes improves anchorage, but does not produce enough functional cartilage to restore joint function. It is most likely impractical over a large area in the horse, because prolonged protection from loading while a functional fibro-cartilage forms is not possible.17,18

In the author’s opinion, grafting of cartilage, chondrocytes, or stem cells faces the same difficulty in establishing an attachment to the subchondral bone as does granulation tissue trying to form fibro-cartilage. Mosaic-plasty (taking small plugs of cartilage and bone from a remote site and inserting them in a denuded area) has been tried, but lack of equivalent donor sites, stabilization of the plugs, and reconstruction of a functional anatomy without damage to the transplanted grafts have all limited use.58,59 Prosthetic replacements in horses, as in people, have little chance to survive the biomechanics of the much larger horse and recreate an athlete in the horse or in fact, in people.

Biologic therapy such as stem cells and platelet-rich plasma is being used and shows promise in aiding joint function, but it is currently not able to substantially replace articular cartilage during mechanical loading with the loads that are seen in the horse.60

So, surgical replacement or augmentation of an injured joint surface is to this point not a reality. If injured joint surfaces can be reconstructed using internal fixation to reconstitute the original anatomy with the original cartilage surface intact or nearly so, surgery shines.

4. The Role of Surgery

The primary role of surgery is to stop debris shedding and restore stability. This is not to disparage the role that surgery plays in the approach to arthritis. It emphasizes the role of prevention of arthritis, which is surgery’s strength, rather than the treatment of arthritis, which is its weakness. With a few exceptions, surgery is primarily a bone treatment in the horse. Surgery stabilizes the reconstructable joint surfaces and removes the damaged bone that would shed debris and further injure the joint. The joint must survive on whatever cartilage can be preserved by reconstruction or by prevention of the degeneration mediated by debris shedding and the subsequent cascade of inflammation that leads to degeneration (Fig. 3).

5. The Role of Medication in a Joint

Symptom-modifying medications can decrease or resolve the clinical signs that accompany arthritis and can be quite useful at buying time for the joint to heal itself, but they can be abused if they are used to cover up a physical problem that continues to damage the joint.4,30 They should be used with a thorough understanding of their benefits as well as disadvantages to the joint. When used to simply cover up a physical injury that results from shedding debris, obtunding the inflammatory response will eventually allow the physical injury to disable the joint (Fig. 4). Disease-modifying medications can promote proteoglycan anabolism, prevent destruction, and to some extent, negate biologic debris that damages the joint, but they cannot stop physical debris shedding within the joint that results from unstable bone.13 It is the physical debris that
is shed from traumatic and developmental lesions that does the ongoing permanent damage to the hyaline cartilage and therefore, to the joint.5

To understand the reason for the ongoing damage a traumatic fragment of bone or developmental fragment of cartilage and bone causes on normal joint surfaces, one must review the way bone heals in the unstable situation and understand the concept of continual debris shedding. To conceptualize the unstable fragment of bone as a “stone in your shoe” far underestimates the pathology done by an unstable fragment.

6. Gap-Strain Bone Healing and Its Effect on the Joint

Bone has the capability to exactly reconstruct itself after fracture injury. It can do this through two primary mechanisms. If it is stable, bone can fill a gap with new bone (primary bone healing) and remodel to re-establish pre-injury anatomy. This will normally occur only in the case of a fracture that is non-displaced or has been surgically stabilized. In unstable bone healing, the bone goes through a series of steps to attempt to fill the gap between the bone ends with other tissue, which eventually converts to bone (secondary bone healing).31–34

The relative motion of the bones in relation to each other dictates the kind of tissue that forms between the fracture ends in secondary bone healing. This “strain” between the bone ends is defined by the “change in length of the gap (motion) divided by the unit length (the gap).”30–33 In a fracture, the amount of strain at the fracture gap can be restated as “the amount of motion divided by the size of the gap.”31–34

Bone is a very stiff and strong structure and as such, has a very low strain tolerance until rupture, ~4% in cortical bone and ~5% in membranous bone. In stable situations, motion is not a factor, because the motion and therefore, the strain is under 4% and the gap can easily fill with replacement bone.31–34

In unstable situations, the strain is greater than the bone can tolerate and therefore, newly formed bone simply ruptures. Cartilage has a 15% strain tolerance, so it can tolerate 3–4 times more motion than bone; granulation tissue or immature fibrous tissue has a 100% strain tolerance.32,33 Therefore, with high motion between the two ends of a fractured bone, the bone must start by forming granulation tissue, because it will tolerate the highest strain. As more granulation tissue forms and the tissue holding the two ends of the bone together enlarges, it becomes stiffer, thereby reducing the strain between the bone ends. The reduced strain allows less strain-tolerant tissue such as cartilage to then form, and the mass, known as callus, gradually creates a union by progressively enlarging and stiffening until it becomes stiff enough to form bone.34

We recognize this process in most fracture healing by the expanding callus that eventually stabilizes and unites a fractured bone, which then reorganizes and replaces the callus with the stronger, smaller, and more organized bone. Because this process of secondary bone healing by callus is controlled biomechanically and dependent on the local biomechanics of the gap-strain healing process, which is a ratio, the gap size and motion are both important. The surgeon can facilitate healing by reducing the strain by fracture reduction, immobilization, and restriction of exercise, and by aligning the bone ends to a normal anatomical configuration, because the ends of the bone must be in close proximity to allow favorable local biomechanical conditions to unite the bone. Counterintuitively, a very small gap will have a higher strain than will a large gap (motion divided by a small number results in a higher strain than the same motion divided by a larger number). To reduce strain, the gap must enlarge before healing can occur. We recognize this process when unstable fractures get wider radiographically, increasing the gap, before they begin to heal (Fig. 5).

If the strain is >100%, it is too high to allow granulation tissue to unite the bone, and the granulation tissue simply covers the raw bone ends as it does in any raw bone surface in a joint. Increasing the size of the gap, as long as the bone ends are still approximated and aligned and the strain can be reduced below 100%, will aid fracture healing. Increasing the gap will reduce the strain, help to get it below the 100% limit of granulation tissue, and allow the callus process to begin, because fracture gap strain is a ratio (motion divided by the gap size). This is a locally mediated phenomenon stimulated by the local conditions surrounding the fracture. The radiographic widening results from softening of the bone by demineralization and disorganization of the bone, opening the space between the pieces of the fractured bone, reducing the strain, and allowing secondary bone healing to proceed. This is a
novel and effective way to promote fracture healing if the bone is not loaded at the time.

This same process occurs in the joint when a fragment is present. Healing between the parent bone and fragment is attempted, but this is a very high-strain environment because the motion is high in a joint and the gap is very small. Therefore, the parent bone actively softens and disorganizes to try to open the gap and allow healing to occur. But, if the bone is loaded during this process, it breaks up and is shed as it is demineralizing. Marked reduction of motion will sometimes accomplish healing, and a chip fracture will heal. Even cast immobilization is not enough to heal most fragments, and the secondary bone healing process persists and accelerates with resumption of exercise. Because of the loading with motion, this progressive destruction of the parent bone at the interface with the fragment or OCD results in tremendous amounts of bone matrix and mineral continually shedding into the joint. This constant debris shedding is the primary cause of the degeneration of a joint rather than the presence of the fragment and the “stone in your shoe” phenomenon. In most joints, fragment stability is not possible to achieve.

Continued attempts at healing unstable fragments occur if raw bone is exposed with resultant debris shedding progressively damaging the joint. In instances of strain in excess of what the bone healing process can tolerate, neutralization of raw bone interfaces will eventually occur by covering the bone surfaces with granulation tissue, and if it cannot reduce the strain to <100%, then it matures into fibro-cartilage by the cartilage healing process; this is the same scar cartilage that covers all raw bone surfaces in the joint. But, after fibro-cartilage forms, high-level use can erode this cartilage as it does in all high-load situations, exposing bone again, stimulating secondary bone healing, and reigniting the debris shedding. Clinically, the degree of success of fragment neutralization by fibro-cartilage parallels the degree of success of joint surface healing by fibro-cartilage. In the author’s experience, fibro-cartilage can be functional for less strenuous uses, but for increasing athletic activity, the probability of erosion of the fibro-cartilage and reintiation of debris shedding increases with the level of athletic activity.

If the fibro-cartilage fails when exercise resumes, the parent bone again responds by trying to increase the gap size to reduce the strain and heal the bone. The fragment can also respond in the same way if it retains sufficient blood supply, but this is not usually the case. The reaction can be identified radiographically as demineralization at the interface of a mobile fragment and can be seen with the arthroscope as the softening of the parent bone, which is making an attempt to heal the fragment. The most striking evidence, however, is on the normal cartilage surface where the debris gets interposed between the cartilage surfaces and physically scores the normal cartilage. These “score lines” destroy the superficial layer of articular cartilage where the pinnales of the arcades of collagen normally form the tough, gliding surface of the articular cartilage. This damage is permanent and tolerated to a point, after which enough loss of the superficial layer of cartilage has occurred that the cartilage can no longer protect itself and it erodes to bone.

The fact that chip fractures are not created by a single event and are the result of repetitive trauma amplifies this response. So, by the time the fragment separates radiographically, the bone demineralization and softening is well underway from attempted healing, and debris has already been shed into the joint. Since this is an inflammatory process it is hyper-vascular, though the fragment itself is often avascular. This process is an active response, which is minimally influenced by the size of the fragment. Even small fragments stimulate the softening by the parent bone over a large area, shedding debris and doing permanent damage to the joint. The size of the parent bone/fragment interface influences the amount of debris shed at one
time and the rate of damage to the normal joint. Mobile fragments stimulate the debris shedding into the joint, and the response to traumatic and developmental fragments becomes indistinguishable and ends with the same result.5

Many interpret the cause of pain and lameness from a chip fracture as direct physical injury to the joint surface. This is rarely the case. Cartilage has no nerve endings with which to generate pain. The pain originates in the subchondral bone due to the inflammation associated with attempts to heal the chip fracture, and from the interior of the joint as a consequence of the inflammation and disturbance in joint lubrication mechanisms caused by the inflammation.35,36 The healing process results in trauma from small particles of bone being shed as well as creating biologic debris, such as interleukin-1 β, which promotes the synovial inflammation and cartilage destruction that are the subject of much joint research and the target of all joint therapy.19

The primary traumatic injury with secondary debris shedding, if left unchecked, causes secondary degenerative arthritis through a two-pronged process. It is a biologic process that attacks the physiologic function of the proteoglycans and eventually, the collagen; it is also a physical process that directly abrades the hyaline cartilage surface, causing irreversible physical damage to the cartilage structure, especially the collagen, that becomes the degenerative arthritis that disables the athlete. The biologic process alone can eventually reach the degenerative threshold after the collagen has been degraded enzymatically or eroded physically because of a lack of adequate lubrication. But, the physical debris fragments stimulated by the healing response of the parent bone are the most direct path to degeneration through the physical damage and the secondary biologic response that they create. Debris is the primary enemy of the athletic horse’s joint.

7. Debris Management

Natural joint management of debris causes prioritization of joint resources. In the normal situation, the synovial fluid is low in protein, contains almost no fibrin, and is viscous because of the large amount of hyaluronan within the joint.4 One purpose of normal synovial fluid is to lubricate all joint functions and promote decreased friction associated with joint motion. It would be counterproductive, however, to lubricate debris within the joint and encourage it to repeatedly pass through the articulation. So, in an inflamed joint the lubricating capability of the joint is sacrificed temporarily for increases in fibrin and reduction of joint motion to give the joint an opportunity to clear the offending debris. When the insult is neutralized in the joint, the synovial fluid then returns to its normal highly lubricating capabilities. The need to remove debris is a common occurrence in a normal joint.

It is clear, therefore, that allowing persistent debris shedding and low-grade inflammation within the joint is counterproductive in the long term, because it compromises the joint’s ability to lubricate itself and maintain the articular cartilage in a wear-free state. The critical nature of maintaining the hyaline cartilage, because it cannot be reconstructed or replaced, points out the high priority that should be placed on removing even low-grade chronic insults and normalizing the homeostatic lubricating mechanism that preserves the hyaline cartilage.

When fragments of cartilage or bone are shed within the joint, the joint responds by producing fibrin. In the margins of the joint, this fibrin attaches to the synovial villi and catches the debris as it circulates in the joint. The fibrin facilitates the attachment of the debris to the synovial villi for elimination. The debris is, therefore, removed from bearing surfaces of the joint, preventing it from abrading the articular cartilage. The debris is sequestered in the cul-de-sacs of the joint. The tiny granulomatous nodules encasing the debris can often be seen arthroscopically (Fig. 6). The fibrin and small nodules stabilize the debris so that the cellular components, chiefly the neutrophils and macrophages, can remove it.

Debris removal necessarily requires release of potentially destructive enzymes and free-radical molecules as part of the process. When it occurs in short bursts with acute insults, the process rapidly clears the joint of debris and has little ill effect on the articular cartilage and joint environment. It is believed there is not enough wear during a transient period of inflammation to do significant harm, but if the balance of production and degradation is shifted toward depletion of proteoglycan long enough to af-

Fig. 6. This intra-operative picture shows several fragments that have been attached by fibrin to the synovial lining and are being encased by fibrous tissue (arrows). Note the vascular hypertrophy that is responding to the need for fragment removal from the joint cavity (arrowhead).
fect cartilage lubrication, destruction of the cartilage surface is initiated.

This rapid, efficient process of debris removal makes it possible for the joint to respond to an acute insult such as surgery. If the joint were not able to tolerate an acute insult, surgery would not be possible. If the debris shedding is continuous, however, then the continual enzyme liberation within the articular cavity affects not only the debris but also the lubrication mechanisms of the joint and the normal articular cartilage by progressive attrition of the proteoglycan content and loss of the normal lubrication mechanism. This makes the articular cartilage vulnerable because it shifts the proteoglycan equilibrium (production equals destruction) toward destruction, resulting in proteoglycan loss. This compromises the articular cartilage lubrication mechanism, allowing surface wear and fibrillation to occur. This causes normally physiologic loads to gradually erode the layers of the normal hyaline cartilage, a permanent, progressive, and irreversible degenerative change.

8. Surgical Treatment of Joint Injury

The role of surgery in the fetlock joint is simple: restore stability to facilitate primary bone healing or remove unstable fragments to prevent secondary bone healing. With few exceptions, surgery treats bone disease, whereas surgery cannot “treat” arthritis that is underway. If the arthritis is not beyond resolution, surgery can delay or mitigate the progression by removing the inciting cause. Removal of fragments of traumatic or developmental origin keeps the parent bone normal and prevents the debris that does most of the damage. The horse performs on the remaining normal joint surface. The restoration of damaged arthritic joint surfaces by surgical techniques remains a strongly pursued goal, but the “state of the art” at this time is that it does not work very well. The best chance for athletic function in a joint with a fragment is to prevent the primary injury from stimulating the process that degenerates the rest of the joint by removing the fragmentation and associated debris. Joints with major cartilage loss in critical areas remain career-damaging or career-ending challenges. So, surgery is a better prevention than a cure for arthritis with joint injuries in the horse.

9. Surgical Treatment: Do We Use It Enough?

Surgical treatment of joint fragmentation is rapid and effective in stopping debris being shed into the joint. The removal of unstable bone and its associated debris preserves normal joint surface that will be lost if the parent bone attempts to heal an unstable fragment. The process of the parent bone trying to reattach and heal an unstable fragment is often worse than the primary disease. The extreme range of motion of the fetlock joint makes it even more vulnerable to this type of damage than most other joints. Physical debris tends to disseminate throughout the articular surface, scoring the cartilage surface until it sequesters in the cul-de-sacs of the joint (Fig. 7).

Any opportunity to interrupt this debris-shedding process has a positive effect on a horse’s joint. Surgery should be used more often and earlier than we often elect to treat articular injury before it does irreversible damage.

The strategy in treating traumatic and developmental joint injury is to negate the inciting and perpetuating cause, usually an unstable or malformed area of joint surface, and preserve every bit of normal joint architecture and hyaline cartilage possible. Surgical stabilization or surgical removal of the problem, whenever possible, is the most definitive treatment and results in the fastest, best resolution of the problem. In most instances, modifying the clinical signs medically, without eliminating the perpetuating physical cause, risks permanent damage.

10. Examination and Treatment of Traumatic and Developmental Diseases of the Fetlock Joint

Categorizing and Staging Fetlock Joint Inflammation and Its Treatment

It is useful during the examination of an injured joint to categorize the disease. When one evaluates the radiographs of a joint, it is imperative to assess the health of the joint in light of whether or not the perpetuating cause can be removed and the joint preserved. Localized reaction to an insult (focal lymphoid proliferation in an area of fragmentation or fracture and excess synovial fluid without significant fibrous thickening) carries a better prognosis than a generalized reaction (marginal spurring at multiple sites, thinning of articular cartilage, and fibrous thickening). The localized reaction is an indication of the parent bone trying to stabilize the
fragment. Generalized reaction is an indication of chronic disease and irreversible injury from a chronic insult. Localized reaction is often amenable to resolution surgically. Generalized reaction is indicative of permanent change.

Radiographic Markers of Previous Inflammation in the Fetlock Joint

There are two easily discernable radiographic markers of previous severe inflammation of the fetlock joint. Even when previous severe inflammation is resolved, if either one of these markers is present, it indicates that the horse’s athletic career has been compromised. The first is sesamoiditis, indicated by the enlargement and change in shape of the vascular canals of the sesamoid bone. This is a marker for injury to the suspensory ligament insertion and occurs after serious inflammation of the sesamoid bone, usually because of trauma, and possibly, previous fracture of the sesamoid bone. This enlargement and change in shape of the vascular canals is an indication of reduced career performance.

The second marker is supracondylar lysis, which results from chronic inflammation of the synovial lining on the palmar distal aspect of the cannon bone above the articular surface. This radiographic finding is a non-specific indicator of previous severe inflammation of any kind within the fetlock joint. If supracondylar lysis is present, it indicates that some serious previous disease such as septic arthritis, fracture, or other severe trauma was previously present. The presence of supracondylar lysis has been documented as a marker of decreased performance in the horse. However, it is likely not the supracondylar lysis itself that is the problem but rather the disease that previously created the severe inflammation and supracondylar lysis that is of concern. Supracondylar lysis indicates the need for investigation and assessment of the previous severe disease and its effect on the fetlock joint.

Forelimb Versus Hindlimb Fetlock Joint Anatomic Differences

The osseous anatomy of the forelimb and hindlimb fetlock joints is similar; but the ligamentous anatomy is different. The fetlock joint moves in only one plane—sagittal. Motions in other planes play a role in many fetlock joint injuries. The ligamentous support in the forelimb is placed more dorsal, preventing hyperflexion that separates the dorsal articular margins and providing much more resistance to torsion forces. The ligamentous support in the hindlimb is more plantarly placed on the medial and lateral aspects of the fetlock joint, which allows the dorsal articular margin to hinge open in hyperflexion. The collateral ligaments provide much less resistance to torsion, especially in the non—weight-bearing position (Fig. 8).

During weight bearing, the sagittal ridge of the cannon bone and the sagittal groove of the first phalanx and intersesamoidean ligament provide the primary resistance to torsion. When torsion is applied in the partially weight-bearing or non—weight-bearing positions, it is the ligamentous support that resists the torsion and determines where an injury will occur. The different anatomy makes this different in the forelimb and the hindlimb. Torsion damage is much more common in the juvenile animal than the adult, because the bone is still growing and failure at sites of bone growth is more common because of their reduced strength.

Injuries in torsion occur primarily in the non—weighted position. In the forelimb, in contrast to the hindlimb, the collateral ligaments maintain the articular surfaces in apposition when the first phalanx is twisted. This causes the sagittal ridge to
remain firmly apposed to the dorso-medial and dorso-lateral eminences of the first phalanx when the fetlock joint is flexed, and disturbances in bone formation occur commonly in the distal sagittal ridge in the forelimb where the resistance to the torsion occurs. The medial or lateral dorsal eminences of the first phalanx can also suffer damage in the resistance to torsion. This damage results in the occasional chip fracture of an eminence as a result of this trauma. The resistance of the sagittal ridge and grooves to torsion provides some protection to the palmar ligamentous attachments of the fetlock joint in the forelimb, so although palmar first phalanx avulsion fragmentation at the attachments of the distal sesamoidean ligaments occurs, it is not nearly as frequent as in the hindlimb.

In the hindlimb, the caudally placed collateral ligaments allow the dorsal aspect of the joint to subluxate in the non—weight-bearing position when torsion is applied. As this occurs, fragments are commonly created from the axial aspect of the medial or occasionally, the lateral dorsal eminence of the first phalanx. The distal sagittal ridge is rarely damaged in the hindlimb because of this disarticulation. The disarticulation allows the torsion to proceed until the caudal ligamentous support resists. As it resists in the growing horse, the site of growth is the weakest link, and fragments of growing bone are pulled free at the ligamentous attachments of the distal sesamoidean ligaments and collateral ligaments, resulting in planter P-I fragments much more commonly in the hind fetlock than in the fore fetlock. These anatomic differences result in marked differences in incidence of the various juvenile bone injuries in the forelimbs and hindlimbs of the horse.

11. Serous Arthritis

Distention of the joint capsule by synovial fluid without radiographic signs was formerly classified as a disease category in and of itself. Newer diagnostic methods including arthroscopic surgery, magnetic resonance imaging (MRI), and digital radiography as well as better targeted radiographic positioning and improved understanding of the disease processes now cast doubt on whether or not this is a real condition. It is likely that almost all cases of serous arthritis have an underlying pathologic injury. It may be that the pathologic injury is simply an insult to the soft tissues or a comparable insult to the bone that is below the level of radiographic detection, but it is likely there is a pathologic cause for all cases of synovial distention.

The distention of the joint capsule with poor-quality synovial fluid and its concomitant sacrifice of the lubricating mechanism in the interest of the more urgent need for removal or healing of the offending insult results in synovial distention with a lack of radiographic findings. The excess synovial fluid production is a response to some thing. Chronic overuse can be a cause of serous arthritis with minimal radiographic changes as the articular cartilage begins to suffer superficial loss of cartilage structure with fibrillation, but this is usually present in multiple or paired joints with a minimally progressive course. This is a prime indication for disease-modifying medical therapy to support the joint function but denotes caution with symptom-modifying medication that could contribute to the progression of the problem.

Acute onset single-joint serous arthritis normally has a pathologic cause. Current diagnostic imaging capability is identifying a cause for most of these cases. Many of these are lesser versions of well-known injuries, and some can benefit from surgical or medical treatment.

12. Diseases of the Distal McIII/MtIII First Phalanx Articulation

Osselets (Little Bones) or Capsular Insertion Inflammation

Osselets were formerly, and are occasionally still, an occupational hazard of most horses that work at power or speed. If horses race or work long enough, their fetlock joints enlarge because of a myriad of reasons causing inflammation. These causes are now generally separated and treated as individual diseases. The basic pathology of the condition, hypertrophic proliferation of the dorsal capsular insertions, is caused by any cause of inflammation chronically present in the dorsal fetlock joint. Most commonly incriminated are hyperextension and the impact trauma of the dorsal aspect of the hyperextended first phalanx on the dorsal cannon bone. This occurs primarily in the forelimb. Chronic damage and the resultant inflammation cause softening of the bone where the capsule inserts both on the first phalanx and on the distal cannon bone. Fragmentation will also contribute to the softening of the bone through its healing response to fragments. As the “Sharpey’s Fibers” of the capsular insertion begin to lose their anchorage in the softened bone, they begin to pull free. This results in inflammation of the fetlock joint capsule or capsulitis. The bone proliferates and the capsule thickens in an attempt to reinforce the lost capsule insertion by hypertrophy, and the range of motion of the joint decreases.

The loss of capsule anchorage becomes a self-perpetuating disease with fetlock use pulling more fibers free from the bone; this causes more inflammation and further weakening of the fiber insertion, resulting in cyclic fiber damage. Fibrous tissue proliferation and thickening of the joint capsule reduces the flexibility of the capsule, which increases the extraction forces on the capsule fibers, because the fetlock joint capsule will no longer fold with flexion. This further pulls on the insertion because of the loss of flexibility, further contributing to the damage and fibrosis and perpetuating the cycle. Instead of pain-free motion, the stiffened joint capsule creates yet more thickening. Bone
and soft-tissue proliferation occur as the insult to the capsule insertion progresses until, in some horses, the stiffness becomes so prominent that joint flexion becomes almost impossible. Before routine radiography, if a disabled thickened fetlock joint was examined during a post-mortem examination, it would often reveal mineralization within the fibrosis; these “little bones” within the joint capsules gave rise to the name osselets (Fig. 9).

With the advent of digital radiography and more frequent use of arthroscopic surgery for early removal of dorsal P-I fragments, the most common cause of osselets is now routinely resolved early in the course of the disease, circumventing the bone softening and capsular thickening that is the result of chronic dorsal first phalanx fragmentation. However, if fibrosis of the joint capsule is promoted from any cause, it can become the self-perpetuating chronic capsular thickening. If dorsal fetlock joint capsular thickening occurs without radiographic changes of the bone, it is commonly referred to as a green osselet, but radiographic inflammation of the bones is common and commonly identified.

Fibrous capsular proliferation can be limited early by identifying and treating the primary disease such as dorsal P-I fragmentation. Combined with a period of reduced exercise, treatment will often prevent the marked capsular thickening that follows the chronic inflammation.

The classic treatment for osselets was therapeutic cautery, which historically seemed to be surprisingly useful in some instances. Some people feel that the principal treatment is the time away from training that accompanies the therapeutic cautery, but time alone seldom achieved a similar response. More recently, shockwave therapy is being used in the treatment of the capsular insertion inflammation, and capsular insertion inflammation seems to respond to the shockwave treatment, often allowing continuation of training. However, in my experience, continued training combined with the analgesia of the shockwave therapy sometimes encourages thickening that results from this progressive disease. The most successful approach to the prevention of permanent disability resulting from joint capsule thickening remains the elimination of the primary cause of the capsule inflammation, such as dorsal P-I fragmentation, when possible. Similar to degeneration of the articular cartilage, the progressive thickening of the dorsal joint capsule of the fetlock has a threshold beyond which it becomes self-perpetuating (Fig. 10). If capsular thickening has progressed to the point that it eliminates the flexibility of the dorsal joint capsule, removal of an inciting cause such as dorsal P-I fragmentation no longer halts the progression.

Exostosis of the Lateral Digital Extensor Tendon Insertion on P-I
An exostosis of the lateral digital extensor tendon will occasionally occur on the dorso-lateral proximal first phalanx just below the joint capsule insertion on the first phalanx where the lateral digital extensor inserts on the first phalanx. This is often confused...
with fetlock joint capsular insertion inflammation but is a distinctly different injury. The thickening with proliferation of the insertion of the lateral digital extensor tendon is prominent clinically and radiographically, but it occurs only laterally on the first phalanx in contrast to true capsular insertion inflammation that occurs across the entire dorsal first phalanx. Lateral digital extensor insertion inflammation is located immediately distal to the capsule of the fetlock joint rather than at the capsule insertion (Fig. 11).

Although the reaction is prominent and the enthesiophytes that are produced are impressive, the reaction is accompanied only by mild transient lameness, if there is any lameness at all. This proliferation is cosmetic only and does not require treatment. It has an excellent prognosis for future soundness and only requires attention if the cosmetic lump is a concern, in which case restriction of exercise until the inflammation subsides is recommended.

Dorsal P-I Chip Fractures in the Adult Horse

Chip fractures of the proximal aspect of the first phalanx on the dorsal articular margin are common in racing horses but are also found in horses of other uses. The biomechanics of creation in the adult horse are principally hyperextension of the fetlock joint with the first phalanx dorsal rim impacting on the cranial aspect of the metacarpus/metatarsus at the dorsal proximal fetlock joint margin. This impact is implicated in several pathologic conditions related to the hyperextension including ulceration of the dorsal proximal articular surface of the distal metacarpus/metatarsus, villonodular synovitis, and dorsal joint capsule thickening as well as the fragmentation and ulceration of the proximal aspect of the first phalanx. These conditions will occasionally occur in the hindlimb but are much more common in the forelimb where the most weight is born in the adult horse. Fractures occur more commonly on the medial eminence than the lateral eminence when uniaxial, but biaxial (medial and lateral to the sagittal groove) fragments are common. The fragmentation with cartilage ulceration occurs primarily on the abaxial margin of the eminence in the adult horse.

The fragments are created, in most instances, by repeated impact with eventual structural damage to the bone as a result of the repeated trauma, and
then separation of the bony fragments (Fig. 12). The impact damage as well as the presence of the bony fragments and the bone healing responses that they create result in debris shedding into the joint, inflammation, and pain (Fig. 13). Pain also results from the invasion of the chip fracture/inflammation complex into the sensitive joint capsule insertions on the dorsal aspect of the first phalanx if the disease process progresses deep enough into the bone of the dorsal margin of the first phalanx to affect the capsule insertion. During hyperextension and impact of the chip fracture on the distal cannon bone, the fragments are pushed forward and invade the joint capsule attachments if the fragments are large. Joint capsule fibers also become involved with the gradually softening of the bone of the proximal first phalanx by means of the gap-strain healing response that is created by all unstable fragments of bone. Softening of the bone then allows for avulsion of the capsule fibers in the process that creates the primary dorsal joint-capsule thickening disease described previously.

The simplest, fastest, and best treatment for dorsal P-I fragmentation is arthroscopic removal.\textsuperscript{11,50,52} This results in the best and fastest possible resolution of the pain and lameness caused by the dorsal P-I chip fractures. Given a prolonged period of rest, some horses will be able to heal a dorsal P-I chip fracture, but they usually heal with a more prominent dorsal margin on the first phalanx compared with normal, making reoccurrence of the impact of the first phalanx on the distal cannon bone even more likely than before injury.

Some horses are able to create a pseudo-arthrosis between the chip fracture and the parent bone by healing the raw bone surfaces with fibro-cartilage (Figs. 14 and 15), and for lower level exercise, this is a functional solution for the problem.\textsuperscript{53} But, in athletic horses, the fibro-cartilage rarely survives heavy use, and as the fibro-cartilage wears through and raw bone is again exposed, the debris shedding process is reinitiated. So, for most heavy-use horses, the only lasting solution to the problem is surgical removal of the fragmentation.

The clinical signs can be transiently mitigated with intra-articular medication, but this creates the dual toll on the interior of the joint of stopping the inflammatory response that neutralizes the debris being shed into the joint and compromising the articular cartilage’s ability to lubricate itself and prevent the wear that occurs on normal weight bearing.\textsuperscript{4} So, this is a transient rather than a long-term solution.

Surgical removal and debridement of the debris are effective and rapid ways to resolve the detrimental effects of a chip fracture on the dorsal aspect of the first phalanx. It is one of the most common and most successful surgeries in the fetlock joint and can return the joint to normal after surgery if treatment is undertaken early in the disease process.\textsuperscript{11,50}

**Fig. 14.** This radiograph shows a chronic P-I chip fracture (arrow) that has developed a fibrous pseudo-arthrosis between the chip fracture and the parent bone.

**Fig. 15.** This intra-operative view shows the fibro-cartilage interface (arrows) between the chip (top left) and the first phalanx (bottom); the distal cannon bone is on the right.

**Dorsal P-I Chip Fractures in the Juvenile Horse**

Chip fractures in the juvenile horse are more common in the hindlimb than they are in the forelimb. This seems to be caused by the biomechanically different forelimb and hindlimb action. In the hindlimb, the chip fracture in the juvenile horse is created by torsion force separating a fragment of growing bone from the parent bone at the interface of the first phalanx and the sagittal ridge of the distal cannon bone. The location of the chip fracture is more axial than in the adult, where the chip fracture most often occurs on the abaxial margin of the dorso-medial or dorso-lateral eminence.

The chip fracture will initially be quite small radiographically, but as the horse grows and the separated fragment fully ossifies, it is much larger than it appears initially. The fragment is initially separated from its vasculature but normally regains vasculature with continued growth and healing, and in many instances, it reattaches to the first phalanx.
However, it often alters the contour of the dorsal aspect of the first phalanx and creates a prominence that, on initiation of high-speed exercise, is impacted on the distal cannon bone and detaches again from the parent bone. Therefore, the prominence increases the risk for chip fracture and is extremely vulnerable to high-speed or heavy exercise. When it is again separated from the parent bone, it initiates all of the problems free fragments create within the joint.

There has been some debate as to whether or not these fragments need removal; however, examination of racing performance with these fragments in the joint show that they detrimentally alter the horses’ performances. Surgical removal is advisable for elimination of the debris shedding and the cycle of inflammatory degradation of the joint that occurs.

Proximal Dorsal Fetlock Articular Surface Erosion

During hyperextension, the proximal phalanx impacts the distal cannon bone just above the articular surface. It can have one of many results including chip fractures of the first phalanx, frontal plane fractures of the first phalanx, villonodular synovitis, dorsal joint capsule thickening, or simple erosion of the subchondral bone of the cannon bone proximal articular fetlock joint surface (Fig. 16). Often, combinations of these diseases will occur, but the erosion at the top of the articular surface that results from impact during hyperextension commonly accompanies dorsal fragmentation of the first phalanx.

This erosion of the cannon bone sheds debris; in the author’s experience, this by itself is not a major source of debris and subsequent inflammation, because there are rarely separated bone fragments that initiate a healing response to accompany the direct trauma. Primary treatment of the other conditions accompanying this surface erosion is likely to resolve the problem. So, as a disease condition, the cannon bone remodeling is mostly a sign of other articular pathology and radiographically appears to be related to the severity of the damage in the joint.

When found accompanying other injuries, the proximal articular surface of the metacarpal condyle erosion is debrided of loose fragmentation, but aggressive curettage and invasion of the joint capsule attachments just proximal to the erosion is counterproductive in returning the joint to soundness.

Dorsal Synovial-Pad Thickening (Villonodular Synovitis)

Villonodular synovitis, more correctly termed dorsal synovial-pad thickening, is an overdiagnosed condition. The description villonodular synovitis is an adaptation from human medicine that is slightly inaccurate but denotes pathologic enlargement of the normal synovial fold at the proximal aspect of the articular surface in the dorsal compartment of the fetlock joint. Hyperextension trauma accounts for many diseases in the dorsal compartment and is responsible for this disease. If the first phalanx traumatizes the synovial fold at the top of the articular surface between itself and the distal cannon bone during hyperextension, the synovial fold begins to swell and enlarge (Fig. 17). Calcification within the fold has been described.
The fold enlarges by the deposition of fibrin on its surface. As in any wound, fibrin gradually converts to granulation tissue and eventually fibrous tissue, thickening the fold in the process. If the synovial fold enlarges enough, it begins to encourage additional trauma by its size, and the process becomes cyclic with enlargement creating additional trauma and additional trauma creating enlargement. True equine villonodular synovitis involves enlargement of the villonodular pad alone with no thickening of the joint capsule and relatively little other disease within the dorsal compartment. When the synovial pad becomes large enough, a smooth erosion of the dorsal cortex occurs from the cyclic direct pressure on the bone.

The confusion in diagnosis comes from the synovial-fold enlargement, which occurs with any dorsal compartment pathology when all of the dorsal soft tissues of the dorsal fetlock joint begin to enlarge.\(^56\) This includes osselets, thickening of the dorsal joint capsule, dorsal P-I chip fractures, and proximal articular surface ulceration. Only when the joint is normal except for an enlarged synovial fold and there is no additional pathology is the term villonodular synovitis or synovial-pad thickening appropriate. If true villonodular synovitis is present, it can often be detected with ultrasound examination of the dorsal compartment of the joint; however, ultrasound is sometimes unable to separate thickening the villonodular pad or the joint capsule.\(^57\) The most diagnostic examination is a contrast arthrogram, which will most accurately show enlargement of the villonodular pad.

Ulceration of the top of the articular surface with no enlargement of the villonodular pad is commonly misdiagnosed as villonodular synovitis by veterinarians when there is no thickening of the dorsal synovial fold or simply thickening of the joint capsule. Hyperextension trauma of the dorsal aspect of the first phalanx impacting on the distal cannon bone creates traumatic ulceration and demineralization in a similar location but usually just distal to the true villonodular pad erosion above the articular surface, and the dorsal articular surface ulceration is normally not well delineated and is not smooth in contour as is the pressure-induced erosion of villonodular synovitis.

In the author’s experience, any disease of the dorsal compartment that creates inflammation can create thickening of the villonodular pad as well as the other soft tissues of the joint. Part of any arthroscopic procedure to remove other pathology involves villonodular pad examination and surgical reduction as necessary. Treatment of the primary disease and reduction of the inflammation in the dorsal compartment will result in regression of the edema of the villonodular pad caused by those diseases. However, if true fibrous thickening occurs, then arthroscopic removal of the villonodular pad is the treatment of choice. Intra-articular medication can reduce the size of the villonodular pad and the secondary thickening that accompanies other diseases, but this is not true with villonodular synovitis where thickening of the pad is caused by fibrosis.

The prognosis is favorable for resolution of lameness as well as normal anatomy of the dorsal fetlock with arthroscopic removal of the villonodular pad, if it is the primary condition.\(^57\) In conditions where the pad is thickened secondary to another primary disease, it is the primary disease that determines the prognosis.

Frontal Plane P-I Fractures

Frontal plane P-I fractures occur in a plane parallel with the dorsal cortex of the first phalanx at the proximal articular surface of the first phalanx.\(^58\) These fractures occur more commonly in the hindlimb than the forelimb and can occur medially or laterally. Larger fractures involve the medial and lateral dorsal eminences of the first phalanx. The size varies from small fragments that are similar to dorsal first phalanx chip fractures to the very large biarticular fractures that bisect the first phalanx, affecting both the fetlock and pastern joints (Fig. 18).

Chronic hyperextension and impaction of the first phalanx onto the dorsal surface of the distal cannon bone is the mechanism for creating this fracture. The fractures are found in all degrees of completion from just a fine fissure at the articular surface to complete and unstable fractures; this is highly suggestive of stress-fracture formation with propagation of varying degrees.

The very small fragments are unstable, similar in all aspects to the more common chip fractures in the transverse plane, and should be treated with removal similar to those fractures.

The intermediate-sized fractures involve only one eminence of the dorsal P-I and are almost always stable at less than high-speed exercise, because they are stabilized across their entire face by the fetlock joint capsule attachment and do not enter the joint deep enough into the articular surface to be unstable in normal weight bearing. If the exercise level is reduced, the fractures heal well without intervention or internal fixation because of the large stable joint capsule attachment. Interestingly, in the author’s experience, the fractures are sometimes present for some time and are well into the healing process before they cause enough lameness to limit performance. They are also frequently bilateral, occurring simultaneously in both hindlimbs or both forelimbs. Removal from training results in reduction in biomechanical strain and allows healing in most intermediate-sized fractures.

The large fractures are deep enough into the articular surface that they are unstable at rest or with simple walking exercise. In large fractures, weight bearing causes the round distal articular surface of the cannon bone to separate the dorsal and plantar articular surface components. The approach to this
fracture must be internal fixation to preserve the articular surface using techniques similar to the sagittal version of the first phalanx fracture but in the frontal plane.

Distal MClII/MtIII Sagittal Ridge OCD

A proximal sagittal ridge OCD is a frequent occurrence in any of the four fetlocks in horses. This juvenile disease, like most other forms of OCD, results from trauma to developing bone. Proximal sagittal ridge OCD has been categorized based on the reaction of the subchondral bone and the presence of a fragment. A simple defect in the subchondral bone without clinical signs is of little consequence. A defect in the subchondral bone with associated inflammation in the bone is of concern, but unless a fragment develops and is visible radiographically, there is a good chance of spontaneous resolution.

If a fragment develops in the area of inflammation, then it is important to remove the osseous lesion if the inflammation persists. As with chip fractures, small bone fragments can be surrounded by fibrous tissue and fibro-cartilage and can become quiescent; also like chip fractures, the interface of fibro-cartilage is vulnerable to high-speed exercise. Fibro-cartilage is vulnerable to erosion, causing another intrusion into the subchondral bone, initiating secondary bone healing response and debris shedding, and reinitiating inflammation and chronic degeneration within the joint. Radiographs generally underestimate the size of an OCD fragment at the proximal aspect of the sagittal ridge (Fig. 19). The cartilaginous fragment is only partially ossified in the immature horse, and the cartilaginous dimensions are much larger than the radiographic osseous fragment. Continued growth and maturation enlarges the proximal sagittal ridge OCD fragment, thins the cartilage cover as the fragment matures and the bone further ossifies, and increases the chances that a bone-to-bone interaction will be initiated to try and heal the fracture.

In the author’s experience, the severity of joint inflammation caused by OCD lesions of the top of the sagittal ridge in the fetlock is directly related to their ability to shed debris within the joint and is related to the size of the fragment and its bone-to-bone interface. If synovial effusion and clinical signs are persistent either at the time of the development of the OCD or appear later in the horse’s career, surgical removal is indicated. If the joint is totally quiescent, monitoring may be all that is needed, and if clinical signs occur, then surgical removal would be the treatment of choice. The probability of clinical signs is related to the size of the lesion, location, and intended use of the horse. A small lesion in a horse intended for moderately stressful use is unlikely to ever cause a problem. A similar lesion in a horse intended for highly stressful activity might cause chronic low-grade inflammation and eventually, joint degeneration that affects performance. Large lesions will likely shed debris and become problematic for most horses. Symptom-modifying medication warrants caution and monitoring for ongoing degeneration. Surgical

Fig. 18. (A) The first radiograph shows a small frontal plane fracture that is best removed. (B) The second radiograph shows a moderately sized frontal plane fracture that requires no treatment, only removal from exercise, and (C) the third radiograph shows a biarticular frontal plane P-I fracture.
removal with debridement of the parent bone is a routine arthroscopic procedure in the dorsal aspect of the fetlock joint and is one of the most straightforward arthroscopic procedures undertaken.

Occasionally, an OCD lesion will occur in the caudal aspect of the joint at the very proximal aspect of the sagittal ridge, but its treatment is no different than the OCD lesions of the proximal sagittal ridge in the dorsal compartment.

An OCD of the distal sagittal ridge OCD in the forelimb is a different situation from the proximal sagittal ridge OCD fragment. This lesion varies from a few millimeters to over one centimeter in size and is visible on the flexed lateral and the dorsopalmar (D-P) radiographic views (Fig. 20).

This OCD lesion occurs only in the forelimb, is primarily a non-ossified area of joint surface involving the distal sagittal ridge, and is rarely accompanied by a separated osseous fragment. It is the result of disruption of the interface between the calcified cartilage and subchondral bone at the base of the sagittal ridge and is centered ~2 cm below the top of the ridge (Fig. 20). The lesion results from a torsion force by the first phalanx loading the sagittal ridge perpendicular to the ridge and parallel to the articular surface of the condyles. Torsion in the forelimb, which causes the sagittal ridge to receive pressure from the medial or lateral eminence of the first phalanx because of the collateral ligament’s restriction of motion, results in damage to the growing bone at the base of the sagittal ridge. The trauma results in cleavage between the calcified cartilage and the subchondral bone and is a different version of OCD than is commonly seen in the horse (Fig. 21).

The lesion is more like OCD frequently seen in the human juvenile male where the cleft is through the...
superficial layers of bone rather than through the interface of cartilage and bone, like in most instances in the horse. The difference in the disease is that the ability of the parent bone to reattach calcified cartilage is far superior to the virtual inability to reattach cartilage to bone as with most OCD seen in the horse. With distal sagittal ridge OCD lesions, the cleft will heal in nearly all instances unless the lesion is disrupted, displaced, and unstable.

After the interface between the calcified cartilage and parent bone is bridged, normal maturation of the cartilage can occur, and the distal sagittal ridge OCD matures, resolving the radiographic defect. If a fragment separates, there is severe synovial effusion and lameness. Unless synovial effusion or lameness is present, distal sagittal ridge OCD lesions are normally not treated but just monitored. If clinical signs are apparent, then arthroscopic removal is necessary.

The difficulty with arthroscopic removal of this lesion is that it leaves a large deficit in the sagittal ridge and the radiographic appearance of the joint is markedly altered. Although it has little effect on the joint’s function and the prognosis for soundness is good, if the horse is ever considered for purchase, it often creates considerable concern because of the stark change in radiographic appearance caused by removal of the distal sagittal ridge.

The prognosis after removal of sagittal ridge OCD lesions is favorable if the lesions are confined to the ridge. If the lesions spread onto the condyles, the prognosis worsens proportional to the amount of joint surface lost. The biggest risk from a sagittal ridge OCD is a decision to leave it in situ when it is shedding debris. If the articular surface is irreparably damaged removal of the OCD no longer has any benefit. The damage done to the articular surface by the shedding debris is permanent, and the hyaline cartilage lost is irreplaceable. So, if persistent effusion is present, damage is being done to the joint, and without removal, the articular cartilage is being lost. When this lesion separates, the debris shedding is significant, so the synovial effusion and lameness is significant as well, indicating the aggressive ongoing damage to the joint surfaces.

13. OCD Lesions of the Dorso-Medial or Dorso-Lateral Margin of McIII/MtIII

OCD lesions of the dorsal margins of the cannon bone can occur in forelimbs or hindlimbs. Torsion of the joint stretches the joint capsule attachment to the abaxial margin of the cannon bone, resulting in the avulsion of a portion of the growing bone. (Fig. 22).

These fragments will sometimes heal, but if a fragment is present, the effect is similar to a fragment at the top of the sagittal ridge. Small fragments in this location are frequently innocuous; but if they are large enough to incite the secondary bone healing response that results in debris shedding into the joint, they are indications for arthroscopic removal similar to other fragments. Also, similar to other fragments, size of the fragment and use of the horse are factors, but if the fragment generates synovial effusion, it is an indication that it is shedding debris into the joint and is best removed to protect the joint if the horse’s career warrants.

14. OCD

Lesions of the Distal Metacarpal or Metatarsal Condyles

OCD lesions of the distal metacarpal or metatarsal condyles are normally found in the neonate and are often associated with foals that have other delayed development diseases. They can be painful but rarely form a true dessicans lesion. Therefore, osteochondrosis, which indicates delayed development, is likely a more appropriate term than osteochondritis dessicans. The lesions are more common on the more cranial aspect of the cannon bone articulation with the first phalanx, although they can occasionally occur on the palmar or plantar articulation with the sesamoid bone (Fig. 23).

In most horses, the osteochondrosis lesion matures and disappears with time. Depending on the
size of the lesion, restriction of activity may be needed. Large lesions are weak compared with normal bone and may cause lameness with exercise. Rarely, a true flap-like dessicans forms and requires removal. If this occurs in the dorsal articulation with the first phalanx, this is a straightforward arthroscopic procedure, if the cartilage flap is small. On occasion, a large flap involving much of the condyle develops; these are career limiting and are the principle concern when a distal cannon bone osteochondrosis is found. Because these lesions are so large that the damage is irreversible by removal after they develop, restricted exercise is recommended until the distal cannon bone matures.

More commonly, the thickened weakened articular surface will crack, and a subchondral bone cyst of the distal cannon bone will form. If the cyst persists, then it must be dealt with surgically.

Osteochondrosis lesions of the palmar/plantar cannon bone articulation with the sesamoid bones are normally less extensive and normally resolve with time, but the same concerns with size and the possibility of cartilage separation apply in this location as well; so, monitoring the size of the lesion and the maturation in relation to the amount of exercise stress is appropriate.

Confusingly, the palmar distal cannon bone degeneration of the racehorse has been termed OCD in some reports. This degeneration is a traumatic lesion related to the stress of high-speed exercise and is not related to the developmental disease that we normally call OCD. Preferentially, we should keep these two conditions separated in terminology and refer only to the developmental disease as OCD.

Distal Metacarpal/Metatarsal Cysts
Cystic lesions of the distal aspect of the cannon bone begin as osteochondrosis lesions on the condyles of the distal cannon bone. They are normally found in the metacarpus.61 These cysts are frequently biaxial. Osteochondrosis lesions of the condyles frequently improve with time, become ossified, and heal, remodeling into the parent bone. However, as long as the thickened articular cartilage persists, the articular surface is vulnerable to trauma.

If trauma creates a crack in the abnormal cartilage of the articular surface, synovial fluid can be pushed through the slit into the subchondral bone. The subchondral bone will begin to form a cystic cavity in response to the cyclic pressurization. Cyclic loading causes intermittent pressurization of the cystic cavity with the synovial fluid being pushed into the subchondral bone with each weight-bearing stride, causing the bone to reabsorb and form the cyst. The cyst will enlarge until it becomes large enough that the amount of synovial fluid that is pushed through the slit in the articular surface is no longer sufficient to increase the pressure within the cystic cavity; then, a sclerotic border forms around the cyst as pressure equalizes and the bone attempts to remodel and neutralize the cyst and its biomechanical effect on the bone (Fig. 24).

The inflammation associated with the active formation of the cyst often causes pain and lameness as does the increased pressure within the bone that results from the pushing of synovial fluid into the cyst on weight bearing. Distal McIII cysts often get to a steady pain-free state with time; on increases in the horse’s level of activity, the pressure may increase, and the cyst may again become reactive and painful to the horse, creating lameness. Therefore, as long as the slit-like defect in the articular surface persists, the danger of lameness from a cyst persists as well.

Cysts almost always form on concave/convex articular surfaces, because the shape of the articular surface facilitates the pressurization of the joint surface on weight bearing, forcing synovial fluid into the subchondral bone.62 Therefore, the cysts of concern are usually on the condyles.

The treatment for subchondral cysts requires a change in the joint-surface biomechanics to interrupt the pressurization of the cyst. The most effective treatment is the removal of the OCD lesion that predisposed the articular surface to forming a cystic
cavity. Enlarging the slit-like articular surface changes the biomechanics of the cyst and no longer allows the entrapment of synovial fluid within the cystic cavity. Weight bearing then does not presurize the cyst, allowing the bone to remodel and heal, often reducing the size of the cyst until it remodels the bone to normal architecture. This process may take months to eliminate radiographic evidence of the cyst. The elimination of the articular surface defect and decompression of the cyst, however, will normally restore soundness as soon as the condyle remodels. The articular defect is only of consequence if the defect in the articular surface is sufficiently large to change the shape of the surface and create a biomechanical injury to the joint surface or if the cyst has shed sufficient debris to significantly damage the adjacent articular surface.

Removal of the unsupported OCD over the cystic cavity to change the biomechanics within the cyst does not require enucleation of the cystic cavity, and this author often just removes the OCD and leaves the contents of the cyst. Hypothetically, cell populations within the cyst changed from predominantly bone-forming cells to bone-removing cells to form the cyst, and therefore, removing the stimulus for this change should revert the population to bone-forming cells within the cystic cavity, allowing the cyst to fill with bone more quickly than if the cyst is totally enucleated.

Injection of the cystic cavity with corticosteroids to reduce the inflammation has been described. This seems to be an effective means of eliminating lameness, but this treatment does not change the mechanics of the articular surface or materially alter the size of the cyst. The biomechanics of cyst creation remain, and in the author's experience, recurrence of lameness can occur with resumption of strenuous exercise. Removal of the unsupported articular surface is recommended, if the cyst is causing lameness.

In a growing horse, the articular surfaces, which contribute to growth until the horse is mature, will sometimes grow progressively from the margins and close the articular communication. If this occurs, the cyst will disappear spontaneously. After a cyst loses its communication with the articular surface, it will remodel and resolve. This is an indication for not treating cysts surgically unless they are enlarging or causing lameness.

The majority of distal metacarpal cysts that are found when radiographing horses for other causes are innocuous, many resolving on their own, but if they are causing lameness, arthroscopic removal of the predisposing OCD lesion can be an effective means of reversing the progression. The prognosis depends on the size of the cyst, the amount of remodeling of the condyle, and the condition of the interior of the joint should the cyst be shedding biologic or mechanical debris into the articulation. Cysts seldom resolve after they begin to cause lameness in the fetlock joint, so lameness is an indication for treatment.

Proximal P-I Cysts
Proximal P-I cysts are rare and will occasionally result from an OCD, but they are more likely a sequel to sagittal fractures of the first phalanx in the adult horse. If the sagittal fracture has not healed, then compression of the fracture by lag-screw fixation is the best treatment to close the defect in the articular surface. But, cysts seldom form until the fracture has partially healed, preventing compression and closure of the surface defect.

The fracture-induced cysts follow the same patterns of development as the OCD-induced cyst, being initiated by the traumatic slit in the articular surface that is created by the sagittal fracture. Treatment, however, is less straightforward, because the joint surface where they are formed cannot be accessed from the joint cavity, so an indirect approach of drilling from the dorsal aspect of the bone is normally used. An attempt is then made to disrupt the articular cartilage slit from beneath the cartilage and enlarge the communication with the joint to prevent pressurization and allow healing. The act of drilling enlarges the cystic cavity and

Fig. 24. This radiograph shows a distal McIII cyst. The intra-operative picture shows the articular surface over the cyst (brackets).
along with opening of the joint surface, reduces the likelihood of pressurization of the cyst on weight bearing, which will allow healing. If the cyst can be adequately exposed, the chances of resolution are good.

Occasional proximal P-I cysts of the condyles of the articular surface will occur, but these cysts normally are part of a process of severe degeneration and do not warrant treatment as a separate entity. If the degenerative process is advanced, they can be part of a severely painful process. Treatment of the severe degeneration sometimes requires arthrodesis to reestablish pain-free use of the limb.

Fractures of the First Phalanx
Sagittal fractures of the first phalanx are a common affliction of the Standardbred and Thoroughbred racehorses trained and raced on turf surfaces. Sagittal fractures acquire their name from the plane of fracture, which is parallel with the McIII sagittal groove, splitting the first phalanx, usually in the sagittal groove. In most instances, the fracture starts at the proximal articular surface of the first phalanx, although occasionally, a fracture will be slightly parasagittal still in the sagittal plane. This fracture has all of the characteristics of a stress fracture, because it is seen in all degrees of propagation—in the sagittal plane but always starting at the proximal articular surface (Fig. 25). Some very short fractures only affect the proximal articular surface where the fracture initiates. Fractures of various lengths extend from proximal to distal into the mid-P-I. The propagation of the fracture is determined by the amount of load subsequent to fracture initiation. The speed and accuracy of diagnosis and the severity of the exercise period that created the lameness determine how far the fracture will propagate.

It is tempting to blame the anatomy and the “screwdriver” effect of the sagittal ridge of the cannon non in the sagittal groove of the first phalanx as the cause; however, this fracture rarely occurs in horses that work twisting and turning. It is most common in horses that train on straight or gently curving paths. If the fracture happens early in the course of an exercise period, then it is likely that the fracture will propagate further into P-I. Nearly all fractures initiate proximally and propagate distally to the small medullary cavity in the distal one-half of the first phalanx. At that point, in some horses, the fracture will exit abaxially, usually through the lateral cortex at the level of the small medullary cavity, creating a bipartite fracture that involves only the proximal articular surface. Alternately, the fracture may propagate distally through the small medullary cavity and become biarticular as it enters the pastern joint.

Short sagittal fractures can be difficult to diagnose. With careful digital palpation of the normally tender dorsal cortex of P-I and digital radiography, one can make the diagnosis in most horses. In most instances, the lameness is significant, and the diagnosis is a differentiation of which of the four bones of the fetlock joint has been injured. More severe fractures have localized swelling.

After the sagittal first phalanx fracture is confirmed, the treatment is straightforward. No treatment compares with lag-screw fixation for speed of healing, quality of healing, and restoration of athletic ability. Therefore, lag-screw fixation of first phalanx fractures is the treatment of choice. Reports of case series describe healing of these fractures using either stall rest or stall rest with external coaptation with casts or supporting bandages. These are treatment options, depending on the needs of the horse. However, they are accompanied by some risk of displacement, because the fracture originates in the sagittal groove of the first phalanx and can be easily displaced by pressure from the

Fig. 25. These three radiographs show a progression of severity of sagittal P-I fractures (arrows). The left fracture is incomplete, the center fracture exits the lateral cortex, and the right fracture is a biarticular fracture.
sagittal ridge of the cannon bone should the horse plant its foot and twist. However, well-documented successes in a large percentage of cases have been published using these non-surgical techniques. Over time, more and more horsemen and surgeons favor the internal fixation alternative because of its speed of healing and quality of recovery.

The surgical procedure is normally done with the horse in lateral recumbency, although it could be done in dorsal recumbency, if desired. The traditional method of fixation is to insert screws in the transverse plane starting just distal to the articular surface. The author’s preference is to use a triangular screw configuration with two screws placed just distal to the proximal articular surface—one cranially and one caudally (Fig. 26). The third screw is inserted distal to the first two screws and proximal to the small medullary cavity of the first phalanx. With the use of two screws just below the articular surface, the restoration of soundness and the amount of callus on the dorsal aspect of the first phalanx is decreased, presumably indicating a higher degree of stability.

The screws are inserted in lag fashion using standard techniques. A cast is normally used for recovery from general anesthesia. The cast is only necessary for the initial post-operative period. It can be removed soon after recovery as per the surgeon’s preference. It is unnecessary for the healing of the fracture, because the lag-screw fixation is very stable and capable of allowing the fracture to heal without external coaptation. Normally, the fracture should be well bridged and beginning to remodel by 60 days; at 90 days, resumption of normal activity can occur. The prognosis for monoarticular sagittal fractures of the first phalanx is excellent, and restoration of athletic ability to the former level is expected. Biarticular fractures are a bit more problematic, because the pastern joint is more likely to become arthritic after this fracture than the fetlock joint. The prognosis for athletic activity is still good if the surgical procedure stabilizes the fracture well and the healing occurs without undue reaction.

Displaced fractures are treated by open reduction and internal fixation, and as long as the fracture remains principally in the sagittal plane, the prognosis is excellent, if the fracture is minimally displaced. Significantly displaced fractures involve the distal sesamoidean ligaments, which can cause persistent lameness.

The progression in severity becomes more difficult to resolve as the fracture becomes comminuted. The simplest comminuted fracture is a sagittal fracture that also fractures one or both of the palmar eminences of P-I (Fig. 27).

After this fracture occurs, permanent damage has normally been done to the horse’s athletic career. The degree of insult that is required to create this fracture normally results in damage to the soft-tissue supporting structure of the palmar aspect of the fetlock joint, principally the distal sesamoidean lig-
ments, and persistent lameness in spite of a good healing response by the bone is common in the author’s experience. Therefore, after the fracture progresses from a sagittal fracture to one with comminution, the prognosis is decreased. The treatment approach is the same: repair of comminuted fractures requires fracture stabilization through lag-screw fixation. However, in this instance, postoperative external coaptation is required and needs to be maintained until the fracture is bridging and stable.\(^{65}\)

As the comminution becomes more extensive, the difficulty of reconstruction increases, and the prognosis decreases. The presence of one cortex that extends from the fetlock to the pastern joint markedly aids reconstruction of the bone and the prognosis.\(^{68}\) After healing, these horses with these fractures have an excellent prognosis for breeding animals and in some instances, are useful for less strenuous activity, but generally, with comminution, the fracture ends an athletic career.

The most severe fracture of the first phalanx is the multiply comminuted axially unstable fracture of the first phalanx.

These fractures have no intact cortex from proximal to distal P-I. In the absence of that cortex to aid the reconstruction, some type of external weight-bearing support using a coaptation frame or a transfixation pin cast must be used.\(^{69}\) The author's preference is the multiple pin cast (Fig. 28). The cast transfers the weight from the metacarpus or metatarsus through the cast to the ground until the first phalanx has healed sufficiently to allow weight bearing.\(^{70}\) The prognosis for healing of comminuted fractures is favorable, although it is accompanied by a higher incidence of laminitis in the opposing weight-bearing limb than would be expected from other types of first phalanx fractures and attempts at salvage have to take this into consideration.\(^{68}\) Treatment of the multiply comminuted P-I fracture is considered only for salvage and not for return to athletic activity. However, if healing occurs in an uncomplicated fashion, the horse can be functional and live a normal, unrestricted life as a breeding animal after the healing is complete.\(^{68}\)

Free Fragments in the Fetlock Joint

OCD lesions and occasionally, chip fractures can be shed to become free fragments within the fetlock joint. When a free fragment occurs within a fetlock joint, it normally moves about randomly until trapped in one of three locations. The most common location for a free fragment is in the palmar or plantar pouch intermingled or encased in the synovial fronds of the proximal plantar or palmar pouch of the fetlock joint. Radiographically, the free fragment can be seen within the palmar/plantar pouch soft tissue. This is an innocuous location for the fragment, because it does not interfere with the articulation of any of the four bones of the joint.

Free fragments in the fetlock joint can be shed to become free fragments within the fetlock joint. When a free fragment occurs within a fetlock joint, it normally moves about randomly until trapped in one of three locations. The most common location for a free fragment is in the palmar or plantar pouch intermingled or encased in the synovial fronds of the proximal plantar or palmar pouch of the fetlock joint. Radiographically, the free fragment can be seen within the palmar/plantar pouch soft tissue. This is an innocuous location for the fragment, because it does not interfere with the articulation of any of the four bones of the joint.

A second location where free fragments are commonly found is in the small depression on the abaxial aspect, medial or lateral, of the cannon bone, usually just cranial to the collateral ligament. The articular surface of the distal cannon bone extends around the margin of the joint into the medial and lateral condylar fossa to facilitate the highly mobile collateral ligament in its path along the abaxial aspect of the bone. The fragment often becomes lodged in the condylar fossa. These are seen as radiographic free bodies adjacent to the edges of the distal cannon bone.

The third location of free fragments is between and distal to the sesamoids in the small cul-de-sac of the fetlock joint just distal to and below the sagittal groove of the first phalanx.

As long as the fragments stay in one of these three locations, they are innocuous. Eventually, most of them become covered by fibro-cartilage and are non-
irritating to the surface of the bone. However, if the fragment dislodges and then passes through the articulation, it can do damage to the articular cartilage, and on passing through the interface between the cannon bone and the first phalanx or the cannon bone and a sesamoid bone, it causes severe pain. A marked acute lameness, in some instances causing the horse to carry the limb for a few strides before reinitiating a normal gait and becoming sound again within a few strides, is characteristic of a free fragment passing through an articulation. Based on the author’s experience, the acute stretch on the stabilizing soft tissues of the joint is severely painful but resolves quickly if the fragment exits the joint surface. A history of acute, severe lameness that only lasts a few strides is highly suggestive of a free fragment. It is possible for some free fragments to become trapped between the articular surfaces, causing severe damage, but most progress through the articulation and end up in one of the common locations.

If the free fragment is causing lameness and/or effusion, it is best removed arthroscopically. Free fragments found coincidentally that are not causing lameness in heavy-use horses are generally removed as well, because, in the author’s experience, the dislodging of a fragment into the articulation is not predictable and may occur at any time. Arthroscopic removal is normally a straightforward retrieval in the palmar or plantar pouch, above or below the sesamoids. Dorsally, the retrieval is done partially by arthroscopic palpation, viewing the cannon bone margin and the abaxial aspect of the joint and palpating the depression where the fragment is lodged. After surgical removal, the prognosis is favorable unless the origin of the free fragment is causing lameness.

15. Diseases of the McIII/MtIII Sesamoid Articulation

Palmar/Plantar Distal Cannon Bone Injury

Inflammation of the Distal Cannon Bone

Inflammation of the distal cannon bone is one of the most common clinical problems seen in the Thoroughbred racehorse. It is also seen in the Standardbred racehorse and can be seen on occasion in horses of other athletic disciplines such as show jumping and dressage. When wear and tear on the bone accumulates because of high-level training stress, the microdamage stimulates the bone to respond. When bone must adapt to high-level exercise by increasing its strength, it has one of two options. The bone can alter its structure or it can alter its shape to prevent the wear and tear damage of increasing levels of athletic activity.

In most situations of high-strain loading, the bone uses both response possibilities to adapt to the new loads. It adds additional stronger bone and alters its shape to better neutralize the new and increased loads being experienced. But, the distal aspect of the cannon bone cannot alter its shape, because it is part of the fetlock joint surface, which has a 270° radius of motion and an unalterable, cartilage-covered joint surface. Therefore, the only possible adaptation that can occur in the bottom of the cannon bone is an alteration in the structural strength of the bone, because it cannot change shape. The bone of the distal aspect of the metacarpus or metatarsus must become considerably stronger by becoming denser. Trabecular bone is nearly converted to cortical bone at the bottom of the metacarpus and metatarsus. This adaptation takes time and sacrifices the stress absorption capability of the bone when the cancellous bone is hypertrophied to the point that it is nearly cortical.

Bruising of the bottom of the cannon bone results from accumulation of damage at a rate in excess of the bone’s ability for repair and is a relatively common cause of pain. This is commonly termed maladaptive bone remodeling, but at this stage of the disease, it is likely that the adaptation is normal but the rate of stress accumulation exceeds the capacity of the bone to respond. A pathologic process is not yet present but the capacity of the normal bone response to manage the rate and degree of stress application is exceeded. Bone bruising accumulates and results in inflammation, pain, and lameness, similar to bruising at any site. If you examine the palmar articular surface of most racing Thoroughbreds grossly, some degree of this bruising is present grossly and histologically (Fig. 29). The cycle of overload of the bone causing microdamage followed by over-repair to strengthen the bone is the cycle that is repeated over and over to train bone. If the over-repair gets behind the overloading, the bone begins to be damaged or bruised. As the bruising reaches a certain threshold of inflammation, pain ensues.

The distal cannon bone bruising is usually bilateral and often quadrilateral when the stress of training exceeds the bone adaptation. The most common presenting complaint is not lameness but rather decline in performance, weakness, and in some instances, even complaints of ataxia because of the gait alteration. Behavioral alterations are also frequent. Horses that formerly trained willingly and then become reluctant to train commonly have bilateral or quadrilateral distal cannon bone bruising.

Predisposition to Injury

Much has been made of conformation abnormalities leading to the higher incidence of certain injuries. Reports document an increased risk of fetlock joint injury associated with certain conformations. Varus conformation of the forelimb has been shown to increase the incidence of dorsal-medial P-I chip fractures in the fetlock joint. But, for all but the extreme conformational abnormalities, the horse is amazingly adaptable to a wide range of conformation variations in the fetlock joint.
Training methods are commonly accepted as having an influence on the incidence of injury in horses. Some trainers have developed methods that rarely result in serious injury and still create horses that are able to compete effectively, but the opposite is also true.

There are two predispositions for an increased risk of lameness and injury that we have inadvertently created in the highly trained equine athlete. These risks encourage damage to the palmar distal cannon bone because of their propensity to increase the load and reduce the ability for repair of the distal cannon bone damage. The first is daily short bursts of high-intensity exercise with prolonged periods of stall rest. This system of training is necessitated by the high concentration of horses in areas where there is limited opportunity to remove them from the stall and by the tenuous shoe/foot structure that causes horses to lose shoes if allowed free-choice exercise in solid footing.

In the ideal scenario, the horse is allowed free choice exercise for much of the day, mimicking the evolutionary natural state. Naturally, horses spend most of the day slowly walking and grazing. Because horses have no valves in the veins of the distal limb, they are prone to limb edema secondary to venous stasis if confined. They also have no significant muscle mass below the carpus and tarsus, and therefore, they have little blood flow demand in the lower part of the limb in most instances. Circulation in the lower limb is stimulated by the way a horse grazes. They eat a few mouthfuls, walk a few steps, then eat a few mouthfuls, and walk a few steps in a continuous pattern covering acres of ground in each day's grazing. But when horses are confined to a stall for the major part of the day, the horse spends most of the day standing. This results in passive edema or "stocking up" in the lower part of the limbs, which we have counteracted by the use of stall bandages to keep pressure on the exterior of the limb and prevent the lymphatic edema that results from circulatory stagnation.

But, these "stall bandages" do little to aid the circulation of the bone, and hypothetically, they actually increase the soft-tissue resistance to the circulatory exit of blood from the bone, which has an interior-to-exterior circulatory pattern. When undergoing high-speed exercise training, bone needs active motion to facilitate blood flow through the bone for the metabolic needs, which are increased by the trauma and repair of the daily exercise trauma.

Bone circulation is a low-pressure system. Passive edema on the venous side of the circulation slows the return of blood to the circulation and increases the circulatory resistance, encouraging interosseous hypertension. External compression can effectively reduce soft-tissue edema, but resistance to blood outflow facilitates intraosseous hypertension, causing inflammation and pain.

In the author's clinical experience, the current management of daily exercise impedes the horse's natural osseous circulation needs and increases chances for remodeling disease. The wear and tear of high-speed exercise concentrated in short periods of time followed by confinement for most of the rest of the day reduces the natural circulation that aids repair and creates the not surprising result of numerous bone remodeling diseases that we see clinically in our equine athletes.

There is no easy solution for current horse-exercise management because of space restrictions, and creation of year-round racing and performance calendars allow this type of management to affect more horses. When horses had forced periods of non-competition, alteration of training regimens and reduction of exercise were the rule, which resulted in better remodeling activity for at least some period each year. This facilitated the healing and prevented the chronic wear-and-tear diseases that seem to be more frequent.

Our inability to maintain shoes on a fit racehorse during periods of free-choice exercise also makes it difficult to use paddock activity for many horses. However, even a period of walking exercise a second time during the day should be helpful to the circulation.

The second predisposition to injury that we have created is traction devices on the forelimbs. Specifically, toe grabs have been associated with the two

Fig. 29. This intra-operative photograph of the distal aspect of the metacarpus illustrates bone bruising (arrows) of the bottom of both condyles of distal McIII. The cartilage has been removed from the lateral condyle. Note that the articular cartilage is grossly normal over the bruise.
major catastrophic structural failures in the Thoroughbred: disruption of the suspensory apparatus and condylar fracture. Experiments using instrumented shoes to examine the effect of toe grabs on the horse’s limb have shown that the acceleration/deceleration force is greatly increased by toe grabs. But, the increase in force is not in the direction that one might first anticipate. Toe grabs are used for traction on the foot during the propulsion phase of the horse’s stride, but the highest forces are generated in the horse’s limb during impact with the ground during deceleration when the toe grab increases the braking effect. On soft surfaces, the toe has a toe-down progression on landing as the toe progresses inward and downward into the soft surface, having the effect of raising the heel. The addition of the horse’s toe grab to the horse’s toe increases the downward progression of the toe in a soft surface. Turning the toe down has the effect of raising the heel, lowering the fetlock, and pre-loading the suspensory apparatus before the application of weight. This change in the position of the foot increases the stress or force that the suspensory apparatus experiences when the horse loads the limb. This anatomic “pre-load” has the effect of increasing the load on the suspensory apparatus and the condyles of the distal cannon bone during exercise.

Because the forelimbs function biomechanically quite differently than the hindlimbs, the effect is likely different in the hindlimb. So, the correlation to injury in the biomechanical forces measured in the forelimbs cannot be transposed to the hindlimbs with the same expected effect. But, the association of traction devices and forelimb injuries encourages moderation or elimination of their use.

If the bruising of the distal cannon bone is bilateral or quadrilateral, the diagnosis can be challenging, because a single limb does not appear to be lame. The diagnosis is best made with diagnostic local anesthesia of the distal cannon bones to confirm the site of pain. Nuclear scintigraphy is not a specific indicator for this disease, because many horses have significant uptake in the distal metacarpus and metatarsus that is not accompanied by significant pain. So, the only diagnostic criterion of dependable significance is the disappearance of the lameness after local anesthesia of the distal aspect of the metacarpus/metatarsus.

Fortunately, diagnostic local anesthesia can be accomplished in a relatively specific fashion using the palmar metacarpal and metatarsal nerves. Local anesthesia of one of the limbs will result in easily identifiable lameness, because the pain remains in the contra-lateral limb. Inflammation of the distal cannon bone (Fig. 30) will occur in either the medial or lateral condyle in the forelimb, but it is almost exclusively a lateral condyle disease in the hindlimb. The reason for this is unclear.

If bruising of the bottom of the cannon bone is diagnosed early and the horse is given rest early before anatomic changes occur, the prognosis for resolution is excellent. The rest from training must be in the form of paddock exercise, however, and not stall rest. Confinement to a stall contributes to the creation of this problem and prevents the remodeling needed to heal the chronic bruising of the bone. Allowing the horse free-choice paddock exercise best facilitates the healing process. Free-choice exercise is the ideal treatment, because it best emulates the natural grazing stimulus of loading and unloading the bone continuously that facilitates osseous circulation and remodeling. Vasoactive drugs to improve the blood flow through the bone, which is assumed to be in a hypertensive state similar to inflammation in other bones, would also be a logical treatment and may help bone remodeling.

If training is continued in spite of the accumulation of damage, one of several pathologic conditions will occur. Condylar fracture, palmar articular fracture, and palmar articular degeneration with alteration of the shape of the distal cannon bone are all possible sequelae. The prognosis is directly dependent on the degree of change in the bone architecture as a result of the accumulated damage.

The most effective approach to this disease is diagnosis before anatomic damage to the cannon bone and removal from training before anatomic derangement occurs. Retrospective analysis of horses treated with a period of rest after a diagnosis of pain located in the distal metacarpus or metatarsus has been shown to be successful in allowing the bone to recover.
The process of accommodation of the articular surface of the distal cannon bone occurs in most horses without incident; however, bruising and inflammation of the distal cannon bone as a part of the accommodation to high-level exercise is likely the single most common affliction that a high-speed athlete must overcome to reach its athletic potential. There are several major fetlock joint injuries that are part of the disease complex initiated by damage to the palmar/plantar distal cannon bone articular surface. The number of pathologic events that the chronic high-speed exercise loading can take in the distal cannon bone presents somewhat of a dilemma in understanding the pathology. Why some horses accommodate without problems and why some horses sustain parasagittal fractures of the condyles, whereas other horses simply accumulate the load over time, resulting in collapse of the articular surface of the distal cannon bone is not clear. Anatomy is certainly one of the considerations; condylar fractures predominate on the lateral condyles, and palmar articular degeneration predominates on the medial condyle in the forelimb. In the author’s experience, training regimens seem to be factors; many stables rarely experience this problem, whereas some have a high incidence.

Condylar Fracture

Condylar fractures are parasagittal fractures of the distal articular condyles of the metacarpal and metatarsal bones. They are one of the most frequent fractures in the racehorse. Condylar fractures are one of a complex of the distal cannon bone injuries that originate from the stress accumulation within the distal palmar/plantar articular condyles of the cannon bone that accompanies high-speed exercise. When the progressive accumulation of the stress of exercise begins to exceed the rate of repair, structural damage ensues. The condylar fracture originates on the palmar/plantar aspect of the bone in the area where the sesamoid bone articulates with the condyle. It is much more common in the lateral condyle than in the medial condyle for reasons that are not readily apparent anatomically, because the medial condyle is larger than the lateral condyle and accepts more weight. Perhaps weight distribution plays a role in the creation of the fracture.

It is generally accepted that accumulation of stress and creation of microfractures are the initiating causes of the condylar fracture. The microfracture creation in the palmar/plantar condylar area progresses until enough coalescence occurs that a macrofracture begins. The macrofracture begins at the articular surface and progresses proximally in a sagittal plane. Condylar fractures are diagnosed in all degrees of propagation from very short fractures just being initiated at the articular surface to complete displaced fractures. This spectrum of fractures following generally the same plane but in varying degrees of progression is typical of stress-fracture initiation and propagation.

Lateral fractures most often propagate abaxially from the site of initiation on the axial two-thirds of the lateral condyle and exit the cortex 7–8 cm proximal (Fig. 31) to the fetlock joint, but they can occur more axial or abaxial and propagate axially or abaxially. Medial fractures more often propagate axially and can progress proximally as far as the proximal metaphysis of the bone. If the fractures propagate until they exit during exercise, they will displace and set off a chain of events that destabilizes the fetlock joint, which may result in disarticulation. Fortunately, most but not all condylar fractures show signs of lameness before completion and are diagnosed before the fetlock anatomy is destroyed.

Retrospective analysis of a large group of condylar fractures has shown that the configuration into the articular surface plays a role in the eventual prognosis. The presence of articular comminution on the palmar/plantar condyle articular surface reduces the prognosis for a return to racing after surgical treatment of this injury. This comminution seems to be a hybrid of the palmar distal cannon bone semilunar fracture and the condylar fracture. The wedge-shaped articular comminution often occurs in areas of inflammation in the distal aspect of the cannon bone, at the same location that the condylar fracture occurs. The fracture can start as a palmar articular fracture, eventually progressing to a condylar fracture.
Diagnosis in recent years has been greatly aided by a better understanding of the pathophysiology and anatomic configuration of this injury. This understanding has been facilitated by the newer radiographic and scintigraphic technology that has better elucidated the pathology of this injury.

If the condylar fracture is discovered as a fine fissure fracture of only the palmar condyles, then the injury is stable, and no surgical treatment is needed. If the injury progresses proximally and certainly, if it can be seen on the flexed dorsal palmar as well as standing dorsal palmar radiographs, the fracture benefits from surgical treatment by internal fixation in both the speed of healing and the quality of recovery. Condylar fractures that are non-displaced have historically been treated by the non-surgical means of confinement and exercise restriction. But, experience has taught veterinarians and horsemen that the most rapid, most functional outcome occurs with compression and stabilization of the fracture through open reduction and internal fixation with lag screws. The prognosis is directly related to the severity of the injury.103,108

Displacement of the fracture adds to the severity of the injury and decreases the prognosis, as does articular comminution. Articular comminution does not preclude the successful repair if the articular comminution can be reduced and reconstructed with the reconstruction of the primary fracture. But, if a defect in the articular surface persists, the probability of a successful repair is greatly reduced. The goal of most condylar fracture repairs is restoration of athletic activity, which is achievable with successful internal fixation and anatomic reconstruction. Any residual articular defects or malreduction of the condylar fracture greatly reduces or eliminates the chance of racing.108 The margin for error is quite low in this heavily loaded joint surface.

The surgical repair is normally approached from the ipsilateral aspect of the injury, and non-displaced fractures can be stabilized without articular visualization for reduction.108 Displaced fractures, however, should be visualized arthroscopically to assure anatomic reduction has been achieved. It is impossible to assure this with only radiographic examination of the injury intra-operatively, and therefore, arthroscopic or open visualization of the reduction of the fracture is indispensable to the successful repair in displaced fractures (Fig. 32).

After the joint has been anatomically reduced and confirmed visually, the fixation is technically similar in non-displaced and displaced fractures with the placement of lag screws across the fracture. Two screws are most commonly used. The distal lag-screw location is most important. It should ideally be concentrically located in the condyle of the distal cannon bone to provide compression on the entire articular surface (Fig. 33).

Location of the screws is more difficult than one might anticipate, especially when swelling of the injury distorts the anatomy. Some surgeons repair these fractures standing; because the location of the screws in the ideal position is important and a few millimeters can make the difference between success and failure, the method that assures the best possible repair in the situation should be used.

Lateral condylar fractures and medial condylar fractures that do not displace or spiral proximally require no external coaptation for recovery from anesthesia if the internal fixation is acceptably done. Condylar fractures that spiral into the diaphysis of the bone and in some instances, propagate proxi-
mally as far as the proximal metaphysis have increased risk for failure both in recovery and in the post-operative period. Horses under the author’s care have displaced spiraling condylar fractures as late as 6 wk post-injury. Publications have described the placement of multiple screws in a standing horse to obviate the need for recovery from general anesthesia. But, in a situation where the difference between success and failure with condylar fractures can be a matter of millimeters of variance for ideal screw placement, careful anatomic reduction and accurate screw placement in the condyles is critical, and the addition of a neutralization plate to the diaphysis of the bone to protect the bone during the healing period is preferred (Fig. 34).109

The reasons for using this technique are a more rapid return to soundness, reduced risk of displacement associated with the spiraling diaphyseal fracture both in recovery and during the post-operative convalescent period, and rapid, safe dismissal from hospital care. In the author’s experience, horses with spiraling diaphyseal fractures stabilized by screws alone remained lame for weeks, but horses with neutralization plates applied were sound immediately post-surgery. The plate is placed intraoperatively, and then, after fracture healing has occurred, the plate is removed, often with the horse standing, before reinitiating training. Horses of lesser use than racing tolerate a plate on the diaphysis of the metacarpus, but in an experimental study performed by the author, six of six horses became lame as they approached race speeds with a plate in their metacarpus. Therefore, for horses performing high-speed exercise, such as racing, the plate on the diaphysis of the cannon bone must be removed.

After repair, the normal aftercare requires ~3 mo. The prognosis has been well documented, and one can anticipate successful recovery with condylar fractures in most instances, including articular comminution, if the fracture is anatomically reduced and stabilized.

Palmar semilunar fractures occur in some horses subsequent to distal cannon bone inflammation without a sagittal condylar fracture and before the degeneration of the condyles (Fig. 35). If these fractures are small and especially if they are taller (proximal to distal) than they are wide (medial to lateral), training in their presence will result in a condylar fracture. If the semilunar fracture is wider than it is tall, then lameness sufficient to prevent exercise usually ensues before a condylar fracture is created. Palmar semilunar fractures can heal if they do not separate from the parent bone. If they separate from the parent bone, they usually progress to degeneration with reabsorption and degeneration of the articular surface. The
semilunar fracture that separates from its parent bone has no blood supply. It is totally intra-articular. If interface with the parent bone is unstable, there is no chance for healing, and joint function is lost.

Therefore, a period of stall rest is normally the treatment for this fracture. It cannot be stabilized surgically, and exercise encourages instability. If the fracture separates from the parent bone or depresses into the parent bone during the healing process, the prognosis is poor. If healing occurs without alteration of the shape of the articular surface, the horse has a good chance of returning to athletic activity.

Palmar Articular Degeneration of the Distal Cannon Bone
The medial condyle of the forelimb is the most likely to accumulate structural damage to the subchondral bone and undergo alteration of the shape of the bone with collapse and flattening of the articular surface. It is unknown why a condylar fracture is more likely on the lateral condyle and the palmar degeneration is more common medially, but there is an obvious size difference in the two condyles. This may play a role, but either condition can occur on either condyle.

The term maladaptive applies in this condition because of the progressive alteration of bone anatomy in response to exercise stress rather than the accommodation to exercise that is needed. Perhaps this is just application of stress in excess of the ability of the bone to respond. The degenerative remodeling process results in irregular areas of bone absorption and production, and when combined with weight-bearing stress, it causes the collapse of the normal architecture of the condyle.

The change in shape of the cannon bone alters the congruence of the articulation with the face of the sesamoid, increasing the joint surface damage and eventually, causing palmar degeneration of the articular surface. Palmar articular degeneration can initiate fractures of the distal cannon bone, but in most instances, a condylar fracture does not occur in the area of palmar articular degeneration. Subchondral bone collapse is more common than a condylar fracture.

This collapse of the joint surface because of the damage to the supporting subchondral bone is accompanied by damage to the cartilage. If training continues in the face of this alteration and loss of congruence with the face of the sesamoid, a fracture of the base of the sesamoid is the common sequela.

Palmar condyle degeneration may appear radiographically as simple flattening, but after the alteration in shape of cannon bone begins to occur, degeneration of the articular surface always accompanies it (Fig. 36). The degree of damage to the palmar articular surface determines the prognosis. One must be careful not to misdiagnose palmar condyle flattening as the radius of curvature of the articulation of the distal cannon bone with the first phalanx is considerably different than the radius of curvature of the sesamoid bone articulation and gives the appearance of palmar condyle flattening if one is not aware of these different articulations. This leads to the misdiagnosis of palmar condyle flattening in the yearling, which never occurs. Palmar flattening is an acquired disease that occurs during training and usually after a significant amount of race participation, because it takes months of continuous loading to create the response in the distal cannon bone.

Palmar articular surface degeneration and loss of articular surface on the palmar or plantar aspect of the distal cannon bone with flattening of the bone is a permanent change and virtually always results in chronic lameness. Occasionally, repair will occur sufficiently to allow return to exercise at a lower exercise level, but the damaged articular surface rarely allows the horse to maintain its form after radiographic flattening of the palmar distal cannon bone begins. Some horses are surprisingly tolerant of even significant degrees of palmar distal cannon bone ulceration for a time, and they are able to continue racing in a diminished capacity in spite of the fact that the articular surface has been breached with a loss of articular cartilage. However, at some point, the injury becomes intolerable in all horses, no matter what the treatment.

The career-ending clinical development is frequently a fracture of the base of the sesamoid bone that results from the uneven biomechanical loading of the sesamoid bone subsequent to the flattening of the articular surface of the distal cannon bone. Treatment of this type of base sesamoid fracture is unrewarding, because the treatment of the sesamoid fracture is simply treatment of the result and not the cause of the fracture. The primary disease of flattening of the distal cannon bone persists. There is no surgical treatment for palmar distal cannon degeneration.
bone degeneration, because debridement of the pathology on the bottom of the cannon bone simply adds to the loss of articular surface and does not negate the ulceration that accompanies the flattening. This is a career-ending injury and can lead to painful degeneration of the fetlock joint, which may require joint fusion.

**Sesamoiditis**

Sesamoiditis by definition is inflammation of the sesamoid bone. More accurately, it is an inflammation of the insertion of the suspensory ligament into the proximal abaxial aspect of the sesamoid bone where the ligament is anchored into the bone. Sesamoiditis can and does occur in any of the eight sesamoid bones of the horse. Past confusion as to the definition of sesamoiditis was partly resolved by a publication that separated the different radiographic characteristics including enlarged and increased numbers of vascular canals, proliferation on the abaxial aspect of the sesamoid bone, and lucency on the abaxial border of the sesamoid and documented their effects on racing performance.39,112 In this study of the radiographic appearance of sesamoiditis, vascular canals that were increased in number but regular in nature with parallel borders <2 mm in diameter did not affect the horse’s performance (Fig. 37). As the vascular canals became altered in size with the canal borders becoming wider radiographically and usually adopting a somewhat barrel-shaped appearance, the horses’ performances declined both in number and quality of races as the number of canals increased. In another study where a different definition of vascular canal enlargement was used, no difference in performance was shown.11 However, the incidence in this group was very high (79%) and may have clouded the results.113

In my experience a decline in performance with sesamoiditis occurs in horses with all uses.114 Horses place a significant load on the suspensory apparatus in nearly all activities. Because the fetlock is a totally passive joint with no option to reduce the load and protect the joint voluntarily, sesamoiditis affects horses used in most types of performance.

In one study, proliferation and lucency had little effect on athletic performance in racing Thoroughbreds.39 It is probable that lucency in the area of the suspensory ligament attachment is important in the acute stages of the inflammation. Because these studies were done on yearlings by the time they began their athletic careers, it is probable that the lucencies seen may have healed. In another report, osteophytes on the abaxial aspect of the sesamoid bones in hind fetlocks were associated with decreased performance.41

Marked irregularities of the suspensory insertion and significant demineralization in the attachments are certainly suspicious for disease of the insertion of the suspensory ligament into the sesamoid bone, but in the absence of enlarged vascular canals, they are not predictive of decreased performance in yearlings. The enlarged vascular canals, however, were proven to be a good indicator of decreased performance.39 It is likely that, similar to the distal sesamoid (navicular bone), these enlargements in the vascular canal are markers for previous inflammation in the bone.115,116 Unlike the navicular bone where they are not predictive of performance, the damage seems to be irresolvable after the suspensory ligament insertion into the proximal sesamoid bone is damaged. The horse cannot recreate the exact structure of the natural insertion of the suspensory ligament into the sesamoid bone. Therefore, although healing may occur and the attachment of the suspensory ligament to the sesamoid bone may be reconstituted with scar tissue, it is improbable than any type of scar tissue will be able to function in a fashion similar to the undamaged suspensory ligament attachment to the sesamoid bone. When the level of work is sufficiently demanding, the fibrous tissue reconstitution of the ligamentous insertion will fail. Therefore, the higher the desired level of performance expected of the rehabilitated horse with sesamoiditis is, the worse the prognosis.

There is no surgical or medical therapeutic plan that can alter or improve the horse’s reconstitution of the suspensory ligament anchorage into the sesamoid bone after it has been lost. So, after the suspensory ligament attachment is damaged beyond a certain point, it is permanently disabling, and the degree of problem that it causes the horse is directly related to the amount of sesamoid interface attachment damaged.

The prognosis is difficult to alter, because no treatment can reattach the suspensory ligament. However, if active sesamoiditis is underway and the enlargement of vascular canals is ongoing and at-
tended by lucency in the abaxial border of the sesamoid bone at the suspensory insertion, attempts to reossify the sesamoid bone by reducing the inflammation can help to mitigate the ongoing damage of suspensory ligament avulsion. When active sesamoiditis is underway, damage to the suspensory ligament insertion creates inflammation. Inflammation in the bone causes bone reabsorption, further weakening the suspensory ligament insertion, which predisposes to further damage and stimulates further inflammation in a cyclic progressive manner. Therefore, removal from exercise stress and treatment of the inflammation within the bone is indicated.

Because hypertension within the bone is associated with inflammation, it is likely that the same situation occurs at the injured proximal sesamoid bone. Medications that aid blood flow through the bone without hypertension are hypothetically indicated. Aspirin or pentoxyfylline to alter the platelet adhesive characteristics and capillary blood flow as well as vasoactive drugs such as isoxsuprine make therapeutic sense. This is the author’s recommended treatment of choice, and although the response to this therapy is slow and not dramatic, these compounds seem to be the most effective of all the treatments that have any effect on the sesamoid bone and sesamoiditis.

Distal Sesamoidean Ligament Desmitis

There are four distal sesamoidean ligaments, the short, cruciate, middle or oblique, and superficial or straight, that attach to the distal aspect of the sesamoid bones. Sometimes, publications inaccurately call these X, Y, Z ligaments. The short and cruciate sesamoidean ligaments are relatively small, and desmitis of those ligaments is rarely recognized as a clinical entity. They play a role in the creation of palmar/plantar first phalanx chip fractures, but primary desmitis is not discernable. The likely reason for this is that these ligaments are so small relative to the middle and superficial distal sesamoidean ligaments that they are of little consequence in the strength of the attachment of the sesamoid bone on the palmar/plantar aspect of the first and second phalanx in the loaded fetlock joint.

Middle distal (oblique) sesamoidean ligament desmitis causes significant lameness. The middle distal sesamoidean ligament’s origin occupies the majority of the base of the sesamoid; the distal insertion covers a large portion of the palmar/plantar aspect of P-I in a V-shape attachment extending from just distal to the fetlock joint to just proximal to the pastern joint. The middle distal sesamoidean ligament is by far the largest of the distal sesamoidean ligaments in its origin and insertion, and because of its size, it plays the largest role in the support of the fetlock joint below the sesamoid bones. Middle distal sesamoidean ligament injuries are difficult to resolve for high-speed athletic activities at any location other than at the insertion on the palmar or plantar aspect of the first phalanx.

If the middle distal sesamoidean ligament is inflamed, thickening can sometimes be identified distal to the sesamoid bones abaxial to the flexor tendons, but most horses have no outward clinical findings, and the site of pain is identified with diagnostic local anesthesia. Middle distal sesamoidean ligament inflammation at its insertion on the palmar/plantar aspect of the first phalanx is a common finding in horses that perform twisting and turning athletic events. Fortunately, the insertion of the middle distal sesamoidean ligament on the first phalanx has considerable redundancy, and the insertion enjoys a biomechanical advantage in its oblique insertion into the bone, so structurally important injuries of the distal insertion are seldom encountered. Complete disruption rarely, if ever, occurs at the first phalanx insertion, and in most cases, desmitis of the distal insertion is only recognized after radiographic appearance of insertion proliferation shows that the insertion has been damaged or with sophisticated diagnostic techniques.

Inflammation of the middle portion of the middle distal sesamoidean ligament is a major injury. The middle distal sesamoidean ligament is difficult to image because of the oblique nature of the ligament fibers. Therefore, the ligament must sustain a significant amount of damage before the ultrasound examination can dependably detect the injury. The best diagnostic tool to determine the degree of damage is MRI. The MRI is dependably able to detect and determine the severity of middle distal sesamoidean ligament desmitis.

Injuries of the middle distal sesamoidean ligament origin from the base of the sesamoid bones are very problematic for high-speed activity. Unlike the large oblique insertion into the first phalanx, which is biomechanically advantageous, the origin of the middle distal sesamoidean ligament into the base of the sesamoid proximally is perpendicular to the base of the sesamoid bone and therefore, repair of that insertion is very difficult. It carries a decreased prognosis similar to the suspensory branch insertion into the abaxial aspect of the sesamoid bone, because it requires reconstruction of a nearly non-reconstructable origin on injured bone.

After desmitis occurs to the degree that it shows clinical signs for both mid-ligament and proximal ligament injuries to the middle distal sesamoidean ligament, the horse is often permanently injured. Therefore, rest and rehabilitation is imperative, but early diagnosis is the most important, although most difficult, component of treatment. This ligament heals slowly and relatively poorly in most horses. Treatment through medical or surgical means can resolve some injuries, especially the more distal injuries in performance horses. Racehorses with this injury are a much more difficult problem, partially because they tend to be injured near the origin on the sesamoid bone.

Biologic therapy, such as stem-cell augmentation, theoretically has the best chance to optimize repair
but has yet to show efficacy, and in the author’s experience, it has not been very helpful. We have yet to master the ability to insert these cells and affect any significant repair. The use of these techniques for biologic repair has yet to be dependably successful. The prognosis for an injury to the body or proximal insertion of the middle distal sesamoidean ligament is unfavorable.

Injury of the middle distal sesamoidean ligament origin at the base of the sesamoid bone is relatively common in the foal, and the small fragment avulsions from the base of the sesamoid that are found radiographically in young horses are markers of injury for this ligament (Fig. 38). These fragments are an indicator of a predisposition to further damage as high-speed exercise levels are reached. Removal has been recommended, but removal of the fragments removes the result of the injury and does not negate the primary problem of loss of distal sesamoidean ligament attachment; it may, in fact, add to that problem because of the difficulty of accessing this area without damaging additional fibers. A few horses can overcome the presence of an injury to this area, but most with fragments from the middle of the base of the sesamoid become lame before they reach strenuous levels of exercise. The lameness is not the result of the osseous fragments but rather is caused by the loss of the fibers of the distal sesamoidean ligament that attach to the base of the sesamoid. The remaining fibers usually prove insufficient for high-level exercise without injury and lameness.

Injury to the superficial (straight) distal sesamoidean ligament will occasionally occur on the distal abaxial aspect of the sesamoids on the abaxial distal border of the sesamoid, but the majority of those fibers insert into the more flexible base of the intersesamoidean ligament scutum between the sesamoid bones. The superficial distal sesamoidean ligaments tolerate insult relatively well at their proximal origin compared with the middle distal sesamoidean ligament and are rarely injured clinically. The proximal attachments often do create osteophytes when they are injured in the juvenile animal, but they rarely cause clinical lameness. The osteophytes are seen on the palmar/plantarolateral or medial margin of the sesamoid bone on radiographs.

Desmitis of the body of the superficial distal sesamoidean ligament is also rare, and injury to the superficial distal sesamoidean ligament insertion at the attachment to the second phalanx scutum is the most commonly identified injury. The prognosis has a direct correlation with the quantity of fibers injured at the distal sesamoidean ligament insertion. But, if the injury has not detached a large portion of the bone at its insertion, they are most often found as an incidental finding on radiographs and rarely, as part of clinical lameness. When injury is diagnosed, it is usually a result of its effect on the pastern joint and not because of ligamentous pain.

The four distal sesamoidean ligaments have distinct biomechanical injury predispositions of their own related to their size and importance. The injury to the distal sesamoidean ligaments that is most devastating is a complete avulsion of all four ligaments from the base of both sesamoids that occurs during high-speed activity in the racehorse. It often occurs with no warning, and because it is a disabling injury to the suspensory apparatus of the fetlock joint and cannot heal, it must be dealt with by circumventing the need for the suspensory apparatus. This is done with a fetlock arthrodesis.

Intersesamoidean Ligament Desmitis

The largest ligament in the fetlock joint is the intersesamoidean ligament. It occupies the entire interface between the two sesamoid bones and forms the back of the sagittal groove where the sagittal ridge of the cannon bone interfaces with the palmar/plantar support of the suspensory apparatus. The intersesamoidean ligament is flexible in that it will allow the sesamoid bones to move independently on their respective condyles of the distal cannon bone, but the intersesamoidean ligament is strong enough that the sesamoid bones cannot move abaxially in relation to each other. This ligament carries a high load. As the maximum weight is applied to the fetlock joint, the sesamoid bones translocate distally on the bottom of the cannon bone; the sagittal ridge of the cannon bone drives between the sesamoid bones while the suspensory ligament, which bifurcates into the two branches, pulls abaxially on the sesamoid bones. The resistance to separation be-

Fig. 38. This radiograph shows an avulsion fracture of a portion of the middle distal sesamoidean ligament origin from the base of the sesamoid bone.
between the sesamoid bones requires a large ligament to stabilize the sesamoid bones in the face of virtually the entire weight of the horse, which is placed on the lead limb at high speeds.

When the intersesamoidean ligament or its attachment to the sesamoid bone is injured, it is often extremely painful for the horse. The degree of discomfort exceeds most injuries to the fetlock joint, because there is no opportunity for the horse to guard the ligament against loading. A very small disruption, if it does not structurally weaken the bone/ligament attachment, is tolerated, but in significant intersesamoidean ligament desmitis or injury, the horse will refuse to use the limb because of the pain.

Fortunately, injuries to the intersesamoidean ligament are relatively rare. Intersesamoidean ligament desmitis, in many instances, is initiated by an infection but can be initiated by trauma. Unfortunately, there is no definitive treatment, and the inflammation of the ligamentous insertion is treated similarly to sepsis by attempting to reduce the inflammation and allow the damaged bone to strengthen the intersesamoidean ligament attachment to the parent sesamoid bone. If this is successful, then restoration of function can occur, but the total resolution of inflammation and pain is difficult and is the exception rather than the rule. Debridement of infection in the bones or ligament is helpful but would seem to hold little benefit for traumatic causes.

Palmar/Plantar Joint Capsule Avulsion

Palmar/plantar joint capsule avulsion from its palmar/plantar insertion on the distal cannon bone is seen in two instances. It is seen in the foal when trauma, presumably torsion, pulls the fibers of the fetlock joint capsule from the growing bone. The injury is rarely diagnosed at occurrence, but it is noticed when the bone and joint capsule respond with a marked proliferative response that reattaches the joint capsule to the palmar/plantar cannon bone. Lameness is rarely associated with the injury. Rather, the appearance of the large response, the size of an adult human finger, attached to the palmar/plantar abaxial distal cannon bone is detected. After the joint capsule has reconnected to the cannon bone, the large proliferation gradually remodels and never persists into adulthood. The radiographic change can be quite alarming in appearance and persist for some time in the foal.

In the adult athlete, an injury to the joint capsule insertion onto the palmar/plantar of the cannon bone is often associated with an injury to the intersesamoidean ligament. The injury to the joint capsule is rarely of long-term concern, but the desmitis of the intersesamoidean ligament can cause lameness and affect performance. The injury to the joint capsule can cause considerable fibrosis, but it rarely causes long-term lameness.

Palmar/Plantar P-I Chip Fractures

Palmar/plantar P-I chip fractures occur from P-I either in the plantar aspect of the hind fetlock, which is more common, or in the palmar aspect of the front fetlock. The palmar/plantar P-I chip fracture is an avulsion fracture from P-I at the insertion of the distal sesamoidean ligaments into the first phalanx. The more axial fragments are pulled free by the cruciate distal sesamoidean ligament. They are more common than abaxial fragments and are normally within the fetlock joint under the sesamoid bone. Most abaxial fragments occur at the attachment of the short distal sesamoidean ligaments and at the attachment of the palmar/plantar bundle of the collateral ligament on the first phalanx. Injuries to the short distal sesamoidean ligament insertions on P-I create fractures from the palmar/plantar abaxial aspect of P-I immediately distal to the abaxial aspect of the sesamoid and are similar in pathology to avulsion of the cruciate distal sesamoidean ligaments.

The fragments are always larger than they appear radiographically in the immature horse and occur when a fragment of growing bone is pulled free from the parent bone by the distal sesamoidean ligament attachment to P-I. The fragment normally continues to enlarge as the horse grows until maturity. The origin site heals simultaneously, often filling with bone. This leaves an enlarged fragment and a diminished fracture cavity. The result is a mobile piece of bone between the sesamoid bone and the first phalanx that often articulates with the distal cannon bone during exercise. These avulsion fractures from palmar/plantar P-I are sometimes erroneously called an OCD. The effect on the joint results from the fragment protruding into the joint. This may result in abrasion/ articulation with any of the three bones that interface with the fragment. The degree of pathology caused by this fracture fragment is directly related to size. Small fragments can exist under the sesamoid bone without interfering with sesamoid bone, palmar/plantar first phalanx, or cannon bone, and after covered by fibro-cartilage and isolated from the joint, they are innocuous. However, after a fragment reaches a critical size large enough to interact physically with the base of the sesamoid, the palmar/plantar aspect of the first phalanx, or occasionally, the distal cannon bone during high-speed exercise. Rarely do they cause overt lameness, although this is sometimes possible. In horses that perform at the gallop, most often the complaint is that the horse is uncomfortable and prefers the opposite lead during exercise. The severity of the irritation is most apparent in the Standardbred, where the complaint is frequently heard that the horse will not stay in the proper gait.

If the fragment causes inflammation in the fetlock joint or if it seems likely to be a problem with increased exercise, arthroscopic removal is a simple
and very effective treatment. The arthroscope is used to visualize the fragment proximally, and the fragment is isolated and removed from the palmar/plantar aspect of the first phalanx.\textsuperscript{128,129} Surgical removal is often accompanied by the identification of erosions and softening on the palmar/plantar aspect of P1 or the base of the sesamoid bone where the fragment is interfering with the adjacent bone. These areas of interface result in debris shedding and also can be irritating, because they begin to affect adjacent ligamentous attachments, creating lameness.

After surgical removal, the joint will be normal. The short and cruciate distal sesamoidean ligaments are not large contributors to weight bearing by the suspensory apparatus and have been disabled by the detachment of the fragment from the first phalanx anyway. Therefore, no significant weakening or damage to the fetlock support occurs with an injury to these ligaments. Removal eliminates the irritating fragment from the joint with little or no residual effect.\textsuperscript{127}

The palmar/plantar bundle of the collateral ligament of the fetlock joint can create avulsion fractures of the abaxial aspect of the palmar/plantar P-I (Fig. 39). These fractures involve the wing of the palmar/plantar P-I and are taller than they are wide in the juvenile animal (Fig. 39). In the adult, they are sometimes wider than they are tall. When they occur in the yearling, they are nearly always extra-articular, but in the adult, they normally invade the joint.

In the yearling, the fragments form delayed fibrous unions that eventually mature to a bone union with time. The collateral ligament avulsion fracture that occurs in the immature animal rarely causes lameness after the acute occurrence, unless there is a concurrent intra-articular fragment created by the distal sesamoidean ligament at the time of the wing fracture creation. The intra-articular fragment may need to be removed, but the extra-articular wing fractures, although they are prominent radiographically, rarely cause lameness. With time, the fibrous delayed union, which occurs in the yearling or 2-yr-old horse, matures to a bone union. The fibrous union is perfectly functional and normally requires no treatment.

Wing fracture of P-I in the adult, however, normally involves the fetlock joint and causes lameness (Fig. 40). The smaller fractures that are wider than they are tall are difficult to repair, and surgical removal is often the only option. The prognosis is affected by the amount of joint surface involved, but this is not a critical area for the function of the fetlock joint; removal of all but the very largest of fragments carries a favorable prognosis.

The larger fractures that are taller than they are wide and significantly invade the fetlock joint are best treated with lag-screw fixation to stabilize and compress the fracture, restoring the fetlock joint articular surface and preserving the collateral ligament attachment. With appropriate internal fixation and healing, the prognosis is favorable for athletic use.\textsuperscript{65}

Fig. 39. This radiograph shows the presence of both a plantar P-I chip fracture that is intra-articular (arrowheads) and a plantar abaxial P-I avulsion fracture that is extra-articular (arrow).

Fig. 40. This radiograph shows a plantar P-I fracture caused by a plantar bundle collateral ligament avulsion in an adult (arrow).
Collateral Ligament Rupture/Luxation of the Fetlock Joint
Collateral ligament disruption with an open wound into the joint is a topic for wound management. However, closed collateral ligament rupture is occasionally encountered. The fetlock joint does not require collateral ligaments in the stance phase of the stride. The sagittal ridge, sagittal groove, and sesamoids make the joint stable when it is under load, and although lameness is present, the limb is stable.¹³⁰,¹³¹ The collateral ligaments maintain the normal anatomy when weight is off the limb. Closed rupture can be accompanied by rupture of the joint capsule as well. In mildly unstable limbs, only restriction of exercise is necessary. In most traumatic ruptures, cast immobilization will aid fibrosis to adequately replace the ligament. Cast immobilization for 5–6 wk, depending on the horse’s progress, is normally sufficient. It is preferential to use a cylinder cast to allow motion of the distal phalangeal joints, because this reduces the tendon laxity that is created by enclosing the foot. The prognosis for recovery to athletic activity depends on the degree of proliferation, stiffness, and lameness that results from the injury; however, complete recovery after the repaired fibrotic tissue remodels is possible.¹³¹

Occasionally, the joint capsule will heal and the collateral support will remain weak, so the ligaments require surgical augmentation.¹³² The author has used prosthetic replacement of the collateral ligament with imbrication of the joint capsule over the prosthesis, which is an alternative that has resulted in athletic soundness.

Supracondylar Lysis
Supracondylar lysis has been documented in the literature and is correlated, in the racehorse, with lameness and in the yearling, with reduced performance.⁴¹ Supracondylar lysis is a radiographic finding characterized by palmar/plantar bone absorption and narrowing of the distal aspect of the cannon bone just proximal to the cannon bone articular surface of the fetlock joint when the fetlock joint is viewed in a lateral-to-medial projection (Fig. 41). It happens in the forelimb or hindlimb. The histologic appearance is one of hyperactive bone reabsorption.⁷⁷

This radiographic sign is correlated with a reduction in performance, but it is unlikely that it is the cause. The lysis occurs in an area of the palmar/plantar cannon bone where most of the vascular foramina enter the distal aspect of the cannon bone, but it is not part of the weight-carrying or supporting ligamentous structure of the fetlock joint. This is an area of copious synovial villa that are involved in removing debris and responding to trauma and inflammation in the fetlock joint.

Supracondylar lysis is a non-specific response to any insult that results in the significant and prolonged inflammation seen after many different injuries. Its correlation with reduced performance is probably more related to the primary injury that creates the inflammation and results in the lysis than it is to the supracondylar lysis itself. Anytime the synovial cul-de-sac of the palmar or plantar aspect of the fetlock joint is chronically involved, bone reabsorption and narrowing of the distal aspect of the cannon bone occurs. In the yearling it is often a sign of previous severe inflammation in the fetlock joint from any of the significant neonatal diseases, including infectious arthritis or previous fracture. In the adult, it is usually a sign of chronic inflammation resulting from repetitive cyclic trauma. Therefore, when supracondylar lysis is encountered radiographically, one should be most interested in identifying the lesion(s) or site of inflammation that created or is creating the lysis. Supracondylar lysis occurs only palmarly and plantarly and is sometimes incorrectly diagnosed at the dorso-proximal aspect of the fetlock articular surface. Demineralization dorsally is the result of impact damage from hyperextension and the first phalanx impacting the distal cannon bone with resultant erosion and demineralization or rarely, villonodular synovitis.

The primary disease that is causing the supracondylar lysis determines the prognosis. Many horses with supracondylar lysis can perform well in spite of the radiographic finding if the causative insult has been resolved and the joint has had a chance to recover. If the primary disease persists, the prognosis remains poor.

Fig. 41. This radiograph shows supracondylar lysis (arrowheads) secondary to a chronic OCD of the sagittal ridge and condyles of McIII.
Foal Proximal Sesamoid Bone Fractures

There are few seemingly minor injuries in a foal or adult that have as many potential long-term consequences as a fracture of the sesamoid bone in a foal. Fractures of foal’s sesamoid bones are very common. Perhaps as many as 25% of all foals suffer some type of injury to the sesamoid bone, most of which go undetected or are found coincidentally when examining radiographs for some other reason. Most fractures of the proximal sesamoid bone in the foal, when non-displaced, heal with little consequence to the foal or the eventual adult athlete, but some injuries, particularly if the continuity of the suspensory apparatus is damaged, can disable or limit a horse’s athletic career before the horse is more than a neonate.133

A sesamoid bone by definition forms within a ligament and is normally located at a site where a ligament must change a direction or traverse an angulation. In the horse, the proximal sesamoid bones form within the juvenile suspensory apparatus. In the adult, there is a distinct difference between the very elastic suspensory ligament and the inelastic distal sesamoidean ligaments.134 This change of biomechanics occurs at the sesamoid bones. The sesamoid bones in the neonate are not well developed, and the suspensory ligament/distal sesamoidean ligaments are more contiguous; the ligamentous support rather that the sesamoid bones is the most important in maintaining fetlock joint angulation. In the juvenile, the sesamoid bone becomes a greater component of weight bearing as it becomes stronger, although it can be easily injured before strenuous activity and the strengthening of the bone.

The most common predisposition to injury for a foal’s sesamoid bone is the young mare, often with her first foal, that is recently unburdened of the 200 lb or so of pregnancy and feels the urge to race across the field unimpeded. The neonatal foal is required to keep pace with the mare sprinting across the open field; as the foal fatigues, it tries to maintain contact with the mare, and the juvenile muscles are no longer able to dampen the stress. With fatigue, the foal fractures one or more of the sesamoid bones within the suspensory apparatus.133 It is very common that paired injuries occur, for instance, medial or lateral sesamoids in both forelimbs. Fractures of the hind sesamoids are much less common, because they are less vulnerable to fatigue than the forelimbs. Restriction of the mare to a confined area and gradual increase in exercise as the foal gains strength prevents the mare from exhausting the foal until it gets strong enough to sustain the exercise. It is important to allow foals exercise early to encourage muscular and skeletal development and coordination, but it should be restricted to a relatively confined area, gradually increasing to field exercise as the foal grows.135 If a neonate or its mare requires restriction of exercise to a stall for any reason early in life, graduating the return to exercise is critical to the prevention of sesamoid bone fractures.

Foal apical sesamoid fractures are often non-clinical and often discovered coincidentally if the foal or yearling is being radiographed for some other reason (Fig. 42). Apical sesamoid fractures rarely cause lameness in the foal and generally heal without incident, but the sesamoid bone sometimes forms a non-union, even at this early age, because of the biomechanics that impair bone healing in a sesamoid fracture. These non-union apical fractures need to be dealt with before training, usually by removal, but in the absence of lameness in the foal, they are not an urgent problem.

Mid-sesamoid fractures are less common in the foal than the apical fractures. They almost always heal spontaneously unless markedly displaced. The “tell-tale” result of a previous mid-sesamoid fracture is the elongated sesamoid (Fig. 43). Elongated sesamoids result from a displaced mid-sesamoid or large apical fracture that has healed, filling the fracture gap as the healing process occurs. Elongated sesamoids that are elongated proximally with the base of the sesamoid in the normal position are a predisposition to unsoundness, usually sesamoiditis, and sometimes also re fracture with heavy training. The gap in the sesamoid bone that fills in as the fracture heals disrupts the normal suspensory ligament insertion. The fracture healing with its attendant inflammation and the elongated sesamoid bone, which changes the angle of loading of the suspensory ligament fibers on full weight bearing of the fetlock joint, all predispose to

Fig. 42. This radiograph shows a chronic apical sesamoid fracture (arrow) in a yearling that occurred as a foal.
inferior insertion strength of the suspensory ligament fibers. As more strenuous exercise is undertaken, the suspensory insertion begins to fail, and sesamoiditis/suspensory branch desmitis results. Mid-sesamoid and base sesamoid fractures are usually accompanied by significant lameness initially, but the lameness normally resolves quickly if the structural strength of the suspensory apparatus is not lost (Fig. 43).

Abaxial and base sesamoid fractures with any displacement severely damage the foal’s athletic career. The abaxial sesamoid fracture detaches some or all of the suspensory ligament from the sesamoid bone, and the basilar sesamoid fragment detaches some or all of the distal sesamoidean ligaments from the sesamoid bone (Fig. 44). Either situation is a severe threat to the foal’s athletic career.

Treatment of abaxial or base sesamoid fractures in a foal is difficult; removal is not an option, because it will assure disability of the suspensory apparatus. Reconstruction would theoretically be desirable, but surgical implants have little to purchase in the fractured fragment of the sesamoid to reconstruct the sesamoid bone. The detached fragment is principally ligamentous with only a small shell of bone. Normally, the foal must heal the fracture on its own.

If the fracture fragment separates from the parent bone and loses contact, a fibrous union develops, and the foal’s athletic career is ended. If the base sesamoid heals without proximal displacement of the sesamoid bone, base sesamoid fractures will likely be functionally sound for most athletic pursuits. Abaxial sesamoid fractures are usually more devastating, because an abaxial injury normally pulls the entire suspensory ligament insertion away from the parent bone, detaching the support from the sesamoid. These can heal, but rarely do they heal sufficiently to allow athletic activity. If they do heal, they are accompanied by marked sesamoiditis, and the suspensory ligament insertion is rarely sound enough for high-speed exercise.

In general, sesamoid injuries in the foal result in inflammation during the process of healing that causes sesamoiditis, as defined by the enlarged vascular canals that are a marker for permanent damage to the suspensory apparatus and compromise the function of the suspensory apparatus for the rest of the foals life. A foal with a sesamoid fracture needs to be restricted until lameness and clinical swelling have subsided. After the lameness and clinical swelling have resolved, gradually increasing exercise is indicated. Normal care can be initiated if no distraction and no lameness or pain on palpation persists. If the foal is lame or the sesamoid fracture is distracted, then absolute confinement is imperative or loss of all chance of athletic career will occur. After the bone has fully united, then gradually increasing exercise is appropriate. If the sesamoid fracture heals without change in shape of the sesamoid and without development of the enlarged vascular canals, the sesamoid bone will be normal for future performance. If elongation of the sesamoid to a significant degree in the proximal direction results

Fig. 43. This radiograph shows an elongated lateral sesamoid (bracket) as a result of a previous healed fracture.

Fig. 44. This radiograph shows a typical base sesamoid fracture in a foal.
from the injury or if enlargement of the vascular canals occurs as a marker of significant inflammation at the suspensory/sesamoid insertion, then the foal’s future athletic career will be compromised.\textsuperscript{39}

The worst mistake that can be made with a foal with a sesamoid fracture is to support the limb in a cast or heavy bandage. The flexor tendons support the limb when the suspensory apparatus strength is compromised by a sesamoid fracture. If external limb support is used in foals, the flexor tendons weaken. On removal of the external support, the limb no longer has any structural support from the flexor tendons or the suspensory apparatus and the fetlock joint collapses, which further deters the sesamoid fracture and risks total collapse of the limb. Therefore, sesamoid fractures are best left with the limb unsupported. If the sesamoid bone injury is biaxial or the collapse is present at the time of injury, the prognosis is poor and hope for survival would require fusion of the fetlock joint.

Sesamoid Fractures in the Mature Horse
Fractures of the sesamoid bone in the adult are a somewhat different topic than fractures in the foal. Although the horse is still growing, sesamoid fractures that stay in close apposition have a reasonably good chance of uniting. In the adult, after growth has stopped, sesamoid fractures, other than very fine fissures, almost never heal. These adult fractures do not heal, because they are exposed to the conditions favoring non-union of fractures. Tension, lack of a periosteal blood supply, high motion, and continuous cyclic loading all mitigate against fracture healing, and all are present in a fracture of the proximal sesamoid bone in the horse. Therefore, when active growth ceases, sesamoid fractures rarely heal with a bone union, and a chronic non-union develops with its attendant inflammation and weak fibrous union.

The two fragments of a fractured sesamoid bone continually attempt to mount a secondary bone healing response. This continual reaction to the instability of the fracture and a progressive attempt to bridge the fracture are parts of the pathology that predisposes to lameness in a sesamoid fracture. Attempts at fracture healing in a sesamoid, just like with a fragment in a joint, fail, because the strain is too large across the fracture gap. The bone’s response is to open the fracture gap by demineralization of the fracture interface to reduce the shear stress. This progressive loss of bone further weakens the injured sesamoid bone.

Consequently, a weakened, inflamed, and demineralized sesamoid bone suffers further progressive avulsion of the ligamentous insertions. After ligamentous attachments are weakened and fail, they can never be reconstructed to the strength of the original attachment. So, fractures of the sesamoid bone are particularly troublesome; they not only disable portions of the suspensory apparatus by detaching the fracture fragment from the parent bone, but additionally, they progressively weaken the parent bone, predisposing to loss of additional suspensory or sesamoidean ligament attachment during the healing process.

To neutralize sesamoid fracture’s natural attempts to heal, one must either negate the motion that occurs between the two fragments of bone to facilitate healing or eliminate one of the fragments of bone to stop the healing process. With small fragments, the treatment of choice is to remove the fractured fragment and stop the healing response by the parent bone to preserve the remaining normal ligamentous insertions. With large fractures, such as mid-sesamoid fractures where elimination of one of the fragments of the bone would compromise the integrity of the suspensory apparatus, reconstruction of the bone is the only alternative.

Axial Sesamoid Fractures
Axial sesamoid fractures are rare and are generally seen in the presence of a distal cannon bone condylar fracture.\textsuperscript{136} They occur when the normal joint mechanics are disabled by the unstable condylar fracture. As weight is applied and the displaced condyle is pushed dorsally, the ipsilateral sesamoid is displaced dorsally and the intersesamoidean ligament is bent over the disrupted interface of the parent cannon bone at the fracture site. This results in fracture of the axial aspect of the sesamoid bone that articulates with the injured condyle.

There is no treatment for this fracture; it is a career-ending injury, even if the displaced condylar fracture is reconstructed and heals perfectly, because the intersesamoidean ligament injury that results from an axial sesamoid fracture causes chronic lameness and does not heal.\textsuperscript{136} Occasionally, axial sesamoid fractures will be seen spontaneously without a predisposing condylar fracture (Fig. 45).

Reconstruction of the condylar fracture results in a breeding animal, but the lameness caused by the axial sesamoid fracture in a broodmare or stallion takes as long as 1 yr to resolve whether or not from a predisposing condylar fracture or spontaneous fracture (Fig. 45). These fractures can be extremely troublesome; the horse walks reasonably well after the condylar fracture has been replaced and stabilized surgically, but it is uncomfortable for the horse to stand with tension on the axial sesamoid fracture with or without a condylar fracture. Some horses will be lame for many months. Stall rest is more painful than walking, so paddock exercise should be maintained or initiated as soon as the condylar fracture repair will allow. The long-term prognosis for a breeding animal is favorable.

Apical Sesamoid Fracture
Apical sesamoid fractures from one-fourth to one-third of the volume of the bone are common fractures in all breeds of racehorses (Fig. 46).\textsuperscript{137} They are infrequent in horses of lesser use levels. The explanation for this was elucidated in research,
which found that training caused the suspensory ligament to become stronger to a greater degree and at a more rapid rate than the sesamoid bone, making the sesamoid bone the weak link in the high-speed athlete.\textsuperscript{134}

Apical Sesamoid Fractures Occur Primarily in the Racing Breeds

It has been postulated that an area of differential remodeling located at the proximal one-third to one-fourth of the sesamoid bone predisposes it to fracture, although there is information to both support and refute this concept.\textsuperscript{138} There is good information that the location of the fracture is relatively consistent, suggesting that an apical sesamoid fracture is not a haphazard occurrence.

Acutely, fractures of the sesamoid bone cause lameness, which gradually abates and even becomes tolerable in some horses. Eventually, in high-level athletes, the mobile apical fracture begins to produce the inflammation caused by the healing response of the parent bone and begins to weaken the remaining normal suspensory ligament fibers that insert into the parent sesamoid bone. It is the preservation of these fibers that is the goal of treatment of an apical sesamoid fracture. After the sesamoid fracture occurs, it is the remaining normal suspensory ligament that supports the horse’s athletic activity, because the suspensory ligament fibers that are attached to the fracture fragment are detached from the distal aspect of the suspensory apparatus. Sesamoid fractures are a disease of the suspensory apparatus and not the fetlock joint itself, because they rarely lead to any significant degeneration of the joint; rather, they cause lameness through their effects on the suspensory apparatus.

Because an apical sesamoid fracture in a mature horse will never heal, surgical removal is the treatment of choice and in fact, was the first arthritic surgery described in the horse.\textsuperscript{137,139} Surgical removal preserves the normal parent sesamoid bone as well as the remaining suspensory ligament attachment from progressive fiber loss caused by the healing response provoked by the apical sesamoid fracture.

Although removal of apical sesamoid bone fractures through an open incision was a common and successful surgical procedure for some period of time, the preferred treatment is now arthroscopic removal.\textsuperscript{140} Currently, the open incision has been replaced by arthroscopic extraction of the apical fragments in almost all instances because of the reduced morbidity to the fetlock joint. The prognosis has been well defined in both the Thoroughbred and the Standardbred, and it is favorable for apical sesamoid fractures as a group.\textsuperscript{140,141} Further refinement of the prognosis was recently published in a paper that compared the different locations within the horse with the prognosis.\textsuperscript{142,143} Apical sesamoid fractures of lateral sesamoid bones in the forelimbs and all four sesamoids in the hindlimbs routinely have an excellent prognosis for returning to pre-injury level in adults and achieving performance levels comparable with uninjured siblings in juvenile horses, if they are removed arthroscopi-

![Fig. 45. This radiograph shows a spontaneous axial sesamoid fracture without a predisposing condylar fracture (arrows).](image1)

![Fig. 46. This radiograph shows an apical sesamoid fracture (arrow).](image2)
cally. But, a medial apical sesamoid fracture of a forelimb, in both adult and juvenile horses, carries one-half the prognosis for a hindlimb apical sesamoid fracture or a lateral sesamoid fracture in a forelimb. The marked difference, present both in the unraced and raced individuals, seems to be related to the anatomy and the importance of the medial sesamoid bone in the forelimb.

Careful examination of each individual apical sesamoid fracture can further refine the individual injury prognosis. As a rule of thumb, the prognosis declines as the suspensory ligament involvement increases. Suspensory ligament injury occurs from the fracture and from concurrent suspensory ligament desmitis. Concurrent suspensory ligament desmitis decreased the prognosis. So, refinement of the prognosis for an apical sesamoid fracture can be done using radiographic estimates or ultrasound measurement of the suspensory ligament involvement.

Post-operative rehabilitation of the horse with an apical sesamoid fracture is routinely assessed by the examination of the suspensory ligament before resumption of training is desired. In the author’s hospital, horses with apical sesamoid fractures and no suspensory ligament damage are examined at 4 wk post-surgery with an ultrasound exam, and if the suspensory ligament is not inflamed other than where the fragment was removed, exercise is reinitiated.

Articular Abaxial Sesamoid Fractures
Articular abaxial sesamoid fractures are generally similar to apical sesamoid fractures in their assessment and treatment (Fig. 47). They occur within the heart of the suspensory ligament insertion, so a smaller fragment of bone damages a larger amount of suspensory ligament insertion than with an apical sesamoid fracture.

The prognosis is determined by the amount of suspensory ligament loss created by the sesamoid fracture and any attendant suspensory ligament injury. Frequently, there is significant suspensory ligament damage, and the guarded prognosis reflects this difficult concurrent injury. Arthroscopic removal is similar to removal of the apical sesamoid fracture in surgical technique and aftercare.

Mid-Sesamoid Fracture
Mid-sesamoid fractures in the mature horse are a difficult problem, because they totally detach one-half of the suspensory apparatus from the distal sesamoidean ligaments (Fig. 48). Removal of a fragment nearing one-half of the mass of the sesamoid bone simply assures no hope of reconnection between the components of the suspensory apparatus. Therefore, the only potential salvage for athletic activity is to attempt fixation and achieve reunion of the two halves of the sesamoid bone. Without fixation, displaced mid-sesamoid fractures have no chance to heal after the horse is mature.
oid bone, making it likely that the structural diminution and diminished strength predispose the bone to fracture. However, many sesamoid bone fractures have no apparent predisposition for their occurrence, and the explanation for failure of a radiographically normal sesamoid bone is unclear.111,145 The best information shows that continuous high-level stress over a long period of time is highly correlated to the occurrence of mid-body sesamoid fractures, but why a particular sesamoid fracture occurs is still unclear.146

The treatment through internal fixation can be done in two principal ways: the use of screws or wires, one or multiple, to adapt the bone fragments or to reconnect the bone.147 In most instances, a bone graft is added to the fixation to stimulate repair because of the poor biomechanics of sesamoid fracture healing. Lag-screw fixation can take place from the apex, the base, or the abaxial surface, depending on the configuration of the fracture.148 The lag screws are inserted in normal A-O fashion, and it is the author’s preference to coapt the sesamoid bone, insert the screw, and then subsequently loosen the screw to insert the bone graft, although it can be done in any order.

The surgical procedure is technically challenging because of the interposition of the foot into the location where one would like to orient the drill to create the hole; the fracture is best reduced in flexion, but the surgery is easiest with extension of the fetlock joint. In addition, the healing process is fraught with many potential difficulties. In addition to the limited bone to insert the fixation, the bone heals slowly, and if the healing process is accompanied by significant sesamoiditis, as it usually is, even a perfect reduction and healing of the sesamoid bone may not result in a sound horse. Wire fixation can similarly adapt the two portions of the sesamoid bone like the screw, and a wire is easier to insert but has been shown to be less likely to result in an athlete than lag-screw fixation.148,149

The prognosis for successful treatment of mid-sesamoid fractures is limited by the technical difficulty and the probability of subsequent sesamoiditis. Repair is normally attempted only in horses that have no use other than their athlete career. Although many horses can return to racing, it is difficult to keep them sound because of the sesamoiditis that is caused by the fracture and the healing process.148

Base Sesamoid Fractures
There are two distinct categories of base sesamoid fractures. The most common category is the base sesamoid fracture that is predisposed by the change in shape of the distal metacarpus caused by palmar distal cannon bone degeneration. The flattening of the distal palmar aspect of the metacarpus and the alteration of the congruency of the articular surface of the sesamoid bone with the distal cannon bone result in abnormal stress concentration and fracture of the base of the sesamoid. These fractures often involve the entire width of the base of the sesamoid, are normally a thin portion of the base of the sesamoid bone, and frequently are comminuted.150,151

Treatment is unrewarding for these secondary fractures of the base of the sesamoid bone. They are impossible to fix, and removal detaches significant sesamoidean ligament insertion. However, more importantly, even if the fragment is small, surgical treatment of the base sesamoid fracture is treating only a result, not the primary disease, which is the ongoing degeneration of the palmar aspect of the metacarpus. This degenerative change is a career-ending injury in the horse, so surgical treatment is not normally elected.

Small base sesamoid fractures that are not accompanied by palmar flattening of the distal aspect of the metacarpus can be treated with arthroscopic removal (Fig. 49).150,151 It is preferable to have less than one-half of the width of the sesamoid bone involved in one of the two dimensions, dorsal to palmar/plantar or medial to lateral, to be successful. If the middle portion of the sesamoid bone is involved in the base sesamoid fracture, then the important middle distal sesamoidean ligament has become involved, which decreases the prognosis. However, fractures of the base of the sesamoid that involve only the dorsal margin and therefore, the short or cruciate sesamoidean ligaments have a favorable prognosis with removal.150,151

Arthroscopic removal is similar to the surgical procedure used for the removal of fracture fragments of the palmar/plantar aspect of the first phalanx with two arthroscopic portals and dissection to free the fragment before removal.
accompanied by the lucency and demineralization that further compromises the strength of the suspensory ligament/sesamoid bone insertion. If allowed to continue, the horse seldom stays sound, so elective removal before permanent disability is a critical decision.

These fracture fragments involve the suspensory ligament insertion but are not directly accessible for arthroscopic removal. Therefore, some suspensory ligament must be sacrificed if the fragment is to be approached. The shortest distance to the fragment with the least suspensory disruption is the approach of choice. In some instances, the arthroscope can be used to make an incision in the suspensory ligament through the joint and access the fracture fragment from that aspect; in other instances, direct access of the fracture fragment through the abaxial surface of the suspensory ligament does the least damage.

Non-Articular Abaxial Sesamoid Fragments
Abaxial sesamoid fractures that are non-articular present a somewhat different problem (Fig. 50). Large fragments that detach large numbers of suspensory insertion fibers are not amenable to surgical resolution. However, horses with small fragments that detach only a small portion of the suspensory ligament insertion may benefit from fragment removal, because the unstable fragment provokes the same healing response as does an articular fracture fragment that involves the abaxial aspect of the bone, which further damages the suspensory ligament. The healing process continually inflames and softens the parent sesamoid, allowing progressive loss of more suspensory ligament fibers. Some of the smaller abaxial sesamoid fragments within the suspensory ligament are tolerated without a response by the parent bone. If the parent bone is not trying to heal the fragment, then it is likely best left undisturbed. But, most unstable fragments contribute to progressive loss of suspensory ligament anchorage and eventually, permanent lameness by provoking a healing response by the parent bone. These fragments should be considered for removal.

One must be cautious and use judgment in recommending removal, because if the sesamoid fragment is quiescent and not stimulating a response by the parent bone, the surgical invasion that sacrifices suspensory attachment may be worse than the disease. So, judgment is important, but in most instances when high speed exercise occurs with a sesamoid fragment in place, the sesamoid bone responds and attempts to heal the fragment. This is accompanied by the lucency and demineralization

References

Fig. 50. This radiograph shows a non-articular abaxial sesamoid fracture (arrow) that creates a danger of progressive loss of the suspensory insertion into the parent bone.


42. McIlwraith C. From arthroscopy to gene therapy-30 years of looking in joints. 51st American Association of Equine Practitioners Annual Convention 2005;65–113.


