EQUINE FOOT WOUNDS

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Wounds of the horses foot are common, and many are simple and heal without untoward consequences. Unfortunately, others may cause lasting changes in hoof function, and sometimes even be life threatening. The term foot describes the hoof and all structures contained within it. It is part of the appendicular skeleton and as such most of the same elements, i.e. bones, joints, tendons and ligaments encased by the integument. What makes the foot different is its integument the hoof because of its specialized function, bearing weight, as well as the other functions of skin. This function necessitates a specialized structure, growth pattern and attachment to the rigid structures of the appendicular skeleton, all of which are important considerations in the treatment of foot wounds and restoration of normal function.

The management of horses feet spans both the farriery and veterinary professions, and this is no less true for foot wounds than any other problem. The diagnosis, and medical and surgical treatment of foot wounds lies within the province of the veterinarian. However, a farrier is commonly the first professional called to examine a horse with a foot problem, and therefore, needs to recognize injuries that need veterinary attention. Also, during the treatment of these wounds farriers are commonly called upon to design and apply surgical shoes, and long after medical and surgical management of the injury has ceased, it is the farrier who must often ensure that optimal function of deformed feet is maintained by careful trimming and shoeing. A basic understanding of the types of foot injury that occur and how they heal is essential for obtaining the optimal result and providing the owner the most accurate prognosis.

THE NATURE OF FOOT WOUNDS.

The very position of the foot at the end of the limb in repeated contact with the ground places it in constant jeopardy due to trauma of sharp objects, despite its admirable design.

Superficial injuries to the keratinized layers of the epidermis seldom incite an inflammatory response or cause infection and unless they cause instability, resolve uneventfully through the natural process of hoof replacement. Injuries that penetrate the basal layers of the epidermis, dermis and deeper are potentially much more serious. The most common wounds are punctures and lacerations. Puncture wounds occur most often due to a nail in the sole and frog.

They are characterized by a small entry wound that may be hard to find once the nail has gone, particularly in the frog and sulci where the rubbery nature of the hoof causes the margins of the wound to spring back together. This type of wound gives no indication of the depth or direction of the injury and therefore little indication of which structures are damaged. They are often very painful.

Lacerations of the hoof more commonly involve the wall and adjacent skin than the sole. They are caused by tearing of tissues, and in skin this is seen as wounds with ragged margins and varying depth, though usually fairly shallow an may be associated with loss of the integument. The damaged tissues are well exposed and in contrast to puncture wounds the superficial appearance of the wound exaggerates its seriousness, and are not necessarily as painful as expected. Because of the semirigid nature of the hoof capsule, a flap of hoof is torn away from the laminae. This flap may involve only the laminar portion of the wall, or may also include the coronary band and the skin proximal to the coronary band. In incomplete avulsions, the flap is usually hinged along one or sometimes 2 borders. In complete avulsions the flap has become completely detached and the wound must heal by secondary intention. Incomplete avulsions that do not involve the coronary Band are best resected and the defect allowed to heal by secondary intention. Incomplete avulsions that do not involve the coronary band are best resected and left to heal by secondary intention. Incomplete avulsions that involve the coronary band have

Accidental incised wounds that result from a cut by a piece of sheet metal or cut glass (as opposed to deliberately incised wounds made with a scalpel) without avulsion of the surrounding integument are rare. The sides of the wound do not separate; unlike puncture wounds the entry wound is readily visible and the direction taken by the foreign object apparent, therefore, it is easier to predict what structure are involved compared to puncture wound, but harder than with lacerations.
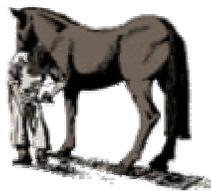
Subsolar abscesses from an interesting case. There is no break in the keratinized epithelium except sometimes a small crack. When the keratinized epithelium is pared away to expose the abscess cavity, it is usual to find a thin soft white layer of epithelium that covers the deep surface of the wound. The presence of a complete layer of epithelium on the deep surface of the wound regardless of the size of the defect and the duration of the abscess implies that the abscess is intradermal and not subdermal because complete epithelial defects must epithelialize from their margins (as explained later), which takes several days and in the meantime the defect would contain granulation tissue. Therefore, it is likely that exudate from the abscess dissects the keratinized epithelium away from the basal layers to form a false sole. Occasionally an abscess is subepidermal as evidenced by the presence of granulation tissue on the deep surface, or necrotic dermis which will be replaced by epidermis.

WOUND HEALING: A GENERAL DESCRIPTION

Unfortunately, as far as I am aware, there is no original research on the healing of foot wounds. Therefore, our knowledge of this process is based on clinical experience and extrapolation from research on the healing of skin wounds.   Consequently, it is best to examine the way a wound through the skin heals, and then consider the differences between this and wounds through the hoof.

Classically wound healing is divided into 4 stages: inflammatory, debridement, repair, and maturation. In the inflammatory phase, the small vessels transiently constrict to limit hemorrhage is rapidly followed by vasodilation. The wound is infiltrated by a plasma-like fluid and white cells that form a clot. which form a protective barrier over the wound surface. that later becomes a scab.

Debridement involves the removal of all dead tissue, blood clots, and foreign material from the wound surface. This occurs by phagocytosis by specialized white cells called macrophages, sloughing from the exterior surface of the wound, or surgical debridement.

Repair occurs through the action of 3 processes: epithelialization, formation of granulation tissue, and contraction. Epithelialization occurs through the proliferation of epithelial cells at the margins of the wound and their subsequent migration across the surface of healthy dermis and granulation tissue. This migrating cells form a very thin layer of epithelium across the surface o the wound that increases in thickness once the movement of the basal cells is arrested. Fibroblasts proliferate at the from all healthy tissues in the depth of the wound and begin to fill the defect. The fibroblasts are responsible for the laying down of collagen which provides strength to the wound repair. Newly formed capillary loops closely follow the advancing border of fibroblasts. Together, the fibroblasts and capillaries form granulation tissue. All healthy tissues are capable of forming granulation tissue, though dermal tissues granulate faster than bone, ligament and tendon. Concurrently, where elasticity of the wound margins allow, contraction of the wound surface area occurs through active development of tension in the wound margins.

Wound maturation proceeds once the wound has completely granulated and epithelialized, there is a gradual loss decrease in vascularity and cellularity of the wound. At the same time the scar tissue increases in strength due to reorganization and cross linking of the collagen.

This description implies that a wound goes through chronologically distinct phases, whereas the process is of course a continuum. Not only that, but different areas within a wound are likely to be at different stages at any one time.

FACTORS EFFECTING WOUND HEALING

Many factors effect the rate at which wound heal including the systemic status of the animal, the local wound environment, systemic medications, and topical medications and bandaging. Discussion of the effects of systemic status and nutrition on wound healing are beyond the scope of this manuscript.

Local factors that impede wound healing include the presence of necrotic tissue, foreign material, infection, blood clots, tissue edema, poor blood supply, dessication, movement and low temperatures. Necrotic material and foreign matter must both be harbor bacteria and allow infection to persist, and present a physical obstacle to wound repair. Infection causes persistence of inflammation and further tissue damage which prolongs debridement and delays granulation and epithelialization; the accompanying exudate is a physical barrier to wound healing.

Topical medications are usually aimed at controlling infection. Both the product and formulation are important in determining the effect of any medication on wound healing. In brief, solutions appear to be more beneficial than ointments and creams. Topical antiseptics are beneficial in low concentrations, e.g. 0.1-0.2% povidone iodine and 0.05% calcium gluconate, but are harmful in higher concentrations. There appears to be no place for tinctures of iodine or formalin in the treatment of foot wounds, and while they may appear to be beneficial in treating superficial infections of the epidermis at best, they are a poor substitute for adequate debridement. They are severely destructive when placed in direct contact with living tissues.

The nature of any surface dressing is also important. Adherent dressings, such as cotton gauze, lift adherent tissue off the surface of a wound when they are removed; this effect is beneficial when débriding a wound but deleterious to a healthy granulating and epithelializing surface. Nonadherent dressings are subdivided into occlusive and non-occlusive. Non-occlusive dressings allow exudate to pass through, but occlusive dressings cause retention of exudate at the wound surface with deleterious results. Even within the different types of non-occlusive dressings there are differences. Petrolatum impregnated gauzes promote wound contraction and granulation tissue formation, but inhibit epithelialization. In contrast, Telfa pads do not impede epithelialization

Bandages assist wound healing through several other effects other than the obvious protection from further mechanical injury. At the same time as wicking exudate away from the wound surface, they prevent dessication. Accumulation of exudate effects a wound negatively by macerating tissue surfaces as well as functioning as a barrier between wound surfaces. Dessication of the epithelial margins causes scab formation that inhibits epithelial migration across the wound. By directly exerting pressure on the wound surface, a bandage may help to control tissue edema. Also, a bandage may retain heat at the wound surface, enhancing the rate of wound healing.

WOUND HEALING: THE FOOT AS A SPECIAL CASE

The similarities between the healing of foot wounds and wounds elsewhere on the limbs far outweigh the differences, but it is inevitably the differences that attract attention. The most noticeable differences between healing of skin an hoof defects relate to the differences in physical characteristics and pattern of normal replacement. Also, the position of the foot at the extremity means that there are less adjacent soft tissues compared to the more proximal limb, it is cooler than the more proximal limb and it is more prone to severe contamination.

The physical properties of the hoof and the nature of the injuries that occur make suture closure of wound less practical compared to skin wound. Fortunately, this is seldom warranted except for injuries of the coronary band.

The rigid structure of the hoof wall and sole cause it to act as a cast around the structures it encompasses. Like a cast over a wound, this is both good and bad; a cast stabilizes and protects the tissues within, but does not allow observation of the wound surface underneath it, and it impedes movement of air an fluid to and from the wound surface.. When the hoof is punctured or lacerated, the margins of the wound do not retract. Similarly, the rigid nature of the hoof prevents contraction of the wound margins of defect during the repair process. In the frog, the spongy nature of the epithelium actually causes the edges of small wounds to spring back to close over the entry wound. Because the hoof does not expand, the inflammatory response causes the pressure to increase within the foot, an inherently painful process. Also, the drainage of exudate and the sloughing of necrotic tissue is inhibited. Hence the need to expose many wounds by removal of at least the stratum medium of the epidermis.

However, the cast like nature of the hoof is not all bad in relation to wound healing. If wound debridement and control of infection can be established leaving the hoof wall intact, the hoof constrains the growth of granulation tissue until is epithelialized, preventing exuberant granulation tissue (proud flesh) from developing. Having said this, in my experience the development of proud flesh on the foot is quite rare even when large portions of the wall are missing, though it is common on the heel bulbs and pastern. When exuberant granulation tissue does occur, it is most common on the sole of laminitic horses following prolapse of the distal phalanx. One explanation for this is excessive pressure at the dorsal margin of the distal phalanx preventing epithelialization.

Another reason for maintaining the structural integrity of the wall as much as possible during the exploration of foot wound is the preservation of stability of the hoof. Motion of any wound impedes healing, but this is perhaps particularly important in the hoof because hoof defects that parallel the line of wall growth may persist as hoof cracks.

The epithelium and dermis of the skin are relatively uniform in structure wherever they are on the limb and hence the process of epithelialization proceeds fairly uniformly. In contrast, the subspecialization of the epithelium and dermis of the foot to form the coronary band, the wall, the sole and the frog means that the return of full thickness epithelium over the hoof is not necessarily a uniform process.

Granulation tissue formation and epithelial cell migration over the surface of the defect in these areas proceed as they would elsewhere on the limb, but the manner in which the epidermis returns to normal thickness varies with location because of the specialized way in which the hoof wall and sole are replace in the process of normal growth. Defects in the sole increase in thickness by proliferation locally. In contrast, once laminar defects have epithelialized and the initial epithelialization has taken place, there is limited local proliferation and keratinization of the epithelium, but full thickness replacement of the stratum medium is derived from the coronary epithelium as it moves from coronary band distally.

Some hoof avulsions start in the laminar region, span the coronary band, and extend up into the skin of the pastern region. Epithelium migrates from all three areas to fill the defect. Therefore what type of integument results over the surface of the wound depends on where the epithelium migrated from. Therefore, epidermis derived form the skin may replace part of the defect below the coronary band, or hoof forming epithelium may appear proximal to the coronary band. Of particular importance is the nature of the epithelial tissue replacing the coronary band because this determines the structure of new hoof wall growth. It should be borne in mind that surgically created wound involving the coronary band heal in the same manner as accidental wounds, hence the extreme reluctance of most surgeons to disrupt the coronary band to gain access to deeper structures of the foot. Separation of the hoof wall at the coronary band caused by a gravel should not be confused with a complete epithelial defect; usually in these circumstances the keratinized epithelium has separated from the germinal epithelium. Hence the defect grows out and is replaced by normal wall.

It is interesting to note that the distal phalanx appears to respond differently to injury compared to the other phalanges and the metacarpus. When part of the distal phalanx has débrided, the defect does not become mineralized radiographically, nor does the distal phalanx mount an aggressive periosteal response on the adjacent surfaces as seen elsewhere. In fact, avulsion wounds that remove large portions of the wall adjacent to the distal phalanx, but do not damage the bone itself cause considerable bone demineralization. Despite this loss or demineralization of coffin bone, the future function of the foot appears to depend more upon the structural integrity of the hoof than the radiographic appearance of the distal phalanx..

The restoration of function of all injured body parts including the integument is the ultimate goal of any reparative process, but given the greater expectations of the hoof compared to skin makes it seem all the more important.

DEEP DIGITAL INFECTION

Infection within the horses hoof is a common cause of lameness. Fortunately, most are subsolar abscesses, familiar to both farriers and veterinarians. Subsolar abscesses usually do not extend deeper than the dermis and respond favourably to treatment. In contrast, deep digital infections, though less common, extend deeper than the dermis and may have far more devastating consequences. Horses with both superficial and deep digital infections are often severely lame upon presentation. Unfortunately the depth of the injury often is not appreciated upon initial examination or the significance is underestimated. Therefore deep digital infections are often initially treated as superficial infection; they are not recognised for what they are until they fail to respond to "normal" therapy. Consequently appropriate therapy is often delayed. It is important that both farrier and veterinarian have a thorough understanding of both deep and superficial infections. However, farriers are often the first to examine a horse with a suspected subsolar abscess and therefore act as a sentinel that all is not as it seems.

Deep digital infections occur most commonly from direct introduction of bacteria into the deeper structures of the foot following puncture wounds, but may also follow lacerations and extension of infection from subsolar abscesses. A detailed knowledge of digital anatomy is essential in predicting the potential involvement of tissues based on the location of the entry wound, the direction and depth of penetration of the foreign object, the likelihood of infection spreading from one structure to another, and the possibility of iatrogenic damage occurring during treatment. The most commonly used diagram to demonstrate the relationship of different structures to each other in the hoof is a midsaggital section. It is important to remember that these relationships change away from the midline.

Of particular concern are the distal phalanx, the navicular bone, the navicular bursa, the collateral cartilages and the distal interphalangeal joint as these are the structures associated with a guarded to poor prognosis when infected because the infection is either refractory to treatment or life threatening. Of particular importance is the close proximity of the navicular bone, navicular bursa, distal interphalangeal joint and deep digital flexor tendon deep to the middle third of the frog. This means that more than one structure may be involved because of the original injury. Alternatively spread of infection from one structure to another may occur at a later date by natural extension of the disease process or by iatrogenic spread from surgical interference. Also of importance is the close proximity of the collateral cartilages to the palmar reflections of the distal interphalangeal joint capsule. Hence the need for extreme caution when exploring and debriding in these areas.

DIAGNOSIS

A good history is invaluable in evaluating any disease process. It is important to know the speed on onset, the duration and progression of the problem. Typically deep digital infections are associated with a rapid or sudden onset of a severe lameness that shows no signs of improvement. However, removal of a foreign body from a wound may cause a transient dramatic improvement only to be followed by recurrence of severe symptoms as the infection becomes established in 24 - 72 hours. Knowledge of the nature of the original injury may speed up the diagnosis; lacerations are usually obvious but puncture wounds may be difficult to locate if not seen at the time of injury. Prior treatment and its effect, if any, should be determined. Deep digital infections have often been treated as subsolar abscesses and failed to respond.

On presentation the horse is usually extremely lame in the affected limb. Examination of the hoof may indicate increased temperature. The location of any swelling proximal to the coronary band should be noted. Pain may be evident with systematic application of hoof testers. Lacerations are usually obvious but paring out the sole and sulci is often required to identify puncture sites seen as areas of discoloration. Draining tracts may be apparent on careful visual inspection or palpation of the coronary band.

Wounds and draining tracts require exploration to identify structures that may be damaged. This is greatly facilitated by local anesthesia. After aseptic preparation lacerations can be explored digitally, punctures should be cautiously explored with a sterile probe. If infection of the distal interphalangeal joint or navicular bursa is suspected, aspiration of synovial fluid from a site unaffected by the infectious process distant to the injury is indicated; cytolology may provide definitive evidence of inflammation and strong support of infection. Following aspiration of synovial fluid, distension of the synovial space with saline may demonstrate communication between the synovial cavity and a wound.

Radiography is an important adjunct to the diagnostic examination and a useful technique in monitoring progression of the disease. If a foreign body is present it is important to radiograph the foot prior to its removal because this can accurately demonstrate the path taken by the object. While fractures of the distal phalanx or navicular bone might certainly explain the severe lameness, they may or may not be related to a puncture injury as they may have been pre-existing. Radiographic evidence of lysis of the navicular bone or distal phalanx is often indicative of osteomyelitis.

Sequestra, pieces of dead bone that act as a foreign body, are suggested by the presence of an isolated fragment of bone that shows no periosteal response surrounded by a zone of osteolysis. Sequestra around the margin of the third phalanx are usually obvious but sequestra within the saucer shaped cavity of the solar surface may be occult. Septic arthritis of the distal interphalangeal joint may initially show no radiographic abnormalities, an increased joint space due to an increased volume of synovial fluid, or a decreased joint space due to loss of articular cartilage; with time, lysis of subchondral bone and periarticular new bone are apparent. Subluxation of the distal interphalangeal joint with dorsal displacement of the distal phalanx indicates rupture of the deep digital flexor tendon. Proximal displacement of the navicular bone indicates rupture of the impar ligament. Gas shadows in the soft tissues most likely indicate the presence of a cavity connected to atmospheric air but may be caused by gas producing organisms. As only osseous tissues are radiodense, soft tissue injuries are not apparent. To assist in delineating tracts radiodense probes or liquids can be inserted or instilled into tracts and when 2 radiographic projections at 90 degrees to each other are used, the site of the tract or cavity can be deduced. Liquid contrast media are better able to delineate the margins of a cavity and can go around corners, but are not as radiodense as metal probes.

Bacterial cultures should be taken from tracts after aseptic preparation and prior to exploration, and from synovial aspirates.

GENERAL THERAPEUTIC PRINCIPLES

Symptomatic therapy, including pain control with phenylbutazone and broad spectrum antibiotics should be started immediately. Thereafter, treatment of horses with deep digital infections is best performed in an equine hospital and not on the farm. An accurate assessment of the injury is imperative before surgical therapy can be planned. Good restraint is advisable; the author prefers to work on difficult foot wounds with the horse under general anesthesia, but sedation and local anesthesia may on occasion suffice.

Aseptic preparation is required especially when a synovial structure is involved. As the size of puncture wounds are misleading in relation to the underlying damage adequate exposure is essential. The hoof is not readily mobilized as is skin, therefore, the overlying epidermis and dermis have to be removed. All obviously devitalized tissue should be debrided. Debridement of tissue of questionable viability depends on its location; complete debridement reduces contamination and minimizes healing time.

However, in order to reduce the risk of iatrogenic contamination, it may be beneficial to leave questionable tissue immediately adjacent to synovial structures that are not currently infected; these tissues can be re-examined and debrided later if necessary. Further bacterial cultures may be taken at surgery.

Postoperative antibiotic therapy is modified based on the results of the culture and sensitivity. The duration of antibiotic administration varies with the duration of the problem and the structures involved. For most soft tissues and bone, antibiotic coverage can be discontinued after all surfaces are covered with healthy granulation tissue. For infection involving synovial structures, antibiotics should be continued for 2 weeks after the synovial membrane has sealed and the symptoms resolved. The wounds should be protected with a bandage, treatment plate or plastic boot until epithelium has covered the defect and begun to keratinize. The author usually lavages the wounds daily for the first 4-5 days with a 0.1-1% solution of povidone iodine (1-10% Betadine) in saline, but uses only sterile saline with antibiotics when lavaging synovial structures. Wounds should be bandaged with nonadherent dressings until completely epithelialized. The author uses gauzeI prefer a Telfa pad backed by cotton gauzes moistened with 0.1-1 % povidone iodine (1-10% Betadine). If a synovial cavity is exposed I prefer to moisten the dressing with sterile saline and an antibiotic, usually penicillin or gentamicin.

INFECTION OF SPECIFIC STRUCTURES

Septic Pedal Osteitis

Septic pedal osteitis usually occurs following direct penetration of a foreign body, extension of infection from a subsolar abscess or following laminitis. Sequestra may also be present. The treatment involves debridement as described and curettage of the exposed bone. Caution must be exercised when the diseased tissue is adjacent to the extensor process or insertion of the deep digital flexor tendon because of their proximity to synovial structures. One report describes the treatment of 9 cases, including 2 horses with laminitis, of which 7 returned to soundness and 2 died of unrelated causes.[1] However, it is commonly believed that the prognosis for horses with septic osteitis secondary to laminitis is not as good compared to other causes; recurrence of drainage weeks to months later is not uncommon.

Necrosis of the Collateral Cartilage of the Distal Phalanx

Necrosis of the collateral cartilage of the distal phalanx, commonly called quittor, occurs following heel lacerations, puncture wounds or ascending infection from subsolar abscesses. Swelling over the proximal margin of the cartilage associated with draining tracts is almost pathognomic for quittor. The swelling is painful and the horse shows a variable degree of lameness. The diagnosis is usually straight forward based on the clinical signs, but may occasionally be confused with a gravel or abscess at the coronary band. A probe inserted into the tracts should abut cartilage and indicate the extent of the injury. The probe may be radiographed in place to confirm its relation to the cartilage and palmar process of the distal phalanx. Treatment involves sharp excision of all necrotic cartilage and curettage of the margins of the wound. When the infected tissue extends below the coronary band, drainage through the abaxial hoof wall should be established at the most ventral aspect of the wound. The skin wound may be sutured and the drainage hole if present packed with povidone iodine soaked gauze. Approximately 75% of horses treated surgically become sound.[2] Conservative therapy has been attempted, but not found to be as successful. The prognosis appears to be better with a shorter duration of disease.

Septic Arthritis of the Distal Interphalangeal Joint

Septic arthritis of the distal interphalangeal joint is most commonly caused by lacerations to the foot, but may also occur after punctures, extension of subsolar abscesses and after intra-articular injections. These horses are usually extremely lame. There is painful swelling dorsal and proximal to the coronary band. Confirmation of the diagnosis is by cytology of a synovial aspirate or by demonstration of communication between the joint and a wound. Initially the joint space may appear normal, increased or decreased in width without osseous changes; later there may be evidence of osteomyelitis of the second and third phalanx and the navicular bone. Treatments applied in the past have included systemic and local antibiotics, joint lavage, indwelling drains and bone grafting. The client must be given a poor prognosis for survival, though in one study 7 of 11 horses treated survived and 2 became sound.[3] Interestingly the distal interphalangeal joint fused in 5 of the 7 horses to survive.

Septic Navicular Bursitis

Puncture wounds to the frog and adjacent sulci are the most common cause of septic navicular bursitis. By the time of presentation these horses are usually extremely lame. Frequently there is swelling of the heel proximal to the coronary band and application of hoof testers across the frog may elicit a painful response. As with septic distal interphalangeal joints, the diagnosis is

established by cytology of fluid aspirated from the navicular bursa or demonstration of communication between the navicular bursa and a wound. Care must be taken to determine whether other structures are involved because the infection can readily extend to the distal interphalangeal joint, the navicular bone and the digital flexor tendon. The treatment generally recommended is surgical debridement of the wound, and establishment of ventral drainage by removing part of the frog and fenestrating the deep digital flexor tendon (called a street nail procedure). By the time most horses have arrived at a referral center this is certainly the case. However the surgical procedure itself is not benign. Therefore, in the unusual circumstance that a puncture wound involving the navicular bursa has been diagnosed soon after it occurred, the penetrating object was relatively clean, and following removal of the foreign object the lameness improved, more conservative therapy may be used initially. This includes broad spectrum antibiotics, flushing the navicular bursa with sterile saline and antibiotics, and debriding the wound down to but not including the deep digital flexor tendon unless the latter is severely traumatized. If the lameness worsens or after 2-4 days the lameness has not improved, establishment of ventral drainage is indicated.

Complications of septic navicular bursitis and its treatment include osteomyelitis of the navicular bursa, infectious arthritis of the distal interphalangeal joint, rupture of the deep digital flexor tendon, septic tenosynovitis, and septic deep digital flexor tendonitis, all of which negatively impact on the prognosis. After surgery it is important to provide pain relief by elevating the heels. The author has found that Redden wedges (Redden Ultimates) wedge shoes work well if the Lily pad is removed and double up as a removable treatment plate. The prognosis for life is gaurded. In one retrospective study 31.6% of horses had a satisfactory outcome defined as suitable for some sort of athletic performance, breeding or considered useful by the owner.[4] The prognosis was best in horses that had a hind leg affected and were operated on within one week of injury. In another survey 10/19 horses survived; this study also emphasized the benefits of rapid treatment.[5] More recently 6 horses with septic navicular bursitis that were treated by placing bone grafts in the surgically created defect have been described and 4 survived. Thoses these numbers are small, they certainly offer encouragement for the treatment of they condition in the future.[6]

It should be apparent from these retrospectives that some are rewarding to treat while others are frustrating, disappointing and time consuming. Also the treatment cost varies greatly, but it may be very expensive. Technological advances may slowly improve our ability to treat these diseases and minimize complications. The referral of some of the horses in these retrospective studies for appropriate treatment sometimes occurred months after the injury. Therefore, our greatest immediate impact as practicing farriers and veterinarians is by rapidly identifying injuries where one of these problems is likely to ensure prompt treatment. Early referral of horses that turn out to be false alarms is far better than the late referral.

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