On Bone and Fracture Treatment in the Horse

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1. Introduction
Long bone fractures in horses have long been a major problem for horses, owners, trainers, and veterinarians. The practice of humane destruction of horses with open comminuted fractures or for that matter any long bone fracture was long thought to be the only “satisfactory” treatment. For years the public at large thought all horses that had fractures were shot. To my consternation some still do. It was common for people to believe that horse bone didn’t heal. “They Shoot Horses Don’t They” a Horace McCoy novel of the 1930s, made into a movie more than 30 yr later had little to do with the horse but portrayed the sentiment.

To review the advances in equine long bone fracture treatment it is only necessary to explore the experiences of the last 35 yr. The purpose of this paper is to review some of that history, but more importantly to describe the current state-of-the-art in this Milne Lecture.

2. Bone Structure, Vascularity, and Function
Bone is a unique and fascinating material. It is not an inert structure: it can renew itself. It can add and subtract new bone on its surfaces (change shape) as it grows in a process called modeling (Fig. 1). It may also replace itself going through a remodeling phase that involves bone activation, discrete areas of resorption followed by bone formation in these resorption spaces (Fig. 2). Since bone can only form on surfaces resorption creates the surfaces on which the bone forms to replace itself. Modeling and remodeling can be occurring in the same bone at the same time. Bone does not heal, incorporating the scar tissue as seen in most all other tissues: it regenerates itself. It changes its shape and structure based on its use and if broken can resume 100% of its former strength and function. Any treatment that doesn’t reconstitute the bone into its fully functional state must be considered sub-optimal.

The three major components of bone are osteogenic cells, organic matrix, and mineral. The osteogenic cells include osteoblasts, osteocytes, and osteoclasts, while the matrix consists predominantly of collagen and proteoglycans that constitute approximately one third of the bone mass. The mineral that makes up approximately two thirds of bone is composed of calcium phosphate crystals deposited as hydroxyapatite. Although all bone is made up of these components its structure differs based on its apparent density and porosity. Cortical bone in the horse is made up of primary and secondary osteons or haversian systems (Fig. 3). Cortical bone has an apparent density of ~1.85 g/cm³. Cancellous bone is less dense with an apparent density of ~0.9 g/cm³ or less. Cortical bone may have a porosity of only 5%, whereas cancellous bone may be greater than 20%. In the horse, so-called cancellous bone may
be so dense that it becomes osteonal as in the proximal sesamoids of Thoroughbred racehorses.

Osteoblasts develop through an undifferentiated lineage from stem cells. They lie on bone surfaces and make organic matrix to form new bone. They are very active metabolically when turned on and also participate in matrix mineralization. Osteoblasts may become trapped in their own matrix production and become osteocytes. These cells have long cytoplasmic processes that extend to other osteocytes and osteoblasts to form a network that covers over 90% of the mature bone matrix. These cells control the mineral exchange between the bone and blood and may act as chemical/mechanical transducers to initiate bone modeling and remodeling. Osteoclasts are multinucleated giant cells that resorb bone. These cells form on bone surfaces and resorb bone at the junction of their brush border with the mineralized matrix. They appear mobile and their cell numbers change in response to the “need” for local resorption. In cancellous bone the osteoclasts form Howship lacuna or craters as they resorb bone on surfaces. In cortical bone these osteoclasts form the front of the so-called “cutting cone” that marks cortical bone remodeling.

The organic matrix of bone consists mostly of type 1 collagen. The inorganic matrix is a calcium phosphate that forms a crystalline hydroxyapatite. As the inorganic phase matures, its solubility decreases and mineralization increases changing the mechanical properties of bone making it stiffer (Fig. 3). The modulus (stiffness) of cortical bone increases as the fourth power of the calcium content. The calcium content also affects fatigue life with less calcium yielding longer fatigue life in vitro and possibly in vivo.

Fig. 1. (a) Bone is laid down on cancellous surfaces in a lamellar form. Here the flourochrome sequential bone labels that allow for timing of bone formation are (from top to bottom) orange, green, yellow, red, and again yellow. (b) A transverse section of cortical rib bone in the horse shows the deposition of new bone forming primary osteons. The labels are the same as in 1a. (c) This section was taken from the inside of the rib in a growing horse. It shows the results of osteoclastic bone resorption and secondary osteons undergoing remodeling. This apposition from one surface and resorption from the other shows how the rib grows (drifts) as the animal increases the size of its chest cavity.
Morphologically, bone tissue appears to be under the control of bone cells. Its surfaces are covered by active and resting osteoblasts and osteoclasts, and it is permeated by an interconnected canalicular system in which osteocytes are found. Bone is formed only on surfaces (modeling). Modeling changes bone’s shape and seems to be related to the physical forces that the bone encounters (Wolff’s Law). The bone cells control the composition of the extracellular fluids of mineralized bone matrix within very narrow limits, and at the same time they can remodel the mineralized tissue to meet the anatomical needs of the mature skeleton.

Vascularity of bone is maintained through a medullary and periosteal blood supply. The cortex is largely supplied from the medullary source (inner two thirds) with the periosteal supply covering the outer one third of the cortex. Circulation is mainly centripital in nature. The afferent blood supply consists of arteries and arterioles (nutrient vessels) and the efferent supply is made up of the veins and veinules (periosteal vessels) with the capillaries being the intermediate connection.

The metaphyseal arteries are multiple and enter all the periosteal surfaces of the proximal and distal metaphyses. They provide the entire efferent supply of the metaphyses, and their terminal branches anastomose with the terminal branches of the medullary arterioles at each end of the medullary cavity.

At the microscopic level the central canal of the haversian system is the arteriole with Volkman’s canals being transverse components (Fig. 3). Bone formation and resorption is a vascular phenomenon and does not occur without blood supply. In the dog and horse, no osteocyte is more than 0.3 mm from a blood vessel.

Mechanically, bone functions as a material and as a structure. As a material, bone is made up of organic matrix and mineralized matrix. The type 1 collagen combined with the mineral gives bone material properties that allow for limited deformation and a certain brittleness. This is why bones bend then break. The bending is related to the collagen phase, which has a very low modulus (stiffness), and the breaking is related to the mineral phase, that has a high modulus, which allows little deformation (up to 2% strain) before bone failure. Materials that are made of two or more different materials with different physical properties are called composite materials. Bone is a composite material (collagen and mineral) and its modulus relates to the sum of its parts. Equine cortical bone has a modulus of 18–20 giga pascals (Gpa).

Bone functions mainly as support for the body. Its structure has greater strength in some directions than in others. Bone is anisotropic in that it has different material properties in different directions. Long bones are usually thought to be orthotropic, being strongest in their axial direction and ~20% weaker in their radial and transverse directions. Bone is strongest in compression and weakest in tension. Bending forces produce tension on the convex surface of the bone, hence bones are weak in bending. Torsion forces will resolve into tension forces as well so bones are also weak in torsion. Loading of bone determines its shape: functional requirements may lead bone to model and remodel to change its shape and internal architecture. Bone overload may create injury and fracture. Exercise is an important determinant of bone form and function. Bone gains its strength from its material properties and its sectional properties. Just as an I-beam has a special shape to support a load in a specific direction, bone too develops so that its cross-sectional areal properties will support loads that it commonly encounters. Bone modeling and remodeling are active processes that control this shape and may be modified depending on the loads applied.

Fatigue failure of bone may occur when bone is repeatedly loaded below its breaking strength. This is accomplished by repeated high strain cycles...
as occurs with young Thoroughbred horses in race training. The resultant bucked shins and stress fractures are discussed as a separate topic. Many common racetrack injuries such as P1 fractures or condylar fractures may represent bone failure due to high strain repetitive motion fatigue injuries of bone as well.

Bone material properties and sectional properties give the bones the strength they require to act as levers for muscles, to give form to the soft tissues of the body, and to provide protective cavities for the vital organs. In the horse the skeleton has evolved in a special fashion that allows this rather large animal to run at great rates of speed. Man has adapted this evolution to sport and today most horses in this country are used in sport for racing, eventing, showing, or pleasure riding. Many of these sporting events place the animals at risk for injury that includes bone fractures. Catastrophic racetrack injuries that lead to an animal’s demise involve ~1.5 horses per 1000 race starts. This statistic is about two orders of magnitude greater for injury of the racehorse than college football players in the United States. Obviously many catastrophically injured horses are euthanized because of their value as opposed to injuries in man, but until more recently many of the injuries of these horses could not be treated successfully. That is not to say that all racetrack injuries can be treated successfully but the progression of treatment success seems to have paralleled better surgical techniques and instrumentation.

These techniques evolved with the use of the original Swiss AO system of plates and screws that is known today as the Association for the Study of Internal Fixation (ASIF). The true father of modern equine orthopedics was Jacques Jenny. Jenny was born in Ennenda, Switzerland in 1917 and graduated from veterinary school in Zurich in 1942. He first came to the United States in 1948 to accept a position at the Angell Memorial Animal Hospital. There, working with Erwin F. Schroeder, he gained experience in treating fractures in small animals with the Schroeder-Thomas Splint. After a few months he moved on to Philadelphia and was appointed an instructor in surgery under Mark Alam. Jenny and Alam fixed a fracture in an English Setter using an intramedullary pin in 1948. History records this event as the “first intramedullary pinning performed in man or lower animals in the city of Philadelphia.”

When the New Bolton Center Campus came on line in the 1960s Jenny moved his main efforts to that campus and concentrated on horses. He pioneered the surgical treatment of joint fractures in racehorses. Through his association with Bruce Hohn, then of the Animal Medical Center in New York City, Jenny met and befriended the late Howard Rosen, an MD orthopedic surgeon from New York City. Howard had recently had an epiphany: taking up the AO method. He convinced Jenny, Hohn, and Joe Stoyak to go to Davos Switzerland to take the course then offered only to MDs in 1966. Upon their return the ASIF method as we know it today started to evolve in veterinary medicine, with Jenny leading the effort in the horse. Hohn organized the first human and veterinary AO/ASIF course in North America in Columbus, Ohio in 1970. That course has continued uninterrupted for more than 30 yr. Since those early years the human course split off, whereas the veterinary one developed equine as well as small animal subgroups. The Columbus course remains the premier course in North America for operative treatment of fractures in veterinary medicine and the faculty represent a who’s who in veterinary orthopedics.

The first “long bone” fractures to be treated successfully by using the ASIF techniques were olecranon fractures using a special “Spoon Plate” manufactured by the Swiss group especially for Jenny for use in the horse. John Alexander reported the results of 8 olecranon fractures at the AAEP in 1972. Those that were treated with this new plate survived and were sound. Most other fractures were handled with the available human implants, often using casts and splints for recoveries when results didn’t live up to expectations (Fig. 4). The real world of immediate full postoperative weight bearing in the horse contradicted the AO principles that wanted early joint mobility followed by partial weight bearing. In addition, postoperative recovery from anesthesia proved to be a major hurdle with animals tearing apart in the recovery stall in a few seconds the efforts of many tedious hours of anatomical reduction and fixation of long bone fractures in the operating room. After these initial experiences Jenny designed and tested the pool raft recovery system for horses, which was incorporated into the C Mahlon Kline Orthopedic and Rehabilitation building at New Bolton Center that was dedicated 4 yr after his death in 1971 (Fig. 5).

The improvements in treatment results occurred gradually as the surgeons involved began to understand the mechanics of fixation devices, proper surgical techniques and approaches, as well as an improved ability to cope with an uncooperative patient. Along the way the philosophy of the founding Swiss AO/ASIF group changed as well. In the early 1970s, fractures were fixed with “rigid” internal fixation. This meant that following perfect anatomical reduction there would be absolutely no motion between the fragments. The bone healed by remodeling across the fracture lines (primary bone healing) without callus formation. This healing pattern was documented histologically in non-weight-bearing sheep metatarsal osteotomies. Callus formation was considered a sign of failure of the technique. Because absolute rigidity was a near impossibility even with partial weight bearing, and fractures healed well with plates and screws often with some callus formation the term rigid was replaced with “stable” internal fixation in the
1980s. Callus formation became an accepted component of fracture healing, if not extreme, and the 1990s saw the term biological fixation employed. Here, the concept was not to disturb the fracture hematoma and comminution associated with it. The internal fixation would bridge this area and allow the bone morphogenetic proteins to do their work while the ends of the fracture were held in alignment. Articular fractures were still reduced and stabilized anatomically using interfragmentary compression techniques. While the small animal veterinary orthopedic community embraced this latest concept of biological fixation, the equine group was still looking for increased strength and stability in their fixations as required by immediate full weight bearing in a the horse. Several reasons peculiar to the horse have helped maintain the focus of the equine group on improving the immediate weight-bearing potential of internal fixations. Laminitis, as a sequela following long bone fracture in the horse is a serious problem and may be fatal. Horses don’t experience bone atrophy and “stress protection” associated with their implants to the extent that occurs in man and small animals. Therefore, the need to move the implant away from the bone seems unnecessary and may compromise the strength of the fixation. Cyclic loading of implants, which occurs during weight bearing in the horse, has pointed out the importance of micromotion within the bone/plate composite as it relates to implant failure and fracture healing.

Although the human ASIF group progressed to treat most fracture healing problems successfully by using plates, screws, and intramedullary rods, the problem areas they address are sequela to fracture healing and plate removal. The so called stress
protection phenomenon of bone failing after plate removal and the desire to do less invasive surgical procedures through computer-assisted surgery drives the research and development efforts of this group. This has led to more elastic implants for fixation as well as moving the plates off the cortical bone to limit vascular/stress protection problems. Increasingly, technology is being moved into the surgery room. Magnetic resonance imaging, computed tomography, and computerized surgical guidance systems are being integrated into the surgery to allow less invasive surgical treatments of difficult fractures that previously involved extensive open reduction techniques.

The horse, on the other hand, has not been as lucky as man. We still have significant problems associated with fracture healing and the postoperative management of the patient. Our problems in the horse relate to a 1000-lb animal that is uncooperative and must have immediate full weight bearing to prevent catastrophic sequelae such as laminitis. Infection and pain management are additional significant problems that the surgeon must address with equine patients. Because amputation is not a suitable end result for most equine patients, euthanasia in horses replaces amputation in man. Whereas many high-energy open fractures in man with significant arterial injuries go on to infection and amputation, equine patients with these problems are euthanized.

For fractures to heal there is an optimal relationship between the biology of bone healing and the mechanics of fixation. Understanding this optimal relationship in the horse and then achieving it is a challenge. The conflicting relationships between fixation to support immediate full weight bearing and fixation to optimize fracture healing may never be resolved in the equine patient. Entirely new directions/technology may be necessary. In the interim, research directed at the problem in the horse continues and some advances have been made that seem to improve long bone fracture treatment in the horse.

Plate luting, a technique that uses polymethylmethacrylate (PMMA) interposed between the plate and bone as well as between the screw head and the plate hole, to improve contact, stability, and fatigue resistance to cyclic loading has been used in our hospital at New Bolton Center to improve the outcome of plate fixation in equine long bone fractures since the 1980’s. This methodology of increasing the contact area of the screw head with the plate hole and increasing the contact surface between the plate and bone provided a mechanism that prevented micro motion at the screw head as well as movement of the plate on the bone. In vitro testing using equine third metacarpal bones demonstrated that plate luting did not increase the breaking strength of the bone plate composites but did increase the fatigue life of the repaired bone fracture and changed the fracture mode (Fig. 6). Without plate luting, individual screws failed progressively as they became loaded. With plate luting, the screws were loaded as one and the bone or plate was the usual failure site. This resulting improvement showed that plate luting could increase the fatigue life of the implants by an order of magnitude (300–1200%). That meant that the horse could walk on its internal fixation repair 3–12 times longer before implant failure with plate luting than without it. Because success in the horse is often a race between bone healing and implant failure, it can be
seen that plate luting could make a large difference. Young reported the results of nine tibial fractures that were treated between 1980 and 1987.6 The seven cases where plate luting was used had mostly excellent fracture healing while the two cases where it had not been used showed failure of fixation. Before the use of plate luting one foal with a tibia fracture had been sent home in the previous 10 yr. After the introduction of plate luting five consecutive foals were discharged after tibial fractures. The next foal with a tibia fracture did not have plate luting and did not survive. This dramatic effect of plate luting in foal bone seemed to be repeated in adults as well. Presently, the use of plate luting is a standard technique in treating long bone fractures with plates. The only exception is with olecranon fractures where the plate acts as a tension band and has very little contact with the bone. In addition, incorporation of antibiotics into the PMMA has the added benefit of continuous local antibiotic coverage within the wound of potentially infected fractures post operatively.

The biology of plate luting has been studied in the horse and showed that the addition of plate luting to normal plating procedures did not increase the porosity beneath the plate. Because bone/plate contact would be complete with plate luting it would seem that interference with the vascularity of the bone would produce some change. Seven adult horses were used to study the biological response of the bone to plate luting. Each horse had a six-hole broad plate applied to the cranial surface of its intact mid-radius using six screws. Using this paired model one side was luted and the other side was not. Horses were examined in groups of 2 at 1, 2, and 6 mo after surgery. One horse was examined 15 mo after plate application. Histological examination of the material showed bone densities of 0.9244 for luted plates and 0.9295 for non-luted plates. Studies in sheep showed that plate luting did increase the bone porosity under the plate by ~2% when compared to non-luted plates. The massive size of the equine bone in comparison to the plate size may be responsible for this difference. In addition, individual animal species may react differently to vascular compromise and/or stress shielding. In the horse, plate luting appears to have a great mechanical effect in preserving the internal fixation during cyclic loading (weight bearing) without additional negative biological effects that would influence bone healing.5 The mechanical effects of plate luting allow the implant to work in unison with all its parts. All the implants are locked together including the plate to the bone and therefore get stressed together with externally applied loads. Without plate luting individual screws may be loaded sequentially with subsequent overload and failure.

Locking screws in plates has come to the forefront in the development of new plate fixators in man. The PC-Fix introduced by the ASIF group in 1985 used a Morse taper system that fitted the head of the screw into the plate to provide this head locking principle by using unicortical fixation (Fig. 7). In 1995, the introduction of a threaded head that fit into a threaded plate hole was used to lock the screw head.7 These systems were not compatible with interfragmentary compression, as had been advocated previously by the Swiss group, and therefore did not develop a universal following. Most recently, in 2001, the AO/ASIF technical commission has endorsed the locking compression plate as the new AO/ASIF plate standard for the organization. This plate system takes the advantages of both systems and uses them in one plate design (Fig. 8). This system has not yet been used in the horse as a clinical entity but may have promise. The concept of mechanically locked screws using a threaded interface between the screw and the plate should provide stronger and more certain stability than plate luting. This locking compression plate concept might even allow the use of biological fixation to enter the realm of possibility for use in the horse. All efforts in commercial locked fixators are focused on the screw. The plate is being moved away from the bone so bone/plate friction is not a factor. Plate luting has effects on both the screw and the bone/plate interface which appears to be

Fig. 7. The PCFIX is shown on a non-equine bone. This plate used unicortical screws with a friction locking head (Morse taper) in the plate, but did not allow dynamic compression or screws for interfragmentary compression.

Fig. 8. The new AO/ASIF locked compression plate hole is shown that will allow a threaded screw head to lock the plate or a compression hole that accepts the classical methodology.
synergistic. Certainly continued experience using locking fixators may improve the outcome of fracture fixation in horses as well as man.

External skeletal fixation is another area of exploration for use in the horse. This treatment modality has not been widely embraced in equine orthopedics with “Pins in plaster,” a technique of incorporating transfixation pins in plaster casts being used in man before its reported use for horses in the 1950’s. The use of human and small animal fixators adapted for use in the adult horse have been notable failures in the hands of many different surgeons and have gone unreported in the literature. A resurgence of interest in external skeletal fixation occurs every time a clinician is faced with an unstable, severely comminuted open or closed fracture of a weight supporting bone where reconstruction using internal fixation with screws, or plates and screws, represents a technical impossibility. Use of a cast alone for this type of injury as an alternative to euthanasia often leads to collapse of the fracture with the fracture fragments penetrating the skin ensuring sepsis and disaster. Anecdotal case reports using casts alone have perpetuated their use in these types of cases where they are not indicated. Nemeth and Black documented the use of “pins in plaster” for use in large animals, including horses, as a treatment modality in 1991. This report described the use of a walking bar incorporated into a cast using transfixation pins above the fracture. An overall success rate of 57% was reported for a variety of fractures in 35 horses and 21 ponies. The authors advised using the pins in the distal aspect of the intact bone being cast to help prevent fracture through the pin sites. They reported the major complications, which included infection in nine cases, fracture through the bone or pin sites in six cases and two cases where cast application led to loss of circulation to the distal phalanx. It was unclear from this report how many of the animals had open fractures at presentation for treatment. The reported complications only included 17 of the 24 animals that did not survive.

Two papers by McClure et al. have pursued the “pins in plaster” technique and have shown that the substitution of fiberglass casting material for plaster seems to make the walking bar unnecessary. These authors developed an in vitro test to compare an oblique osteotomy of the first phalanx treated with transfixation pins in the distal McIII using a fiberglass cast, to the same osteotomy adding the walking bar, and to a fiberglass cast specimen alone. They also angled the transfixation pins but could only demonstrate that the configuration without the transfixation pins (cast alone) was weaker than the other combinations. This group went on to test the effect of pin placement in the distal McIII. They used parallel pins and 30° divergent pins and tested them in torsion to failure. Although they reported a significant difference between the tests favoring the divergent pins, their results showed that there was little difference between these two methods of pin placement with the standard error of the mean encompassing both data sets. The distance between pins used in their study was small (2 cm). Previous studies by Diefenderfer showed that stress concentrations in the equine McIII were magnified when the holes were less than 5 cm apart. This study used photoelastic coatings on intact equine McIIIs examined under polarized light to determine the surface strains in this in vitro model when loaded to 10,000 newtons.

Nunamaker et al. described an external skeletal fixation device (Model I) specifically designed for equine use in distal limb fractures in 1986. This device incorporated three transfixation pins in the intact bone above the fracture, with sidebars and a base plate that allowed the transfer of weight-bearing forces around the fracture to the ground. This allowed the animal immediate full weight bearing via the pins and sidebars without loading the fracture. The original report described the device and reported on the first 15 cases, only 4 of which survived long term.

In a follow-up report at the AAEP in 1991, they introduced a new prototype (Model II), based on the original design that eventually went on to be manufactured and distributed. The report added another 5 cases and increased the overall survival rate from 26.7% to 35%. It was interesting to note that only 1 horse survived of the first 10 treated, whereas 5 of the next 10 horses treated with this device survived. Fracture of McIII through the pin site occurred in 3 of the 20 horses treated while wearing the device and in 2 more at the time the animal had the device removed and was recovering from anesthesia. Removal of the device standing eliminated further problems following device removal but the incidence of fracture (15%) while wearing the device was of continuing concern. The commercially manufactured device broke consistently at a threaded junction on the frame and was removed from the market.

In 1994, Nunamaker and Nash reported at the ACVS meeting a newly developed concept of loading the external skeletal fixation pin in shear instead of bending. The concept involved using large diameter sleeves over the transfixation pins that were biaxially loaded in tension and shear. The device used only two pins and incorporated these pins in a stronger lighter frame (Fig. 9). This new concept was developed to try and reduce the bone fractures that occurred through the pin sites in the earlier frame design. The loads to bone failure reported were an order of magnitude higher with this new system than with the older configuration that allowed pin bending but used three larger pins. Because bone failure occurs at a finite deformation (2% strain) it appeared that the larger loads to failure were indicative of lower strain levels in the bone at the working stress level required by the fully weight-bearing horse in the frame.
cal trials using this new device proved efficacious and the device is presently available commercially (Fig. 10).

Strength of the external skeletal fixator is intricately related to the diameter of the transfixation pins, the distance from the outer cortex of the bone to the connecting sidebars, and the strength and stiffness of those sidebars and connectors. Deflection of the transfixation pins is proportional to the cube of the distance between the bone and the sidebars. The stiffness of the pin is proportional to the fourth power of its diameter. Loading of the pins produces bending and the stresses in the pin and outer bone cortex are increased (Fig. 11). The sleeves used in the Model III external fixator are infinitely large in relation to the distance from the sidebar and the loads imposed; hence, the pins do not bend and are loaded in shear uniformly across their length within the bone (Fig. 12). This reduces the bone stresses and allows larger loads to be taken through the pins before bone failure occurs. It also allows for smaller pins within the bone. Pin failure has not yet occurred in the Model III fixator.

Indications for the use of external skeletal fixation have been limited to fractures that cannot be successfully treated with casts alone or with internal fixation using screws alone or plates and screws. These fractures include badly comminuted fractures, open fractures or fractures with “bad skin.” While only distal fractures can be accommodated with the Model III external skeletal fixation device, more proximally located fractures might be better treated with the evolved “pins in fiberglass” methodology. It should be noted that to date the results of fractures distal to the carpus and tarsus are much better than treatment of proximal fractures by any transfixation method.

The decision to use an external skeletal fixation device or pins in fiberglass depends on the situation at hand. The pins in fiberglass technique uses a cast to cover up “bad skin” and or open fractures. This negates treatment of wounds and encourages complications of infection or pressure sores and vascular problems as mentioned in Nemeth’s paper. Incorporation of the transfixation pins into the cast also encourages a moist environment around these pins that may lead to local infection and pin loosening. Positive profile threaded pins should always be used to help decrease the incidence of pin loosening, especially when used with a radial preload. Pin and bone stresses are lower when larger diameter pins are used. Pin size is based on the bone involved. The pin diameter should never be greater than 0.3 times the smallest diameter of the bone being transfixed at the level of the pin site. Cast changes must be done periodically and loss of reduction and collapse of the fracture are possibilities at these times. Loose, severely bent or broken pins must be removed when present, and new pins placed at different levels creating additional holes (stress concentrators) in the bone. The use of multiple small pins may not negate the stress concentrator phenomenon if the pins are close together.

The external skeletal fixation device does not cover up the skin and/or open fracture wounds. It allows for local treatment without compromising the reduction or stability of the fracture. It has only been used successfully distal to the carpus and hock. Fracture healing while wearing this device is not complete. Removal of the device in about 6–8 wk is usually followed by additional support in a cast or Robert Jones Dressing to allow load transfer across the fracture site, which will allow for calcification of the already formed osteoid into bone. There have been no pin failures using the Model III external fixator. Pin loosening may be controlled to some extent by periodic tightening of the sleeved pins that is easily accomplished.

No matter what type of external skeletal fixation is used there are always concerns regarding the
Fig. 10. (a,b) A comminuted P2 fracture in an adult Thoroughbred mare was treated in an external fixator (c). The animal went on to a fusion of the joint as predicted by this 6-mo postoperative radiograph where the animal had full use of the limb.
pinholes that remain. Experimental studies in other animal species suggest that stress concentrations will be remodeled in ~8–12 wk. The holes themselves may never completely fill in. This means that the sectional properties of the bone have changed and the bone may or may not be as strong as an intact bone without any holes in it. With this in mind, presently, treatment of fractures with large diameter external skeletal fixation pins should be reserved for animals that would not be expected to return to athletic pursuits. The original pin diameters (9.6 mm) used in the Models I and II have been reduced to 7.94 mm in the Model III. This diameter seems to be satisfactory in all adult MCIIs.

The choice of pin diameter using transfixation methods is very important. In general, the larger the pin diameter the lower the pin and bone stresses associated with loading the construct. Pin diameters greater than 30% of the bone’s diameter start to remove too much material (reduce section properties) and even though the pin stresses get smaller there is not enough bone substance for support so the bone breaks. If very small pins are used the stresses are very large in the pin and in the bone. Small pins tend to bend (fail) before the bone breaks but use of small pins that bend and break will not guarantee that the bone stresses incurred will not lead to fracture. In general, the small pins that bend and break don’t provide large supporting forces to the bone so more pins must be used. Multiple small holes placed closely together may lead to fracture propagation. This has been nicely demonstrated using the ASIF DCP narrow plates with the screws bending under load and cracks propagating from hole to hole in the cortex under the plate. This often goes unnoticed except in experiments where the bone is labeled and shows the healed cracks in histology sections.

Fracture treatment success has progressed dramatically in recent years. Expectations for successful treatment for different fractures come from descriptions in the literature of case reports and retrospective studies. In addition, continuing education courses such as the Columbus ASIF course will provide information that is not in the literature. Expectations will also be related to one’s own personal experience or that of the practice/institution where the treatment is instituted.

Success of fracture treatment has progressed to the point that decisions involving treatment are often related to financial decisions and not to our ability to save the individual’s life. Euthanasia should no longer be thought of as a “satisfactory” treatment. The recent explosion of information regarding the molecular biology involving fracture healing may continue to improve the relationship of treatment to success. The next 30 yr may be just as exciting for equine orthopedics as the last 30 yr. In any case, the horse will be the winner!

References and Footnote


*Unpublished data, 1982, New Bolton Center, Kennett Square, PA.*