Response of Bone Necessitated by High-Speed Exercise

Larry R. Bramlage, DVM, MS

Author's address: Rood and Riddle Equine Hospital, PO Box 12070, Lexington, KY 40580–2070. © 2013 AAEP.

1. Introduction

Bone was once thought of as inert endoskeleton, but accumulated knowledge assuaged that belief. Bone is a very dynamic tissue, highly sophisticated in its adaptability to increase and decrease in the demands of exercise. It will enlarge to withstand additional load and is capable of reducing its mass when loads regress. As such, bone is the one tissue that is capable of regenerating itself and tuning its anatomy to its needs. The neonate is born with a highly adaptable skeletal model. The phenotype is genetically predisposed but refinement to the functional skeleton is dictated by the biomechanical loads that the skeleton experiences.\(^1\) Unlike organ systems such as the lungs, in which training causes no change in organ size, bones can enlarge cortices to double or triple thickness and tune the enlargement to best neutralize the demands placed on it.

In addition to its role as the biomechanical support for the body, the bone serves as the depot for the body’s reserves of calcium,\(^2\) which must be controlled within very narrow limits in the circulation because of its role in muscle contractility and muscle tone.\(^2,3\) The medullary cavities of bone are also the reservoir for mesenchymal and hematopoietic cells that supply the precursor cells for tissue healing and circulating blood.

The heavily training horse is required to carry skeletal adaptation to an extreme. Race training literally molds the juvenile skeleton into a racehorse skeleton; a horse is not born with a racehorse skeleton.\(^4\) The skeleton is also tuned for horses of other uses, but the racehorse demands the most of the bone because of the repetitive cyclic load the bone must endure during exercise.

It is advantageous for the horse to carry as little skeleton as possible because skeletal load is deadweight during high-speed exercise. The skeleton’s function during racing and training is to carry the weight of the horse and the rider. Redundant bone strength adds extra pounds and demands extra work to carry those pounds during competition, reducing the horse’s competitive advantage. Therefore, it is ideal for the racehorse to carry the minimum skeleton necessary for traversing the distance of the race but to carry sufficient skeleton to complete the race without permanent damage.\(^5\) To accomplish this, there is great rationale in work-specific adaptation of the skeleton to meet the demands it experiences, but to maintain a minimum of redundancy.
The extreme number of cyclic loads that must be withstood by the racehorse requires localized adaptation between bones and within the same bone to withstand these loads.\(^5,6\) The most extreme examples of this occur in the dorsal cortex of the metacarpus/metatarsus and the posterior cortex of the tibia.\(^6\) These cortical bone sites literally double or triple their cortical thickness on the heavily loaded cortex to enable the bone to withstand the cyclic loads that are produced by high-speed racing and training, but the trans cortex remains virtually unchanged (Fig. 1).

There are basically two cell populations that increase or decrease the size of the skeleton and remodel the skeleton to better withstand exercise: the osteocyte and the osteoclast populations.\(^7,8\) The osteocyte, which is the mature osteoblast encased in the bone matrix, is the permanent cell of bone. “Bone-lining cells” are the osteoblast source cells and are present within the medullary cavity and on the periosteal surface of bone. Growing bones have high populations of bone-lining cells until the end of growth, when the cells are no longer needed. These quiescent cells are able to quickly respond to increasing demand by becoming osteoblasts and initiating bone formation. When not used, these cell populations atrophy.\(^9\) Therefore, it is ideal to initiate training at the end of growth before the cell populations and supporting blood supply atrophy after growth, so they can be converted to the tasks of bone modeling and adaptation of the skeleton to training with as little interruption in the bone formation process as possible. This is desirable because of the economy of converting the blood supply and cell populations from one job to the next without significant “retooling,” but it is also advantageous to have a homogeneous bone structure without the distinct interfaces (cement lines) of markedly different ages of bone that are created when bone formation stops for a time, then is re-initiated due to changing stimuli (Fig. 1). Definitive “cement lines” are sources of stress concentration until the normal remodeling process eliminates them and homogenizes the bone cortex.\(^10\)

Bone-lining cells become osteoblasts under the biomechanical stimulus that exercise creates. Osteoblasts do not make bone directly; they make the proteinaceous precursor of bone called osteoid, which contains the collagen matrix component of bone. The osteoblast then excretes the mineral component, which forms the crystalline mineral hydroxyapatite, \(\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2\), within the osteoid matrix to form bone. The osteoblast eventually surrounds itself and becomes entrapped by the bone it produces, becoming an osteocyte.\(^11\) Though an osteocyte is trapped within the bone, it is far from isolated from the other osteocytes and the external environment because osteocytes maintain communication with other cells and the supporting blood supply by means of multiple cytoplasmic dendrites that lie within the numerous bone canaliculi.\(^9\) The dendrites interconnect with other osteocytes, with the vessels of the Haversian canal circulatory system of bone, and the periosteal surface of the bone.

Osteocytes are capable of adding to or subtracting from bone mass by adding bone or reabsorbing bone from the walls of their lacunae and canalicular spaces.\(^3\) Therefore, they can increase or decrease the density of the bone per unit of volume of bone tissue. Bone formation and dissolution must go on at low levels continuously to make calcium available to the body as needed to maintain the circulating calcium levels, should the ingested calcium be insufficient to supply these needs. It can be accelerated or decelerated as needed with physiologic demands. Calcium ingested in the diet and calcium excreted in the urine are the macro-sources of calcium acquisition and excretion, but the stability of the narrow physiologic range of serum calcium requires the stability of the bone’s supply of calcium to fine tune systemic calcium levels. The ions in flux during the process of increasing or decreasing bone density as bone is added or subtracted from the intra-osseous canalicular spaces produce the body’s readily available calcium ions.\(^9\)
Adding hydroxyapatite to the surfaces of the canaliculi and lacunae increases the density of bone per unit volume. This process can adjust the mass of mineralized bone per given volume, altering the strength of the bone. Bone generally increases in density with time. This is done principally by controlling the pH of the space between the cellular cytoplasm and the bone surface and the flux of hydroxyapatite into or out of the bone. Inflammation in an area of bone can demineralize bone by the same effect of decreasing the pH in the area, causing the loss of hydroxyapatite crystals, and therefore causing the loss of bone mass per unit volume. This has implications for the understanding and treatment of inflammation in bone. The lifespan of an uninjured osteocyte is estimated at 25 years in the human; we assume that this is similar in the horse and that the undisturbed osteocyte may outlive the horse itself.

The destructive arm of the bone remodeling system is the osteoclast. This is a multinucleated giant cell whose sole responsibility is bone reabsorption. Osteoclasts are manufactured on demand by the fusion of monocytes into a multinucleated giant cell with the capability of attaching itself to a bone surface and rapidly removing bone by localized acid digestion and dissemination of the resultant byproducts into the circulation and local environment. Osteoclasts work by attaching themselves to and sealing off an area of bone and demineralizing it with acid and then digesting the remaining collagen. They can do this very rapidly should the demand arise. The demand is created by physical damage, marked need for Ca\(^{2+}\) ion, or the need for bone remodeling. Osteoclasts and osteoblasts normally work in concert under the influence of the osteocytes to add or remove bone as the biomechanical load indicates the need. Osteocytes direct bone remodeling in response to loading, or the lack of loading, of the bone. The net result will be bone formation or bone elimination as dictated by the biomechanical input to the osteocyte, which mediates the response on behalf of the body as a whole. Osteoclasts are totally silenced in times of high mechanical demand to preserve bone for the short term.

Bone senses load by determining tension and compression. This is done to some degree by the deformation of the hydroxyapatite crystals, which create positive and negative charges on the surface of the crystals. This is a physical property of all crystals and was thought to stimulate bone formation. More recent consensus indicates that the cytoplasmic extensions of the osteocytes are sensitive to stretch and relaxation and the osteocyte dictates the addition of bone to neutralize the deformation. Compression deformation always dictates bone formation, whereas tension tends to create remodeling or sometimes bone loss. The compressed surface of the bone remodels the bone internally by adding to the bone density and models the external surface of the bone by adding bone mass to the most heavily loaded areas. This shapes the bone to best resist the stress of training. The dorsal surface of the cannon bones and the caudal surface of the tibia are very active examples of this bone modeling to neutralize ongoing bone deformation. Over time, the modeling/remodeling process alters the bone to the shape and strength that best prevents deformation on loading. At this point, the load is balanced on the long axis of the bone unless the workload changes. The modeling process then slows or ceases as long as the exercise load is not altered, but the remodeling must continually occur to repair “wear and tear.” Biologic systems never become “elastic” to the point that deformation does no damage. Biologic systems are always “plastic,” meaning that all loads do some degree of damage, which must be healed on an ongoing basis.

If damage is occurring too rapidly for the repair capabilities then the damage begins to accumulate, forming micro-fractures, then stress fractures, and eventually gross fractures and structural damage to the bone if unimpeded. Damage also accumulates if the modeling process to neutralize the loads of increasing training is overwhelmed by the damage of progressive exercise in the naive skeleton. The horse is particularly vulnerable to this damage because the heart and lungs of the horse are so capable that they tend to train faster than the skeleton. This makes the skeleton the “rate limiting system” in almost all training horses.

Osteoblasts and osteoclasts cooperate in the principal remodeling mechanism of bone called the bone remodeling unit (BRU), also sometimes called the bone metabolic unit (BMU). The BRU is a phalanx of osteoclasts, which cut a well-defined tunnel through and across existing bone, removing old bone in need of remodeling or strengthening. The osteoblasts then follow the osteoclasts in close concert, forming new bone (osteoid, which is then ossified to bone), creating new osteocytes during the bone remodeling, rejuvenating the bone into a newer form of itself. The BRU can only operate within mineralized tissue and cannot invade other tissue forms such as fibrous tissue, cross-gaps, or cross-unstable fracture planes. Gaps must be filled with mineralized tissue first, and the BRU can then remodel and refine the tissue to bone. The BRU is the principal tool the bone uses to heal fractures in the stable fracture healing situation.

The number of BRU functional at any point in time is dictated by the local osteocytes, which sense the biomechanical conditions and local needs with their canaliculi and dictate when remodeling is needed. “Exercise debt” in the form of microdamage is built up during exercise and then repaired between periods of damage. Injury and death of an osteocyte is the strongest stimuli for BRU activation and the remodeling that will replace the bone that has lost its resident osteocytes with
new bone containing living osteocytes. This raises the question of whether the mechanism of stimulating remodeling is actually the loss of the inhibition of the BRU that a living osteocyte may produce.

Training is the next most powerful stimulus for bone modeling and remodeling. Exercise trumps all hormonal and metabolic stimuli for bone remodeling, and its effect on bone lasts for years. The micro-damage, stimulated by exercise, causes overcompensation by the bone through the modeling and remodeling process, strengthening the bone mass and changing its shape to prevent the micro-damage from occurring in subsequent exercise sessions. If the exercise is then increased, the bone is overloaded again and over-repairs again, further strengthening and modeling the bone to neutralize the load. This stepwise stimulus of hypertrophy by “overload” then “over-repair” is the basis for training in all tissues.

The bone’s ability to respond to training is, however, not an unlimited resource. There is a maximum rate of response that the bone can generate. It takes time for the osteocytes to increase the density of the existing bone and for the osteoblasts to change the size of the bone. The “art” of training is to use the hypertrophy response to produce a continually stronger athlete, without progressing too rapidly for the bone to adapt, causing injury.

The question is often posed “Why is the horse so prone to injury and lameness?” The answer is not so much that the bone is weak but that the other systems are so strong. The heart and circulatory system are so highly developed that they can adapt rapidly to nearly any level of sequentially increasing exercise that they encounter. Bone is more limited.

Overload in excess of repair results in structural damage, injury, and eventually, pain. Pain is demonstrated as lameness. Lameness is the shifting of weight from one limb to another to avoid loading of the painful limb. This makes the horse asymmetric and then often precipitates a secondary additional lameness. Few horses performing at high levels are compromised by a single minor lameness. It is often the second or the third painful area that creates the decline in form. A horse will protect and demonstrate lameness on the most painful limb until that pain is relieved by treatment or diagnostic anesthesia. The horse will then protect the next most painful site. Progressive elimination of one lameness at a time until the horse is sound is the best way to determine all of the sites of pain. It is often not the most painful lameness that is the original and therefore most important lameness site. Treatment of the secondary lameness alone is prone to failure because the inciting lameness is still present and the secondary lameness will re-occur. The key or underlying lameness must be identified and treated as well if the horse is to regain soundness. This is the “art” of lameness treatment that must be combined with the “science” of understanding the mechanism of pain in bone and how the primary problem can be treated without endangering the horse or its performance capabilities over the long term. Lameness is a sign of a problem, not the problem itself. This concept is difficult for some owners and trainers to grasp.

Pain in bone and the resultant lameness is a sign of damage and results from instability, hypertension, or invasion of sensitive structures such as the periosteum, ligaments, or joints by the structural damage. Pain results in lameness, therefore lameness must be properly regarded as a sign of a problem, not the problem itself, to correctly interpret and treat it. Veterinarians should be well equipped to help the client with the understanding and interpreting of this normal response to training.

Trainers often regard lameness as “the problem” rather than a sign of a problem. This understanding is difficult for some and should be the purview of the veterinarian to understand the cause and treat the cause rather than only the symptom of the problem. Treatment of the symptom rather than the cause is often only temporarily effective, often damaging in the long term, and can be dangerous if pain indicating progressive structural damage is eliminated without regard to its cause.

What causes lameness in the racehorse? The most common causes are cyclic loading, load accumulation, plastic deformation, and structural damage to the bone. Joint inflammation and overload of tendons and ligaments is frequently a secondary sequela.

Bone is very good at adapting to gradual changes in loading levels or loading types, but this adaptation is work-specific and dictated by the biomechanical load the bone receives. Julius Wolff described this a century ago, and the adaptation of bone to specific needs has come to be known as “Wolff’s Law,” which states “bone is laid down where strength is needed and removed where strength is unnecessary.” Few animals carry this to the extreme of the training horse. The overload/over-repair cycle literally molds the racehorse skeleton (Fig. 1).

The bone can take two actions when faced with the increased need for strength dictated by exercise. It can change the material make-up of the existing bone volume, or it can change the size and shape of the bone to better resist the loads. Changing the material make-up occurs by adding bone mass per unit bone; filling in trabecular, canalicular, and lacunar space with more osteoid and hydroxyapatite to increase density increases the amount of osseous mass in the same volume of bone. Bone can also change its shape to neutralize the applied stress by adding bone as occurs in the dorsal cortex of the metacarpus/metatarsus and the caudal tibia. With the use of the same amount of material, changing the shape of a material from a rod to the cylinder doubles the strength of the structure in resistance to...
bending; therefore bone enlarges its cylindrical shape whenever it has the opportunity.  

The adaptation of bone to training is different than most tissues because it trains to the level of work rather than the volume. Bone requires only a limited number of loads in a specific exercise period to stimulate its response. Experimentally, this has been determined to be approximately 36 similar cyclic loads. Two thousand similar loads were no better than the first 36 loads at producing hypertrophy of the bone. In fact, in an exercise period of more than 36 similar cycles, the excess cycles become destructive, adding excess damage that can then begin to accumulate overwhelming the bone’s ability to repair the damage. The end result of this can be destruction of the bone rather than hypertrophy and eventually injury in the form of a stress fracture. Soft tissues such as the heart and muscles require a volume of work to train. The “interval training” of these tissues to increase the volume of work at a higher level revolutionized the training of people, but, when tried in the Thoroughbred racehorse, the skeleton could not withstand the volume of work. Endurance horses and Standardbred racehorses are able to somewhat more successfully apply the interval training principles, but it results in too much skeletal damage in the Thoroughbred to be successfully applied.

Because damage and response to training is work-specific, the damage to a racehorse’s bone can be mitigated somewhat by varying the gait and the training surface. Mixing more trotting and the use of different training surfaces varies the load the bone encounters and therefore varies the type of damage done by the training. This allows the volume of work to be increased without as much cumulative damage to the bone. Monotonous large volumes of similar training, such as lots of repetitive galloping over the same surface, becomes damaging to bone rather than stimulating of bone adaptation.

As the level of exercise increases, the tolerable volume decreases. Epidemiologic data shows “a horse that had accumulated a total of 35 furlongs of race and timed-work distance in 2 months, compared with a horse with 25 furlongs accumulated, had an estimated 3.9-fold increase in risk for racing-related fatal skeletal injury (95% confidence interval = 2.1, 7.1).” Mindlessly ignoring the ability of the horse’s bone to respond to the amount of exercise it is performing will eventually result in damage accumulation and injury.

The diaphysis of the metacarpus provides a dramatic example of the adaptation to exercise in a more macro-example of the adaptation that is occurring in the entire skeleton and how it can be disrupted if the adaptation does not occur fast enough or completely enough to maintain soundness. Exercise produces new bone at sites of compression but not at sites of tension strain during exercise. The best evidence indicates that this is an active “biologic” process mediated by the osteocytes in the loaded bone that monitor the loading by means of their large dendritic networks within the canaliculi of the cortical bone. Compression loading results in new bone production that models the bone to best resist the compression (Fig. 1). Bone is produced in the areas of the most compression. This new bone then reduces the compression differential until the load is balanced on the long axis of the bone and the bone receives neutral forces during loading. Overload of compressed bone results in the classic stress fracture configuration that occurs in all materials. The shear stress of compression results in an oblique fracture at 45° to the long axis of the bone (Fig. 2). It propagates into the bone until a lamellar plane that is of different stiffness is encountered, and the fracture then propagates along that plane in the long axis of the bone. If no lamellar plane is encountered, the stress fracture may propagate through the cortex into the medullary cavity (Fig. 3). In the young training horse, radiographically evident multiple stress fractures can sometimes be identified in horses that have pain and lameness originating from the metacarpus (Fig. 4). When training is proceeding faster than the bone can ossify and strengthen, the newly formed bone is
overloaded before it can become strong enough to neutralize the damage (Fig. 4). Stress fractures are part of the modeling process, though they do not become radiographically apparent in most horses. Multiple stress fractures are easier for the horse to overcome than is a single fracture that begins to predominate. If one fracture begins to predominate, it becomes a single stress concentrator, the stress is focused at this one site, and the rate of damage increases.\textsuperscript{10} When one fracture begins to predominate, it is difficult for the horse to remodel the bone fully enough to eliminate the stress concentration; therefore, surgery to accelerate the remodeling and promote healing is beneficial.\textsuperscript{23}

Because the stiffness of the older bone of the yearling metacarpus is greater than the newly formed

Fig. 3. Stress fracture propagated from the dorsal cortex into the medullary cavity. The horse was removed from training and stall-rested. The periosteal callus covers the exit of the fracture on the dorsal cortex, but the deep remodeling of the fracture is incomplete. Surgical treatment is often elected because of the prolonged remodeling time necessary for stress fracture healing.

Fig. 4. Radiograph of the metacarpus shows multiple stress fractures developing in the newly formed bone because the training is proceeding faster than the newly formed bone can be formed and strengthened in response to training.
bone (because the osteocytes have fully mineralized the bone), it is more susceptible to stress fractures than is the newly formed bone that is less stiff\textsuperscript{14,18} (Fig. 5). The stiffer the material, the less the elasticity, and the sooner the plastic limit where cumulative damage is done is reached. This biomechanical loading will often fracture the older, stiffer bone first. If bone of multiple ages is present within the same bone, the bone that is the most vulnerable will fracture. This is the bone that is the weakest combination of stiffness and strength (Fig. 6). As long as the fracture does not penetrate the surface of the newly formed shin, there is no pinpoint tenderness on the surface of the shin, though the horse is lame and the surface of the metacarpus is often diffusely tender to deep palpation. The newly formed bone re-enforces the metacarpus until exercise no longer causes deformation and stress fracture, but if exercise continues at a level higher than the rate of repair, it overwhelms the new bone formation and the new bone may be overloaded by the rate of damage caused by the exercise. As a stress fracture deep in the bone progresses and becomes more plastic, it may fracture through the newly formed bone to the surface of the metacarpus and cause progressively increasing lameness, focal tenderness on the surface of the metacarpus, and eventually, an unstable fracture if exercise is not slowed (Fig. 5). When the periosteal surface is reached by the stress fracture, pinpoint tenderness along with increased lameness is seen because the highly innervated periosteum is violated. If the newly formed bone is sufficient to stabilize the stress fracture the metacarpus is then strengthened and the pain subsides.

If the process has not yet developed a dominant fracture, modifying training to reduce the stress can aid the bone in neutralizing the overloading force by allowing it to get ahead of the damage caused by exercise.\textsuperscript{21} The most damaging force to bone is monotonous, repetitive training: after a finite number of cycles necessary to stimulate strengthening of the bone, the remaining cycles become trauma, contributing to the stress fracture creation.\textsuperscript{17} Modification of training reduces the trauma.

On occasion, acute severe lameness will occur after exercise in the absence of palpable tenderness on the surface of the bone. When lameness is localized to the metacarpus and radiographs are taken, the interface of the yearling shin and the bone formed since training was initiated is visible on the radiographs (Figs. 7 and 8). This appears to be a separation of the bone at the cement line interface of the two densities of bone caused by a differential stiffness, as can be seen on anatomic specimens (Fig. 1). This process can occasionally also be seen in the tibia, but it is harder to define radiographically because of the much larger soft tissue coverage of the tibia (Fig. 8). If the horse is given a break from training and the radiographically visible interface disappears, the lameness subsides as well.

![Fig. 5](image_url). Radiograph of a dorsal metacarpal stress fracture was initiated in the yearling shin because it was stiffer than the newly formed shin and more susceptible to cyclic stress. Open arrow marks the demarcation between the yearling shin and the bone formed in response to training. Once the fracture became unstable, the newly formed bone fatigued and broke as well. As the fracture penetrated the dorsal cortex and the periosteum, the lameness increased.
There is some individual variation in the ability to respond to training, as with most biologic systems. Some individuals can mount the response at a rate that virtually precluded their bone being overloaded and some individuals cannot respond fast enough for even the most moderate levels of training. The art of understanding this biologic variation is another quality needed to properly dose training.

Whereas the addition of bone to the surface of the bone to add strength and improve resistance to overload is a workable option for long bones, it is not an option for joint surfaces such as the distal cannon bone.20,24 Because the anatomy must be preserved in the joint, the bone does not have the option to simply hypertrophy the size of the distal metacarpal or metatarsal surface because it would distort the joint surface. Therefore, in its adaptation to training, the only option available for the distal metacar-

Fig. 6. Radiograph of the dorsal metacarpus shows a stress fracture in the middle of three layers of bone. The horse had a period of rest, and the lameness re- occurred during the second training episode. Black arrow marks the demarcation between the yearling shin and the bone formed after a period of training; white arrow marks the new bone formed after the period of rest. It is likely that the stress fracture in the middle layer of bone was incompletely healed during the period of rest.

Fig. 7. Left figure (A), with the small arrows, shows a milder case of separation of the yearling shin and the newly formed bone that was formed in response to training. Right figure (B), with the large arrows, shows a more severe separation of the yearling shin and the newly formed shin with more periosteal response than in the left figure.
The bone must do this by largely infilling the trabecularization in the subchondral surface to produce “cortical-like” bone because of the needed strength. This has the unwanted side effect of eliminating the ability of the trabecular subchondral bone to absorb load through minute distortion and energy absorption. The eventual end result is increased bone and cartilage loading between the relatively unforgiving distal metacarpus or metatarsus and the opposing phalanx and sesamoid bone. The transient result is subchondral bruising of the subchondral articular surface with frequent progression to other disease conditions such as condylar fracture, palmar articular degeneration, performance limiting lameness, or alteration of the shape of the articular surface and damage to the articular cartilage, predisposing to degenerative arthritis, or fracture of the opposing sesamoid bone.

This is especially true if the performance-limiting pain is mitigated to some degree with intra-articular medication and training is carried on despite the lameness.

This injury to the bottom of the cannon bone has received much attention in the literature, recently. It has been given many names such as bone bruising, mal-adaptation, and repetitive cyclic stress, all of which indicate damage to the bone in excess of the bones ability to repair that damage. Initially this was thought to be a disease only of the racehorse, but, more recently, the same syndrome has been implicated in reining horses, cutting horses, eventing horses, and jumping horses. It is less common in these uses but it makes sense that any horse subjected to similar exercise levels would sustain the same pathophysiologic response, which, if insufficient to compensate for the cumulative damage, would then result in pathology that creates performance limiting lameness and clinical disease. This pathology is a very common cause of a horse’s sudden dislike of training or refusal to perform routine tasks that it once enjoyed. In the racehorse, this is often seen as a sudden refusal to enter the starting gate when ordinarily the horse is unfazed by the loading process. It is also manifest by the lack of desire to train or go to the racetrack when the horse’s normal response has been enjoyment of training, as would be expected. We once termed this behavior “track sour,” though we now realize because of better diagnostic methods and better understanding of the pathologic process that this is simply an avoidance of the pain that the racing and training induces in the bruised distal cannon bone, if the training is proceeding faster than the bone can respond. Many clinical descriptions of this syndrome, its diagnosis, and its treatment exist in the literature. The most successful approach to the treatment of this disease in the author’s hands and in the literature is the use of free choice exercise to allow the horse to heal the injury before reinitiating training.

The premise for this treatment is that the horse is normally a grazing animal. As such, the evolution of the horse’s distal-limb circulatory system evolved with the horse spending much of its time grazing in a field. Horse grazing involves a few bites of grass followed by a few steps across the field and then a few more bites of grass followed by a few more steps, for hours at a time. Contrary to this evolution, we have altered the training horse’s daily routine to involve a short time, perhaps an hour, of high intensity exercise followed by many hours of stall confinement. This creates the trauma of exercise but

![Fig. 8. Lateral radiographs of the tibia show separation of the yearling cortex and the new bone formed in training, similar to that in the radiographs of the metacarpal III’s (McIII’s) in Fig. 7.](image)
eliminates the natural stimulation of circulation that would best support repair. We see the result of this perturbation of the horse’s natural routine as the accumulation of edema within the distal limb (“stocking up”) of the horse when they are stall-confined for long periods of time after intense exercise. We circumvent the edema with the application of “stall bandages” to reduce the edema or “stocking up.” The elimination of the edema eliminates the clinical symptom but does not resolve the primary condition of accumulated damage to the bones of the distal limb of the horse. Returning the horse to the natural grazing environment for at least 8 hours of every 24 for a 2- to 3-month period of time resolved the clinical lameness seen with the repetitive cyclic loading and allowed a return to racing in 95% of the horses treated.27

Another man-made predisposition to subchondral bone injury in the distal metacarpus is the addition of the excessive “toe grab” in the Thoroughbred racehorse.28 The toe grab adds a braking affect to the impact of the horse’s foot in the traditional dirt-racing surface. Experiments with instrumented shoes have shown that the forces that these horses receive at the impact of the foot with a toe grab into the traditional racing surface exceed the load received with weight-bearing.29 The toe grab increases the resistance to slide of the foot in the forward direction and causes the toe to turn down into the loose cushion of the classic dirt-racing surface. Lowering the toe creates a functional elevation of the heel of the foot, which then translates into a reciprocal increase in extension within the fetlock. Hyperextension of the fetlock joint increases the load on the suspensory apparatus and the palmar/plantar articular surface of the cannon bone at its interface with the sesamoid bone. This increased load increases the damage to the subchondral bone, cyclically producing damage in excess of repair, bruising, and structural damage to the articular surface of the fetlock joint in this location.20,30 Because the articular surface has a limited means to adapt (because it cannot change its shape), this leads to the myriad of clinical lameness encountered as a result of this process if training continues, proceeding faster than the horse can adapt. The same forces will be in play on other surfaces such as turf or artificial racing surfaces, but experimental studies have shown that the magnitude is greatest on the traditional dirt racing surface.30 This syndrome at least partly explains the high incidence of injury to the third metacarpus of the racehorse fetlock joint when compared with other articulations of the distal limb.

Recent questions have surfaced as to whether we could have inadvertently altered the genetic material and the bone’s ability to adapt by selective breeding, increasing the predisposition of the horse to injury. Progressive decrease in the number of races per year completed by the North American Thoroughbred seems to raise the question, but closer examination indicates the economics of racing are probably a bigger driver of the decreases in starts than the question of soundness.31 Publication of trainers’ statistics concerning the percentage of horses that finish in the top three places and the continual growth of the expense concerned with training without a similar growth in purses encourages more selective entry of horses in races. This increases the amount of training, and the participation in a race occurs only when the connections feel the horse is at peak form. Emphasis on the horse’s value as breeding stock also promotes the level of performance rather than the number of races competed. These economic pressures continually reduce the emphasis on career longevity and reward early retirement with lucrative breeding careers. One would suspect that the emphasis on early career “brilliance” and the de-emphasis of longevity would eventually take a toll on the ability to the racehorse to produce durable skeletons and long-lived careers.

In summary, bone is not a purely structural inert material; it is a dynamic support tissue that adapts to the work it is asked to do. Most clinical diseases are the result of training and damage in excess of the horse’s ability to respond to the exercise stimulus. Understanding of the horse’s bone response to exercise and modulation and adaptation of exercise programs to individual horses and to their response to the stimulus greatly aids in the avoidance of injury in the equine athlete.

**References and Footnotes**


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