The fetlock joint is, arguably, the joint that makes a horse a horse,” said Larry Bramlage, DVM, MS, Dipl. ACVS, a partner at Rood & Riddle Equine Hospital in Lexington, Ky. “Its unique anatomy and physiology allow the high-speed, medium-distance activity that has led to the unique place for the horse in society, historically and currently. The fetlock is a joint, a shock absorber, an energy storage system, and a stabilizer of the distal limb.”

During each AAEP convention, one veterinarian is chosen for the honor of giving the Frank J. Milne State-of-the-Art Lecture on a topic of importance to the profession. In 2009 Bramlage was selected to discuss the orthopedics of the fetlock joint, from disease and injury to surgical repair.

His is a familiar face, based on his history as an internationally recognized equine orthopedic surgeon, past president of the American College of Veterinary Surgeons and the AAEP, many awards, more than 100 published scientific articles, and his participation in the AAEP’s On-Call program (speaking to the media in equine injury cases at live-broadcast equine events).

Bramlage began his presentation by reporting that the fetlock joint is the joint on which he most commonly operates in his practice. “In the last 10 years, I’ve operated on almost 11,000 horses, and almost 40% of those were fetlock cases,” he said. “This was a diverse population of foals, yearlings, and adults. Many horses stay in our practice their entire lives, so we not only see the follow-up, but also the horse’s racing career. For the last 19 years, I’ve mostly worked on (Thoroughbred) racehorses, so their influence on my understanding of injury to the fetlock joint is quite large.”

**Fetlock Joint Construction**

“The fetlock joint is constructed like a suspension bridge, with structural members incapable of supporting its loads until the appropriate ligament tenses and supports the bone,” Bramlage explained. “It is the most fascinating of the complex of joints that allows a horse to move at high speeds and over rough terrain with little conscious concern. The bones and soft tissues of the joint are so interwoven that we can’t consider them separately.

“Because of the fetlock joint’s complexity, it is vulnerable to a variety of traumatic and developmental problems that are the veterinarian’s purview,” he went on. “Degenerative arthritis in horses is usually secondary to developmental abnormalities or traumatic injury (we won’t address infection today). Developmental abnormalities and traumatic injury are similar—both shed debris into the joint, which is a huge consideration for all joint diseases.”

He described in depth the normal functions of bones, cartilage, soft tissues, and fluids within the fetlock joint—and joint disease that compromises those functions.

**Articular (Joint) Cartilage Function**

Bone ends in joints naturally contact each other when a horse moves, and they must slide easily past each other when this occurs, otherwise the bone surfaces would “grab” each other and stop motion of the joint. Key to smooth joint motion is the articular cartilage covering the ends of bones in the joints and providing a smooth surface for contacting bones to slide along. Bramlage explains cartilage function as follows: “Articular (hyaline) cartilage is like a sponge; it sucks in water (proteoglycan molecules), and this inflates the cartilage ... weight-bearing squeezes it out. Weeping of the fluid (out of the cartilage) allows near friction-free motion, just like a sponge on a countertop. Water lubricates the cartilage like sliding a very wet sponge along the counter. When the weight comes off, the water sucks back into cartilage, and that is repeated over and over.”

Of particular importance in cartilage function are collagen arches, whose “legs” anchor deep within the bone and crest in the surface cartilage, anchoring it to the bone surface. Proteoglycan molecules in the cartilage lubricate/protect the arches; if proteoglycan is depleted, the collagen is more likely to be damaged, which can allow erosion of the surface cartilage (loss of this smooth, lubricating layer).

In an inflamed joint, proteoglycan can be destroyed quicker than it can be produced.
Over time the result is less joint lubrication, followed by surface wear/cartilage erosion. “Surface wear is irreversible,” he warned. “It progressively destroys the most functional part of the cartilage at the point of wear. We can implant chondrocytes, stem cells, or cartilage grafts to fill in the defect—and all of this is okay for humans that at most go up and down stairs, but not racing horses. As soon as you put them back into exercise, they skin that (repaired) collagen right off the bone. The real breakthrough will occur when someone can recreate the collagen anchorages to bone.”

**Joint Degeneration: The Birth of Osteoarthritis**

Bramlage noted the term “degenerative joint disease” is often erroneously used as a catchall diagnosis for joint problems, when the term should be reserved for joints with irreversible changes and reduced joint spaces (between the bones). He also advised that the name of the game when fighting this condition is prevention, because there is no cure in horses.

“How does a horse create degenerative arthritis in two years that takes humans 20 years?” Bramlage asked the audience. “Because of their tremendous size, relatively small joint surfaces, high stress activity, and high pain tolerance. Poor medication plans sometimes contribute.

“We’ve made some huge strides recently in understanding the intra-articular (within the joint) degeneration cycle; we know now that debris fuels much or most of the inflammatory cycle that results in secondary degenerative arthritis in the fetlock,” he said. “Bone’s response to trauma is extremely important in the fetlock.”

To understand how debris and chips cause increasing problems in a joint, we must first understand how fractured bone heals. An important concept is strain, defined as the amount of bone motion at the gap, or fracture line between bone fragments, divided by the size of the gap. “Counterintuitively, a very small gap will have a higher strain than a large gap (motion divided by a small number results in a higher strain than the same motion divided by a larger number),” he explained.

Bramlage said when bone is fractured (whether it’s a long bone fracture or a joint chip), the injury can heal together completely with new bone tissue if the pieces are held together with very little (4% or less) strain. This is the ideal situation, and the one targeted by surgeons stabilizing one piece of bone to another. However, this isn’t always possible, depending on the fracture size and location. If the strain at the fracture line is higher than 4%, softer tissues such as cartilage, fibrous tissue, or granulation tissue must fill the gap first, then bone can form once those softer tissues have stabilized the fracture line enough to reduce strain below 4% and allow new bone formation.

Interestingly, if strain is too high at a fracture line for any tissue to fill in the gap, the parent bone will actually soften and dissolve at the fracture line, widening the fracture gap and thereby reducing the strain to allow soft tissues to start the healing process. Here’s where this phenomenon causes particular problems in cases of joint chips and other joint surface fractures, says Bramlage: “As long as the raw surfaces of (fractured) bone are exposed to each other, the bone will try to heal. But it’s often a high-motion environment, so strain is high and needs to be reduced. The parent bone softens under the chip, trying to heal the bone together. It doesn’t matter to parent bone if it’s a sliver or big chunk of bone broken off, it responds the same.

“Thus, the gap widens, and (the dissolved bone) results in copious debris shedding,” he went on. “Because of the loading (of the fractured joint surface) with motion, the progressive destruction of the parent bone at the interface with the fragment or OCD (osteoarthritis dissecans) results in tremendous amounts of bone matrix and mineral continually shedding into the joint. This constant debris shedding is the primary cause of joint degeneration, rather than the ‘stone in your shoe’ phenomenon (in which it was believed that the bone fragment physically obstructs proper joint movement). In most joints, fragment stability is not possible to achieve.”

Joint chips are typically the result of repetitive trauma and microfractures, rather than a single event, he adds. Thus, by the time a bone chip separates from the parent bone, bone softening/dissolving has been ongoing for some time to heal microfractures that occurred prior to complete separation of the fragment. The debris and accompanying inflammatory processes that deplete proteoglycan, along with the physical abrasion of joint cartilage caused by the chip, cause the irreversible cartilage damage of degenerative arthritis.

“Surgery, however, can stabilize the bone pieces so they can heal together and get it over with, or remove one bone (the chip) to stop the secondary healing response,” said Bramlage. He noted that chips can often be well-tolerated in horses that aren’t working hard, or broodmares, but that athletes generally need them removed.

**Joint Debris Removal**

The fetlock joint is capable of removing debris circulating within it, and it does so on a fairly regular basis, Bramlage said. The problem comes when the debris load is too high or the fragments are too large to be removed naturally. To remove the debris, first the joint’s lubrication will decrease (via reduced proteoglycan production) and fibrin will be produced to trap bits of debris at the “cul-de-sacs” away from the joint’s weight-bearing surfaces. (Maintaining lubrication would allow debris to keep floating throughout the joint.)

The fibrin-coated debris then sticks to numerous tiny villi, or fingerlike projections of soft tissue in the joint cul-de-sacs. Once the debris is trapped, special cells break down and remove it using enzymes that induce a temporary inflammation. Once the debris is gone, this process stops, lubrication returns to normal, and no harm is done. However, if debris is constantly being produced, so are the enzymes, inflammation, and
reduced lubrication; thus, proteoglycan is depleted and we’re on our way to arthritis.

“Continuously shedding debris is like a gas fire raging in the joint,” he commented. “Put out the fire by eliminating the (cause of the) debris. The joint can’t survive if acute debris is not handled.”

Bramlage noted that there are only so many villi in the joint, and large amounts of debris can “max out” their debris removal capacity. “The villi get matted together in more progressive cases,” he commented. “This is seen in jumpers with chronic hind limb problems and as a rapid onset problem in the forelimbs of some racehorses.”

Surgery vs. Injections for Joint Chips

Surgery isn’t indicated for all cases of joint disease—just the ones with separated bone fragments. In such cases, many owners prefer joint injections over surgery to alleviate clinical signs (i.e., lameness,) believing they are cheaper and simpler than taking the horse in for surgery. However, Bramlage states that the two treatments are far from equal in their effects, and as far as finances go, the opposite is often true, especially for high-performance horses.

“The direct cost of surgery is often very low compared to the long-term decrease in the quality of the horse, especially those with high-level earning potential,” he explained, noting that surgery is best used early to prevent arthritis, rather than to try to resolve arthritis after other therapies have failed. “Surgery stabilizes the reconstructible joint surfaces and removes the damaged bone that would shed debris and further injure the joint. I’m not trying to sell a boatload of surgeries, but surgery is often the most effective, fastest cure. Some of the diseases we used to think were nonsurgical are actually very good candidates.”

Medications injected into the joint can act in two ways: alleviating lameness signs without improving the health of the joint in any way (symptom-modifying, i.e., some painkillers), and/or promoting healthier joint cartilage (disease-modifying). The latter is, of course, preferred over the former.

Some commonly used joint medications, such as corticosteroids, can minimize inflammation and thereby protect the soft tissues of the joint, he noted. “However, medications cannot stop physical debris shedding within the joint that results from unstable bone,” he said. “If we don’t take care of the trauma that resulted in the debris, the situation will keep cycling. It is this physical debris … that does the ongoing permanent damage to the hyaline cartilage and, therefore, to the joint.”

One of the biggest mistakes made with these cases is considering joint injections as scheduled “maintenance” to keep a horse performing, he noted. “The worst choice that can be made is to medically treat a horse with a surgically resolvable disease, allow the joint to suffer irreversible damage, then attempt to surgically remove the cause. This provides the worst of both possibilities: The most possible expense and the shortest possible career.”

Fetlock Joint Arthrodesis (Fixation)

The final topic Bramlage covered was fetlock joint arthrodesis, or surgical immobilization. He noted there are two main reasons to immobilize this joint: crippling of the joint that renders it unusable, and pain severe enough the horse refuses to use the joint. Injury, severe laxity or contracture, and severe arthritis can all be indications for arthrodesis. Particularly in the latter case, motion in the joint that has worn out is what causes the pain, and stopping the motion stops the pain.

It’s important to note this is a salvage procedure for companion or breeding stock animals only, and it will not restore athletic soundness. It can, however, resolve pain due to movement almost immediately, making the horse more comfortable. This is especially helpful in protecting the paired limb from laminitis.

The joint is plated from the front of the limb for arthrodesis, but the plate must be supported by supporting the joint from the back, as well. In cases of sesamoid rupture, tension wires wrapping behind the joint might be needed to help support it; in other cases, the sesamoids can be used. He reported that cases with poor blood supply in the lower limb, particularly injury cases, tend to pose the biggest problems with complications. If blood supply isn’t adequate, healing might not occur quickly or at all, and lack of blood supply to the hoof can result in sloughing (shedding) of the hoof weeks later in a process similar to that seen in frostbite in people in which the fingernails shed weeks after the insult.

“Results improve with experience; our success rates with these have improved from 69.2% in 1978-1986 to 87.5% in 2004-2009,” he reported. “This is the most dependable thing in our hands for handling really serious fetlock injury. You have to be able to afford it—they run $10,000-$15,000—but for a valuable enough breeding animal, it can be really worthwhile.”

**“It is at this point impossible to recreate normalcy in a damaged joint surface in an adult horse by any means. The lack of a solution for degeneration places a premium on prevention.”**

**DR. LARRY BRAMLAGE**

**Workload and Detection/Treatment**

Often the complaint for a horse suffering from joint problems is simply reduced performance, which tends to be noticed much more quickly in high-performance horses than those that aren’t working as hard.

“High-workload horses have an advantage in that problems cause rapid, intense signs, so we catch the disease earlier,” he explained. “High-intensity exercise forces earlier decision making, which is a good thing. With lower-level activity signs are more insidious, so we might think we’re maintaining the joint (with conservative measures), although there are considerable dangers in that. Less intense exercise usually leads to later intervention, and debris in the joint can permanently ‘sand down’ the articular (joint) surfaces. You won’t get that (articular cartilage back).”

When physically damaged, the bone and cartilage within a joint can heal to a point. However, the quality of that healed tissue is not as strong and resilient as the original healthy tissue, particularly the attachment of hyaline cartilage to the bone. Each joint has an upper limit for how much joint surface can be lost and tolerated.

“It is at this point impossible to recreate normalcy in a damaged joint surface in an adult horse by any means,” Bramlage stated. “The lack of a solution for degeneration places a premium on prevention.”
“Remember the red, white and blue – the original formula.”

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