Bone Remodeling of the Equine Distal Limb

Mark D. Fischer M.D.
Orthopedic Surgeon
University Minnesota, USA.

Sheri L. Fischer R.N., B.S.N.
Fischer Equine Lameness Foundation
www.healthehoof.com

Introduction

The coffin bone (third phalanx) provides structural support and an internal mold for the hoof. It also transmits weight up the axial skeleton of the legs.

Conditions which result in the loss of bone tissue in the coffin bone are devastating, because loss of substance from this bone typically cannot be recovered, and the change in the architecture of the coffin bone results in pathologic adaptations of the hoof and its supporting structures. (Figure 2 and 3) Alterations in posture and gait to accommodate, lead to further maladaptations. Much of the rationale for trimming is to preserve the position and integrity of the coffin bone. This paper will describe the cellular mechanisms of bone loss as well as bone formation. The concept of bone remodeling and the circumstances in which it occurs will be discussed. The paper will also describe the influence of mechanical forces on bone remodeling (Wolfe's law).

It is tempting to think of bone as inert structural supporting tissue, but unlike girders in a building, bone is biological tissue which has living cells, a blood supply (pipeline of nutrients), and it reacts to both chemical and mechanical influences by changing its own internal and external structure. In other words, if the chair you are sitting in was like bone, if a series of heavy people sat in that chair, then the chair would make itself stronger.

This particular discussion will focus on the equine terminal phalanx, also known as the third phalanx, or coffin bone. We will discuss some of the structural features that it has which are common to all bones, as well as its unique features.

General Description of Bone Structure in the Vertebrate Skeleton

All bones in the skeleton are composed of a combination of thick dense surface bone, referred to as the cortical bone, and a spongy irregular internal portion, referred to as cancellous bone. (Figure 4)

In the spaces between the spongy dividers resides the bone marrow.

The bones are covered by a vascularized (tissue with a generous blood and usually nerve supply) surface tissue called the periosteum. In the coffin bone, this tissue has a different name: the corium. The periosteum is the source of the blood vessels which grow into the bone, and is also one of the sources of bone cells (called osteoblasts) which add bone to the surface of bones. (Figure 5)

Bone itself is made of a combination of a collagen framework (which is essentially the same as scar tissue), and tendon tissue. In the bone, however, the collagen is impregnated with a inorganic salt: calcium hydroxyapatite. Collagen gives the bone its tensile strength (which resists stretch) and the minerals provided resistance to compression. The collagen framework and the mineral deposition are performed by osteoblasts, the cells which make bone.

Cellular Bone Structure

All tissue in the body is composed of living cells. Although there are other cells which live in and around bone, such as nerve and blood vessel cells, the cells which are the unique producers and managers of bone tissue are the osteocytes, the osteoblasts, and the osteoclasts. Each of these cells has a unique roll in the production and/or management of bone. The great majority of bone itself is extra cellular ground substance secreted by the osteoblasts. (Figure 6)

The osteoblast is a cell which produces bone. In many respects, it is similar to a fibroblast (a cell that produces collagen in tendons, and scar tissue). It produces a collagen-based substance called osteon, and then impregnates the osteon with a calcium-base mineral called hydroxyapatite, which crystallizes and gives bone strength. (Figure 7)

When the osteoblast is finished producing bone, it becomes an osteocyte: it lives within the bone structure, obtaining nourishment from small blood vessels and diffusion, and continues to maintain the bone chemistry. (Figure 8) If the osteocytes die due to lack of blood supply, the dead bone looses it strength and is easily fractured or resorbed.

The osteoclast is a cell which is formed from blood cells (as opposed to the cells which are native to bone). It appears that when there is a need to resorb bone, the osteoclast forms from a combination of cells which would typically be specialized to absorb debris. These cells combine to form a multi-nucleated giant cell which is capable of dissolving bone. The cells are under the control of systemic hormones as well as local chemical mediators which control the production of osteoclasts as well and their activities. (Figure 9)

The interior of the bone is supplied with blood vessels, which carry nutrients to the living cells in the bone: the osteocytes (these are osteoblasts which have completed the work of producing bone and now live inside and maintain the bone). (Figure 8) Other cells living in bone are nerve cells, which are mostly in the surface sleeve of tissue covering the bone; and bone marrow cells, which are the cells that produce red and white blood cells for circulation.

Growth and Development of Bone

Originally, all bones of the skeleton are laid out as connective tissue which becomes cartilage and then bone. The long bones, which include the phalanges (and the coffin bone) change from cartilage to bone in a predictable fashion. Blood vessels grow into the cartilage, and the change in the environment brought on by the vascular ingrowth result in the cartilage changing into bone. Cartilage producing cells change into osteoblasts, which then begin producing bone. (Figure 10)

In most long bones, there two areas, one at each end of the bone, which retain their cartilage during development until maturation (adulthood) occurs. These are the growth plates which continue to produce cartilage, expand (lengthen), and then change into bone throughout the growth phase of the bone. It is this process of organized expansion, followed by changing of cartilage to bone, which produces increased in length in the bones.

Bones also grow by appositional growth. (Figure 7 & 11) Bones are surrounded by a vascular enervated sleeve or envelope of tissue known as the periosteum. (This tissue has a different name around the coffin bone, it is the corium.) It is capable of either producing new bone, when its osteoblasts receive the right stimuli; or resorbing bone, when different chemical messages are received by the cells. Once additional bone is added to the surface of bone, the overall bone becomes larger in diameter. The coffin bone presumably grows wider by this mechanism, as well as growing longer by having one growth plate. The terminal 3 bones of the equine extremity each have one growth plate Bones further up the leg toward the shoulder have two growth plates.

Bones such as the skull and the pelvic bones grow by intramembranous bone formation. This is not applied to the coffin bone, so we will not discuss it in this report.

Bone Remodeling and Wolfe's Law

The term "bone remodeling" refers to the situation where bone adapts itself to various influences, both local and generalized. At any given time, bone is not static. Even if a given bone appears not to change, as if it were a structural element in a building, it is continuously being formed and broken down at the same time. This process is referred to as remodeling. This phenomenon was officially recognized by Junius Wolfe, a German scientist and the 1800s.

Wolfe's law refers to how bone adapts itself to a variety of influences. Bones can remodel in a generalized fashion – that is, affecting the whole bone, or even the whole skeleton; or they can remodel in a very specific fashion in response to a local influence. It is important to remember that bone remodeling is a balance, and many factors can influence the balance, so that the net effect is either bone gain or bone loss. Most of the clinical situations we encounter in both human and equine situations involve bone loss to an extent to which problems occur.

Sorting out exactly what mechanical influences are present in the given situation leading to the remodeling of bone can be challenging. Investigators at the department of biomedical engineering at the University of Tennessee attempted to do this with the equine radius bone. The theory was that the radius is exposed to relatively uniform forces with the anterior cortex (which is the front surface of the second bone down from the shoulder of the horse) being loaded in tension, and the posterior cortex (the bone's back surface) loaded in compression. They were able to identify differences in the architecture of bone based on the alignment of forces within the given bone. This confirms other research done in human and other animal models, which demonstrates that bone is able to remodel itself according to the lines of stress (Wolff's Law).

Generalized Bone Loss

An example of bones remodeling in a generalized fashion is the situation referred to as osteoporosis or osteopenia. In this case, a variety of factors can result in the activation of osteoclasts, which then resorb bone and lead to thinner, weaker bone (Figure 9). These influences include hormonal (such as menopause), or environmental (such as cigarette smoking for humans), but the most important relates to lack of exercise.

According to Wolfe's Law, failure to stress and stimulate bone by the mechanical forces generated by weight-bearing and muscles results in the activation of osteoclasts, leading to generalized loss of bone content and ultimately strength. The importance of exercise with respect to bone strength is well known in many human studies. This would suggest that any program which includes any significant amount of stall rest would promote the loss of bone.

We know from long-term studies in human females, as well as some anecdotal information from Russian astronauts, that after a certain amount of generalized bone loss, no amount of reversal of the mechanical or environmental influences will result in the recovery of bone substance and therefore strength. In other words, in these individuals bones were permanently damaged. In the case of the postmenopausal human females, this is demonstrated by relatively high fracture rates compared to age-matched males; and in the case of Russian astronauts, they were simply unable to walk once they were re-exposed to the gravity of earth.

Unfortunately, the early loss of bone is not easily detectable with simple tests such as x-rays. Human studies have demonstrated that more than 50% of the total bone mass needs to be removed before changes are identifiable on x-ray. Devices called bone densitometers are required to measure more subtle instances of bone loss.

Localized Bone Loss

Some of the same influences can occur in a localized way to involve just one bone or even a part of one bone.

One example of a negative influence to bone would be the lack of mechanical stimulation imposed by rest, or immobilization. When one extremity or a part of the extremity is immobilized (such as in a cast), it is deprived of the normal weight-bearing and muscle-pull influences which preserve bone integrity. Therefore, bone loss occurs in the extremity or bone which is immobilized. This extremity is then vulnerable to fracture once the immobilization or protected weight-bearing is removed. It is important to gradually reintroduce weight-bearing, and stress to this particular bone, so that it is allowed to regain bone mass prior to the application of full stresses.

In a similar manner that cast treatment or immobilization can protect bone from stress, resulting in bone loss, application of a mechanical stress-sharing (i.e., aiding the bone in bearing a stress) device to bone can have the same effect. An example of this would be the use of horseshoes. (The concept of stress sharing will be explained later in this paper)

Studies investigating the effect of horseshoe on the terminal phlanx are rare. One of the situations that has been extensively investigated in human orthopedics is the response of bones which are in close contact with metal. These studies include both plate and screws used for fractures, or metal and plastic joint replacement devices. These investigations identify the fact that when metal and bone are in the mechanical situation where applied stress is shared between the two devices, a phenomenon known as stress shielding occurs. This refers to the fact that because the metal shares the load, the adjacent bone is exposed to an altered load, and therefore becomes osteoporotic and weak. In some cases, weakness can lead to fracture around the prosthesis. In all cases, if the prosthesis is removed, the underlying bone is weak and requires careful reapplication of stress to prevent fracture.

(Dr. Fisher) (I) was not able to find any biomechanical studies related to the the effect of horseshoes on the biomechanical properties of the coffin bone. As described above (in the equine radius study), such investigations are difficult to perform. As one might expect, from our experience with joint prostheses and plates, a load sharing device such as a horseshoe would result in the nonphysiologic adaptations of the coffin bone, such as generalized loss of bone mass, as well as localized adaptations within the bone similar to what was described above (in the equine radius study). These adaptations (often focal weakness in the bone) may lead to a variety problems for both the bone and soft tissue.

Although horse shoes are not applied directly to bone, the shoes alter the normal stress pattern which would be accommodated by the hoof. Because the hoof is connected to the bone by the lamine, the bone then must make accommodations to stresses for which it has not been previously adapted for by evolution.

An example of altered hoof weight bearing stresses affecting bone would be a deformation of the hoof capsule resulting from the horseshoe. The horseshoe puts direct pressure on the sides of the hoof, causing contraction and then bone loss due to altered stresses.

The same type of bone loss (due to stress shielding) is seen when plates used for orthopedic fracture repair are applied directly to bone.

Another adverse influence resulting in the loss of bone is a loss of the bone’s blood supply. If bone is deprived of its blood supply (which is what likely occurs with laminitis and coffin bone rotation), it undergoes death of its cells, which

then fail to provide nutrients to the calcified structure; that structure then deteriorates. Shoeing causes a loss of blood supply to the hoof by arresting hoof mechanism, which in turn leads to bone loss. When bone loses its blood supply, it becomes a target for reabsorbtion by osteoclasts, it can become infected and is reabsorbed as part of the inflammatory reaction, or it can be reabsorbed because it is shielded from physiologic stresses.

The end result of these processes can be that a part of the coffin bone is resorbed by osteoclasts, or simply is sloughed off as "abscess material". This is based on the fact that bone is not just an inert material; to maintain its integrity, bone requires a blood supply and a stream of nutrients. Unlike in the situation of osteoporosis (where there is a scaffolding of remaining bone, to which new bone can be added), if the bone loss is due to a loss of blood supply (as in long term coffin bone rotation with separation in a hoof with most of the circulation gone), the entire structure of bone is now absent, and it is unable to be rebuilt. (figure 12)

Bone Gain (Net New Bone Formation)

Clinical observation tells us that net bone gain can occur in several situations. The most obvious is fracture healing. The fracture healing process most likely involves a variety of chemical and mechanical influences, which call both osteoclasts and osteoblasts to a given location to clean up old fractured bone and produce new bone. Fracture healing will not be discussed in any detail in this document.

All bones can be made to become denser and stronger by increasing loading, both by weight-bearing as well as by the use of muscles which attach to the bone. The exercise process must be controlled, however, because if the applied repetitive stress exceeds the ability of osteoblasts to keep up, then a fatigue fracture occurs (similar to that which occurs in metal which has been repetitively loaded). This is known as a "stress fracture". It is frequently seen in race horses due to repetitive loading of bones which are not ready to accept these loads. In other words, these bones have not been allowed to biologically strengthen themselves gradually prior to being excessively stressed.

We know in a general way from these clinical examples, that the application of stress to bone, according to Wolfe's Law, results in the growth of new bone. Another example from a slightly different clinical situation is the production of osteophytes. Osteophytes are a growth of new bone typically around the periphery of joints, usually in the setting of arthritis. Although this is presumably an adaptive phenomenon of the bone, to stabilize the joint, this explanation has never really been proven. In equine orthopedics, these osteophytes are often referred to as “side bone” and “ring bone”. (Figure 13 and 14) Osteophytes can also occur in response to traction. (Figure 14)

Conclusions

What implications should therefore be drawn from the application of the principles of bone remodeling to the pathology of the equine terminal phalanx? First of all, although little in the way of biomechanical studies have been done regarding the influence of horseshoes on the normal physiology of the terminal phalanx (coffin bone), it is clear from many human studies that there is likely dramatic alteration in the stresses received by the coffin bone during standing and walking. This undoubtedly causes architectural changes within the bone, and possibly overall loss of bone stock which cannot be replaced. The additional changes affecting the laminar connections are described elsewhere.

Removal of shoes, depending on the timing as well as other influences, may allow the coffin bone to remodel. It seems obvious, however, that promoting a situation which several million years of evolution adapted the coffin bone for – that is, not applying horseshoes and keeping the coffin bone ground parallel within the hoof capsule – would make the most biological sense. In other words, never putting shoes on the horse, and keeping the coffin bone ground parallel for even distribution of stress along the edges of the coffin bone, would make the most sense for the bone according to the arguments advanced in this report.

In terms of the health of bone, a physiologic amount of exercise, as opposed to complete stall rest, helps prevent bone loss (osteoporosis) and the need for additional recovery period.

In most cases, bone loss is recoverable once the conditions are changed to promote physiologic stress on the bone and to allow for the inflow of nutrients.

Finally, although we know, according to Wolfe’s Law, that application of various forces across bone results in bone production (ring bone traction spurs), the specific mechanisms for many of these processes have yet to be elucidated.