Traumatic Joint Injuries and Diseases

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Introduction

Traumatically-induced disease of joints is a very common affliction of athletic horses. Many of these cases respond well to medical treatment, the principal ones of which are nonsteroidal anti-inflammatory agents, corticosteroids, hyaluronic acid and polysulfated glycosaminoglycans (Adequan). Over the recent years, there has been a lot of information collected regarding these treatments. Traumatic joint injury may range from a mild "sprain", all the way to a severe catastrophic injury with complete loss of support for the joints. Traumatic joint disease represents one of the most common problems of the horse. Before discussing the various processes and how we can diagnose and treat them, a review of some basic anatomy and physiology of joints is appropriate.

1. Treatment
2. NSAID's
3. Legend
4. Adequan
5. Oral Glycosaminoglycans
6. Corticosteroids

Traumatic Joint Disease

Traumatic joint disease includes a number of conditions that are the result of trauma.

1. Synovitis - inflammation of the synovial membrane
2. Capsulitis - inflammation of the fibrous joint capsule
3. Articular cartilage and bone fragmentation or fracture
4. Ligamentous tearing
5. Osteoarthritis - osteoarthritis is the end result of most severe injuries or inadequately treated injuries. It is represented by progressive loss of articular cartilage on the surface of the bones, which is permanent.

SYNOVITIS AND CAPSULITIS

Figure 7a. Possible etiopathogenic factors for articular cartilage degeneration in the horse. Click to enlarge.
Injury of the synovial membrane and fibrous capsule (synovitis and capsulitis) goes together. It is a common early consequence of day to day cyclic trauma in athletic horses. Acute synovitis and capsulitis can cause significant clinical compromise directly due to fluid swelling and pain and equally important, may contribute to the degenerative process in joints by the release of enzymes, inflammatory mediators, and cytokines. A diagram of the importance of these processes is outlined below (Fig 7a and b).

Figure 7b. Possible factors in enzymatic degradation of cartilage matrix. Click to enlarge.

There are a number of enzymes produced by inflamed synovial membrane that are considered important:

1. neutral metalloproteinases
2. serine proteinases
3. cysteine proteinases
4. aspartic proteinases

Other inflammatory mediators of importance include prostaglandins and free radicals. In addition, cytokines, or intercellular messengers, have achieved much significance recently. The significant ones so far are interleukin-1 (IL-1) and tumor necrosis factor _ (TNF_). Cytokines can be released by inflamed synovial membrane cells as well as cartilage cells (chondrocytes). These cytokines in turn can cause other cells, particularly the chondrocyte and the synovial cell, to release further enzymes. The synovitis process can be from direct trauma or alternatively can come by cartilage and bone debris stimulating the synovial membrane to produce prostaglandin E, cytokines and neutral metalloproteinases.

Effect of Enzymes

**Neutral metalloproteinases** - The most significant enzymes are neutral metalloproteinases (also called matrix metalloproteinases). Three that we think are of particular importance are collagenase, stromelysin and gelatinase. Collagenase is produced by a wide variety of cells and cleaves collagen. Stromelysin has a wide variety of substances that break down but its ability to degrade the various proteoglycans of the cartilage is critical. Gelatinase degrades denatured type II collagen (the collagen specific for articular cartilage) as well as other substances. These important enzymes are secreted in an inactive form but collagenase is activated by stromelysin and stromelysin is probably most commonly activated by plasmin. Some inhibitors to these enzymes have also been identified.

**Serine proteinases** - These are plasminogen activators and can also be produced by inflamed synovial membrane. These plasminogen activators cleave plasminogen to active plasmin and this cascade is important in activating the metalloproteinase enzymes.

**Cysteine proteinases** - Their role in cartilage degradation is uncertain but they are capable of breaking down proteoglycans in the cartilage.
**Prostaglandins**
Prostaglandins (primarily E group) are produced in inflamed joints and can cause a decrease in the proteoglycan content of the cartilage matrix by decreasing synthesis as well as enhancing degradation. We know that prostaglandin E\(_2\) can be released from synovial cells by interleukin-1. The presence of prostaglandin E\(_2\) in synovial fluid from inflamed joints has been demonstrated in the horse. In addition to depleting proteoglycans, PGE\(_2\) in joints also enhance pain perception as well as possibly promoting bone demineralization.

**Oxygen-Derived Free Radicals**
These include super-oxide anion, hydroxyl radicals and hydrogen peroxide and may be released from injured joint tissues. Studies have demonstrated cleavage of hyaluronic acid by free radicals. Hyaluronic acid provides the lubricating qualities to the synovial fluid. Free radicals also break down proteoglycans and there is evidence for a role in breaking down collagen as well.

**Cytokines**
Much of the destructive proteinases previously described are released by cytokines. Cytokines are small soluble proteins produced by one cell that affect the activity of other cell types. Studies of cytokines in joint tissues suggest that IL-1 and TNF\(_\alpha\) influence the synthesis of the important metalloproteinases by both chondrocytes (cartilage cells) and synovial cells and are important agents in joint disease. Both IL-1 and TNF\(_\alpha\) have been shown to be produced by synovial cells and may therefore be of importance in the deleterious effects of synovitis on articular cartilage. It is considered that the normal turnover of the critical materials of the articular cartilage is regulated by the chondrocytes under the control and influence of cytokines and mechanical stimuli and that cartilage degradation in association with disease represents an exacerbation of these normal processes. It is widely accepted that cytokines induce proteoglycan depletion in articular cartilage by either increasing the rate of degradation or decreasing synthesis in association with the release of proteinases and prostaglandins from chondrocytes. Studies done in the horse to date have involved the use of a human recombinant IL-1. Recently the gene sequence for equine interleukin-1 was identified at CSU by Dr. Rick Howard and this will hopefully lead to good specific studies with equine tissues as well as using equine IL-1.

**Summary**
The above pathways represent a number of options for the destruction of the extracellular matrix of articular cartilage. Articular cartilage degeneration is the critical factor when we define disease as osteoarthritis or degenerative joint disease. Much of our treatment modalities are aimed at preventing this event and these various substances previously described will be referred to when discussing the medication options.

In addition, synovitis and capsulitis are important sources of pain and compromised athletic ability. The effusion and reduced range of motion can produce permanent changes in both the synovial membrane and fibrous joint capsule as well.
Clinical Signs and Treatment Of Synovitis And Capsulitis

The most obvious sign usually with these cases is accumulation of fluid in the joint, also called synovial effusion. The joint is puffy and on palpation there is fluctuance due to the cavity of increased fluid. If the lameness is particularly severe, radiographs are always taken to rule out a severe fracture. In all cases, radiographs are ideal to eliminate the presence of any bone damage and to see if there are any chronic changes present. If radiographs do not show a fracture and the lameness is not extremely severe (this could indicate a joint being infected), then we go ahead and treat it as a case of synovitis and capsulitis (Fig 8).

Early and aggressive treatment of synovitis and capsulitis is important to:

1. Alleviate the immediate effects of inflammation, including pain and reduced function
2. Prevent the development of permanent fibrosis in the joint capsule (as a result of uncorrected inflammation), which in turn will then cause decreased motion and decreased shock absorption in the joint
3. Prevent or minimize the development of osteoarthritis (OA)

There are a number of treatment options available for acute synovitis and capsulitis. In many instances, a combination of these options is used.

Physical Therapy
Trainers of equine athletes have long recognized the value of the simple modalities, cold water and ice, in bringing down acute inflammation. These methods have been done routinely and deal with many problems before a veterinarian is required. The principle is to reduce acute inflammation and get rid of inflammatory fluids that could later lead to permanent fibrosis. The comfort to the patient has also been well recognized. The use of physical therapy in the horse lags behind that in humans where people are specifically trained in physical therapy. Passive flexion of joints after surgery is practiced but we do not have the options of nonweightbearing manipulation such as is done in humans. Our best simulation of such a technique is swimming. I feel swimming is useful after suture removal following arthroscopic surgery as it enables weightbearing motion of joints. I prefer to use swimming early in the convalescence period, rather than late. It is a subjective feeling that training a horse with swimming and then tracking immediately after could be deleterious to the joints. Different muscles are used in swimming and lack of muscle tone could affect microstability in the joints. The use of water treadmills is popular in certain areas. This can be an effective way of decreased
weightbearing on limbs while maintaining an exercise protocol. It also can be used as a step further down the road from swimming.

Other physical therapy modalities have been used to treat injured joints, including *therapeutic ultrasound* and *soft lasers*. There is no controlled work in the horse, but anecdotal information from its effect with human sprains and other soft tissue injuries lead one to suspect that with the right regimen they can be extremely useful. Electrostimulation has also been used but again there is no controlled data.

**Nonsteroidal Anti-inflammatory Drugs**

Nonsteroidal anti-inflammatory drugs (NSAID) include phenylbutazone, flunixin meglumine (Banamine), naproxen, meclofenamic acid and aspirin. Phenylbutazone has been the most commonly used due to its predictable action and it being inexpensive. NSAIDs work by inhibiting prostaglandins, which have been mentioned as significant factors both in causing damage to articular cartilage but also potentiating pain. Used at the correct dosage level, the side effects are minimal in adult animals. However, the drugs are toxic in foals and in ponies and should only be given in these situations when there is a critical need. Phenylbutazone will also cause problems in adults at high dose rates (4 gm/day). Toxic effects of NSAIDs include gastrointestinal ulceration and inflammation and nephrotoxicity (kidney toxicity). These agents are commonly used as the first line of drug therapy with joint injury. Phenylbutazone is used by the author for seven days after surgery to minimize inflammation. Some controlled work in humans has shown not only beneficial effects directly after surgery but also long term in terms of function and success rate. Failure to respond to nonsteroidal anti-inflammatory drugs is often an indication for further diagnostic examinations as well as the use of other medications, particularly intra-articular ones.

**Hyaluronic Acid**

Figure 9. Click to enlarge.

Hyaluronic acid is also known as sodium hyaluronate, or hyaluronan (the more correct term). Hyaluronic acid (HA) is a glycosaminoglycan (previously discussed) (Fig 9). It is a normal component of joints but it is generally agreed that there is some depletion of the amount in diseased joints and certainly the function.
HA is an integral component of both synovial fluid and articular cartilage in normal joints. Synovial fluid HA is produced by the synovial cells of the synovial membrane. Other HA that is incorporated in the matrix of articular cartilage is synthesized locally by the chondrocyte. HA confers the property of viscoelasticity to synovial fluid and is responsible for boundary lubrication in the synovial membrane and also is a factor in the lubrication of articular cartilage. HA also influences the composition of the synovial fluid by acting as a high molecular weight barrier over the synovial membrane (called steric hindrance) and preventing active plasma components and leukocytes (white blood cells) from the joint cavity (Fig 10). It is also felt that solutions containing HA change the attraction of various other inflammatory cells. The HA that is in the articular cartilage is important in acting as a backbone for aggregations of proteoglycan molecules (discussed above) and aiding in the compressive stiffness of the articular cartilage.

Possible mechanisms of action of sodium hyaluronate that is injected - Beneficial effects after intra-articular administration of HA have been reported in a number of studies in the horse as well as other animals. However, the mechanism through which beneficial effects have been achieved remains controversial. The therapeutic effect of exogenously administered HA may result from the supplementation of the actions of depleted or depolymerized (decreased molecular weight) endogenous (formed naturally by the joint tissues) HA or alternatively result from other properties that have been suspected based on experimental work (discussed later).

It has been assumed by many for a long time that the primary therapeutic effect is by supplementation of depleted HA. However, while alterations in synovial fluid HA concentration and molecular weight in various joint diseases have been described, the results are conflicting. Generally there is a reduction in synovial HA concentration and molecular weight with equine joint disease. On the other hand, a recent study from equine joints with acute traumatic synovitis is not significantly different in HA concentration than from normal fluids. In the same recent study, the molecular weight of synovial fluid HA was not significantly different from normal equine joints compared with those of acute or chronic arthritis.

It is known that the half-life (time for half of the drug to be eliminated) of intra-articular HA injected into normal equine joints is 96 hours. This half-life is reduced in diseased joints. It is felt that although most of this HA is rapidly cleared from the joint, some remains associated with the synovial membrane and that it provides beneficial activity in the intercellular spaces of the synovial membrane. We know that HA has direct anti-inflammatory effects in inhibiting inflammatory cells. It has also been suggested that HA may also reduce interactions of enzymes or cytokines through the steric hindrance project. It is most recently felt that the decreased inflammatory cell activity is through interaction of HA with cell receptors on the white blood cells. Our research with intravenous hyaluronic acid leads us to suspect that the receptor mechanism is important.
It has been commonly proposed that the injection of HA into a diseased or injured joint results in increased synthesis of high molecular weight endogenous HA by the synoviocytes. Much of this work relies on *in vitro* (laboratory studies with tissue taken from the animal) evidence. These results, however, fit well with our clinical observations of a fairly long term effect when we know that the drug is not lost in the joint fluid (very long, or even less in the blood stream).

**The use of intra-articular HA in clinical equine joint disease** - The clinical use of HA for intra-articular treatment of equine joint disease was published in 1970 from Sweden in which cases of traumatic degenerative equine arthritis were treated with methylprednisolone acetate (a corticosteroid) versus HA/methylprednisolone acetate combination in 20 racing Thoroughbreds and Standardbreds. The investigators concluded that the combination of HA and methylprednisolone acetate (Depo-Medrol) resulted in a better and more lasting improvement than the corticosteroid alone. In 1976, other Swedish veterinarians published on the treatment of equine traumatic arthritis with intra-articular HA alone in 54 joints of 45 racehorses previously treated unsuccessfully by other means. Through a one year observation period, 38 of 45 horses were free of lameness and 32 returned to the racetrack after treatment. Since these early reports, numerous clinical and experimental studies have been conducted to evaluate the efficacy of HA in the treatment of equine joint disease. These clinical reports have generally supported the use of HA but in many of them, the evaluations are subjective (personal feeling) and the definitions for what success is are absent. The duration of post-treatment observation periods are varied and some studies were of short duration. A number of the studies have implied that HA is successful in the treatment of osteoarthritis but in most instances, joints have not had x-ray changes and blocked out with intra-articular analgesia (more typical of synovitis and capsulitis).

There have been some more objective studies in the horse done with bilateral osteochondral fractures created by arthroscopy and also chemically-induced synovitis. In one study a positive response was seen, whereas in the other no response was seen. There have been various studies in other experimental animals in which a chondroprotective (beneficial effects that protect the cartilage from degeneration) effect has been proposed. However, in a recent study in sheep the work implied that after injecting hyaluronic acid intra-articularly the sheep moved better and put more weight on the limb but degenerative changes occurred in the articular cartilage.

It is the subjective feeling of the author that mild to moderate synovitis and capsulitis will respond well to intra-articular hyaluronic acid, particularly in the fetlock joint. However, recent experience with intravenous hyaluronic acid has led to it being used more commonly in this fashion.

**Intravenous Hyaluronic Acid (Legend)**

A new formulation of HA for intravenous use has been approved recently in horses. It is marketed by Bayer as a product called Legend. My subjective impression is the drug can be very useful and in some instances has a more obvious beneficial effect than the intra-articular drug.

![Figure 11a. Total protein concentrations on day 71. Significant differences (P less than 0.05) are noted by the same letters. Click to enlarge.](image-url)
Figure 11b. Prostaglandin E2 concentrations on day 72. Same letters indicate significant differences (P less than 0.05). Click to enlarge.

Figure 11c. Synovial membrane scores of cellular infiltration. Significant differences (P less than 0.05) between groups are identified by the same letters. Click to enlarge.

Figure 11c. Synovial membrane scores of cellular infiltration. Significant differences (P less than 0.05) between groups are identified by the same letters. Click to enlarge.
We recently completed an experimental investigation of intravenously administered HA using a carpal chip model with the horses exercised on a treadmill. Twelve horses were used and six received 40 mg of Legend intravenously on days 13, 20 and 27 after chip fracture and six horses were treated similarly with a placebo of physiologic saline. Seventy-two days after surgery, the joints subjected to osteochondral fragmentation had increased synovial fluid total protein, glycosaminoglycan and prostaglandins E2 levels compared to contralateral joints without fragments. They also had increased synovial membrane inflammation. With treatment with intravenous HA, there were significantly reduced levels of synovial fluid total protein and prostaglandin E2 compared to joints with fragments in nontreated horses and also reduced synovial membrane cellular infiltration and vascularity (Fig 11a-d). Last but not least, horses treated with intravenously administered sodium hyaluronate were also determined to have a reduced degree of lameness compared to nontreated horses. We also showed no deleterious effects on articular cartilage.

The results of this study were very impressive but we still don't know the mechanism as to why intravenous HA is achieving benefit. The time that HA lasts in the blood stream is extremely short, so we assume at this stage that the beneficial effects must be due to the HA localizing in the synovial membrane and working through receptors stimulating other events. We do know that the synovial membrane has an excellent blood supply so that intravenous administration may provide more access to the synovial membrane cells than intra-articular administration. A possible mechanism is illustrated in Figure 12.

**Polysulfated Glycosaminoglycans (Adequan)**

Polysulfated glycosaminoglycan (PSGAG) is currently the only member of a group of polysulfated glycosaminoglycans that include, in addition to Adequan, pentosan polysulfate (Cartrophen®) and glycosaminoglycan peptide complex (Rumalon®). PSGAG has been traditionally used where articular cartilage damage is considered to be present rather than in the treatment of acute synovitis. It has also been used prophylactically to prevent day to day loss of cartilage components.

There have been numerous studies of PSGAG. The structure of PSGAG is very close to heparin. PSGAG has been shown to inhibit the effects of various enzymes associated with cartilage degeneration, including both collagenase and stromelysin, serine proteinases and others. PSGAG also has been shown to have a direct inhibitory effect on PGE2 synthesis and it is suggested that there is an anti-IL-1 effect. PSGAG also stimulates the synthesis of hyaluronic acid in the horse.

The protective effects of PSGAG on equine cartilage are not accepted by everybody. Although it was initially reported that PSGAG causes increased collagen and glycosaminoglycan synthesis in cartilage cultures from normal and osteoarthritic horses, more recent work by another investigator showed little effect on PG degradation. We need more research on the precise mechanisms of action of PSGAG and its interactions with cytokines involved in joint disease.

In live animal studies, the chondroprotective effect of PSGAG was first shown in dogs in which osteoarthritis was experimentally induced with meniscal removal. Work with an arthritis model in rabbits showed that there was a lower neutral metalloproteinase activity, increased chondrocyte counts, and maintenance of proteoglycan content. Both a prophylactic (preventing) and therapeutic (treatment) effect have been shown in experimental models with dogs.
**Studies in the horse** - We investigated the effects of intra-articular PSGAG on an experimental model of osteoarthritis in horses some years ago. Briefly, we showed that if we had chemically-induced degeneration developing in the cartilage, Adequan could greatly prevent it. However, if we made a defect in the cartilage, Adequan did not heal the defect. From this we concluded that the presence of Adequan could reduce ongoing degradation in the articular cartilage but could not heal a defect that was already there. We have seen good responses to PSGAG after surgery when there is significant cartilage degeneration. Although we know it does not heal defects left in the joint (discussed in more detail later), we hope (and presume) that it does decrease the rate of further cartilage degradation that probably ensues in most joints.

**Potential complications of intra-articular injection** - After intra-articular Adequan had been used for a few years, some veterinarians felt there was an increased risk of infection after intra-articular therapy. Based on research done here at CSU, we did demonstrate that PSGAG could have a greater potential in this regard. There were two important findings that came out of this research. The first was that we only need 100 organisms to infect a joint (compared to 1,000,000 to infect a skin laceration). This confirmed the observations of many that aseptic technique when injecting a joint was critical. The next stage of our research was with a subinfective dose simulating chance contamination. When Adequan was given at the same time, it potentiated this risk of infection. In a second study, we showed that conjunctive administration of a small dose of antibiotic prevented any infections.

In the meantime, the concomitant development of intramuscular Adequan led to it being used more frequently than intra-articular therapy. I still like to use intra-articular Adequan in a horse with severe cartilage damage, at least for the first injection after surgery. On the other hand, intramuscular use is far more frequent. However, aspects of how much intramuscular drug reaches the joint and its over-efficacy compared to intra-articular Adequan is still controversial. The dose rate for intramuscular Adequan is 500 mg. Although the manufacturers state it should be given every four days, we tend to use it weekly. The dose rate for intra-articular Adequan is 250 mg.

**Oral Glycosaminoglycans**

There have been a number of oral glycosaminoglycan products that have become available for horses recently and we are frequently asked questions with regard to their efficacy. Current products available include a purified chondroitin sulfate product from bovine trachea (Flex-Free®) and a complex of glycosaminoglycans and other nutrients from the sea mussel, *Perna canaliculus* (Syno-Flex®). More recently, a combination of glucosamine hydrochloride, chondroitin sulfate and what is described as a mixture of other PSGAGs has been marketed as a "nutriceutical" (Cosequin®).

Individual veterinarians and owners have commented on what they consider to be positive results. Cosequin has been evaluated using the Freund's adjuvant model of inflammatory joint disease and no benefit was demonstrated on clinical signs (lameness, stride length, carpal circumference, carpal flexion) in synovial fluid protein parameters.

On the other hand, the oral administration of glucosamine sulfate has been associated with decreased pain and improved range of motion compared to placebo in a controlled clinical trial in humans. In another trial, glucosamine sulfate was as effective as Ibuprofen at relieving symptoms of osteoarthritis in people. *In vitro* studies using glucosamine sulfate have demonstrated increased glycosaminoglycan and proteoglycan synthesis and *in vivo* studies have demonstrated an anti-inflammatory activity through inhibition of enzyme activity and free radical production.

The other question frequently asked is, is the oral product absorbed sufficiently? In studies done, there has been some absorption of an intact molecule but we still await demonstration of this in the horse.

**Intra-articular Corticosteroids**

Corticosteroids are the most controversial drug used in treating joint injury and synovitis and capsulitis in the horse. Statements have been made in the popular press that have no scientific basis. It has been implied by some that intra-articular corticosteroids have been replaced by hyaluronic acid and polysulfated glycosaminoglycan but this is not the case. Many clinicians have returned to or persisted in the use of corticosteroids and in athletic horses this use is common. The first report of intra-articular corticosteroid use in the horse was in 1955. Since then, the untoward effects of intra-articular corticosteroids have been documented by some and questioned by others. More recently, we have attempted to critically evaluate the specific effects of corticosteroids in equine joints and these results are helping us identify a more definite role for these agents in the management of joint disease. Corticosteroids are the most potent
anti-inflammatory drug we have and many beneficial effects have been recognized (discussed further below). The big problem has been the emphasis on the deleterious effects.

The first paper indicting corticosteroids as harmful in the horse was written by O'Connor in 1968. The report was based on some papers in the human literature. The statement, "An endless destructive cycle is set into motion which, if continued, will produce a steroid arthropathy which can render the horse useless" was referenced and the reference was an abstract written by an anonymous author. Six other human-based references were quoted in this paper to compare corticosteroids to Charcot-like arthropathy. Charcot's arthropathy is a neurogenic disease that results in the loss of sensation, loss of proprioceptive control, instability and arthritis (most often seen as a sequel to syphilis). There has never been any scientific demonstration of a comparable response associated with corticosteroid use in horses. The insinuation that corticosteroids "deaden" the joint is dangerous and has caused considerable alarm. A noted veterinary pharmacologist made statements in a chapter on corticosteroids in his textbooks that included "A patient on corticosteroids can walk all the way to the autopsy room" and "A horse can wear a joint surface right down to the bone running on a glucocorticoid-injected joint".

It has recently been questioned whether corticosteroids alter the destructive course of joint disease in humans and in horses. More recent studies looking at the histologic and biochemical changes in equine articular cartilage under the influence of corticosteroids with or without the added effect of exercise have questioned some of the previous dogma.

Effect of corticosteroids - Corticosteroid effects are exerted through an interaction with steroid-specific receptors in the cellular cytoplasm of steroid-responsive tissues. The corticosteroid binds to the receptor and induces changes in the transcription of genes coding various proteins that produce the hormonal effects. Corticosteroids are potent anti-inflammatory agents and inhibit inflammatory processes at virtually all levels. The major effect is their inhibition of movement of inflammatory cells (including neutrophils and monocyte-macrophages) into a site of inflammation. They also inhibit lysosomal enzyme release. They greatly inhibit prostaglandin E2 production by cells and this may be the dominant mechanism for their anti-inflammatory effects. It is now felt that they inhibit the generation of prostaglandins by producing proteins called lipocortins. They exert their effect on the prostaglandin cascade above the level of where NSAIDs affect it (Fig 13). There is also evidence for anti-IL-1 and anti TNF effects. There is also evidence for decreased expression for collagenase and stromelysin by inflamed synovial cells. Low doses of corticosteroids have also been associated with inhibition of plasminogen activator.

Clinical use of corticosteroids in the horse - The three most commonly used corticosteroids are 1) betamethasone (Betavet Soluspan or Celestone, Schering), 2) triamcinolone acetonide (Vetalog, Squibb), 3) 6_-methylprednisolone acetate (Depo-Medrol, Upjohn). All drugs are administered intra-articularly. Veterinary preferences vary as to which drug is used. The length of duration of action varies between the drugs but so might the side effects. The clinical use as well as scientific research will now be detailed.

Depo-Medrol (methylprednisolone acetate) - This drug has been used for the longest period of time and therefore has received the most attention with regard to research. It is also the longest acting of the commonly used corticosteroids.
The drug can be detected in the joint 30 days after administration. It is injected at a dose rate of 80 to 100 mg for a carpal joint, for instance. Some veterinarians use a lower dose rate to avoid side effects but recent in vitro work suggests that a dose rate of 80 mg may be necessary for complete effectiveness.

A number of studies have evaluated the effects of methylprednisolone acetate injected into normal joints. In the first study reported in 1977, there were not any toxic effects demonstrated. A second study involved injecting both upper and lower joints of the carpus (knee) with 120 mg per joint on eight occasions one week apart. The opposite joints were used as untreated controls. Although there were no obvious gross differences in the cartilage, histologically there were decreased cartilage cells (chondrocytes) and decreased rates of proteoglycan and collagen synthesis. This did indicate used as untreated controls. Although there were no obvious gross differences in the cartilage, histologically there were decreased amounts of proteoglycans (glycosaminoglycans) in the cartilage but there was no histologic damage. Horses remained clinically normal during the study and significant radiographic changes were not observed.

In a third study at CSU we injected 100 mg of Depo-Medrol three times at twice weekly intervals. We showed some deleterious effects of Depo-Medrol, but the dosage regimen was far above what would be used in clinical practice. decreased cartilage cells (chondrocytes) and decreased rates of proteoglycan and collagen synthesis. This did indicate that a dose rate of 80 mg may be necessary for complete effectiveness.

We are currently investigating the effect of Depo-Medrol in a less severe osteochondral fragment model (arthroscopic) that we have developed at CSU and the results of this study are pending.

Betamethasone (Celestone) - The author first questioned some of the dogma regarding the deleterious effects of corticosteroids when first operating racing Quarter Horses with arthroscopic surgery for the removal of osteochondral chip fractures. During these surgeries and after discussion with the referring veterinarians, it was found that the amount of secondary articular cartilage damage in no way correlated with the number of times the joint had been injected. In fact, some joints had been injected 20 times or more yet had no secondary articular cartilage damage. Because of this, we then went ahead and developed a study using arthroscopic surgery to create chip fragments and exercising the horses on a high speed treadmill. One intercarpal joint of each horse was injected with betamethasone 14 days after surgery and the procedure was repeated at 35 days. We also investigated the effect of exercise (it had been proposed that if one did use corticosteroids, then no exercise should be done because the cartilage was vulnerable to injury). Six of the horses were maintained in box stalls throughout the study as nonexercised controls and six were exercised five days per week on a high speed treadmill with a regimen of two minutes trot, two minutes gallop, two minutes trot. Three weeks after the second injection, horses were clinically examined for lameness and synovial effusion and radiographs were taken. The results of examination of the articular cartilage grossly and histologically showed that there were no consistent detrimental effects of betamethasone with or without exercise. Histochemical staining for the GAGs showed a decrease in the steroid-treated limbs of rested horses, although the decrease was not significant. What was particularly interesting was that in the exercised horses there were similar levels of GAGs in treated versus control joints. Biochemical assays showed no significant difference in water content or uronic acid concentration (a measure of GAG content) in the treated versus control joints. This data demonstrated that exercise had no deleterious effects on joints injected with corticosteroids and in fact may be somewhat protective (presumably associated with increased synthesis due to exercise).

The research work supported our clinical observations that betamethasone did not have significant deleterious effects in the cartilage of horses. We certainly do not advocate treating carpal chip fractures with corticosteroids but at least in the short term, it appears that it does not have significant deleterious effects. Betavet Soluspan, the veterinary product of betamethasone, has become unavailable in the last year. We now use the human preparation, celestone.

Triamcinolone acetonide (Vetalog) - There has been considerable opinion variation on how potent triamcinolone is and also how long acting it is. It is generally agreed that it is a potent corticosteroid but that the length of action (or at least detection) is shorter than previously thought. Because of its common usage and the anecdotal opinions of some veterinarians that it was the best corticosteroid to use, we tested this drug using the arthroscopic chip-treadmill model. We tested not only the deleterious effects of triamcinolone when injected into a joint with a carpal chip fragment and exercise, but also any effects that remotely-injected (into the other joint) triamcinolone might have. We had three groups of horses—one that was treated with control fluid in both midcarpal joints, six that were treated with 12 mg triamcinolone acetonide intra-articularly in the midcarpal joint without an osteochondral fragment, and six horses that were treated with 12 mg triamcinolone acetonide in the joint that contained the fragment. Triamcinolone and placebo treatments were repeated at days 14 and 28 and treadmill exercise proceeded at five days per week beginning on day 15 and ending on day 72. Horses that were treated intra-articularly with triamcinolone in a joint containing a fragment (Group 3) were less lame than horses in Groups 1 and 2. Synovial membrane from Groups 2 and 3 joints (treated with triamcinolone) had less inflammatory cell infiltration, intimal hyperplasia and subintimal fibrosis indices of synovial membrane inflammation. Analysis of articular cartilage with a standardized scoring system showed that the cartilage...
was significantly better in Groups 2 and 3, irrespective of which joint received triamcinolone. In addition, horses treated with TA in either joint had lower protein and higher hyaluronic acid concentrations in the joint fluid. Staining for glycosaminoglycan was greatest in Group 3. The results of this study showed positive direct and indirect effects of intra-articular corticosteroid administration. There were favorable effects of triamcinolone on various parameters and support for a chondroprotective effect in a controlled model of osteoarthritis. This is in marked contrast to the detrimental effects of corticosteroids on articular cartilage seen in other models with other drugs.

Summary - Based on our research, it seems that we can use both betamethasone and triamcinolone without substantial detrimental effects and in the case of triamcinolone, some chondroprotective effects on articular cartilage. There are obvious differences between the drugs used and the dosages and generalizations are difficult. However, we seem to be coming up with drugs that can offer considerable benefit without deleterious side effects. We are particularly interested in seeing the results of Depo-Medrol in the same model that we have tested betamethasone and triamcinolone.

Clinical use - It is unfortunate that the lay public has been told that corticosteroids purely inhibit pain and therefore permit horses to continue to run and degenerate their joints, because this is not the case. It is also a particular concern that it has been implied by some that corticosteroids can lead horses to "break down". It is also important, however, that we continue to try to find better treatments of the arthritic conditions. The ideal treatment for intra-articular fractures of the joint is definitely arthroscopic surgery.

In the meantime, we know that corticosteroids have specific activity against a number of deleterious products produced by synovitis and previously discussed:

1. Collagenase production
2. Proteoglycanase (stromelysin) production
3. Prostaglandin production
4. Synovial membrane-induced interleukin-1 production

At the moment we are investigating what is the minimally effective dose for each corticosteroid in the hope that we can use less drug and further minimize the chance of side effects. If our current research demonstrates that Depo-Medrol does have deleterious effects compared to the other corticosteroids, then we would make the recommendation that the latter drug should not be used. The development of specific equine interleukin-1 through molecular biology (that has happened here recently) could not only lead to more specific testing of corticosteroid dose rate and effectiveness but also potentially lead to specific inhibitors that block the mediators at the time of initial formation.

VILLONODULAR SYNOVITIS

This condition was initially designated as villonodular synovitis and later described as chronic proliferative synovitis. It is specific for the fetlock joint and involves a proliferative response from the synovial fold in the upper front part of the fetlock joint. It can probably be considered as an advanced localized form of chronic synovitis. Fibrous connective tissue develops presumably associated with repeated trauma. The condition is most commonly seen in racehorses but is not specific to them. Conussion, as well as severe hyperextension in the joint are presumed to be the main factors causing trauma to the involved area. With old large lesions, erosion of the bone at the distal end of the cannon bone under the villonodular mass is seen and is presumably due to local pressure of the mass on the bone, although invasion of the bone by the lesion through vascular channels has been considered a factor in a similar condition in man.

The horse shows clinical signs similar to synovitis and capsulitis but has been unresponsive to intra-articular therapy. Radiographs will show a change if the disease process is advanced. In many there will be no plain radiographic change but the mass can be demonstrated with contrast radiography. The simplest method of presurgical confirmation is by the use of ultrasound. With the advent of arthroscopy, cases are commonly recognized at an earlier stage and commonly seen at arthroscopic examination when operating a chip fragment in the front of the fetlock joint. Some people feel that corticosteroids and rest may be effective at treating the problem. However, because of the fibrous nature of the lesion the author feels that arthroscopic surgery provides the quickest and most predictable means of solving the problem.
OTHER FORMS OF PROLIFERATIVE SYNOVITIS

In other instances, the horses present with a joint that has persistent effusion and lameness but no x-ray changes. When the condition is unresponsive to therapy, diagnostic arthroscopy is recommended and cases of greatly thickened synovial membrane and the synovial membrane villi (projections into the joint) are greatly thickened. These conditions again are best treated arthroscopically with removal of the thickened persistent synovial membrane with a motorized arthrobur. After surgical removal of the synovial membrane, a normal one will regenerate.

SPRAINS AND DISLOCATIONS

A sprain may be defined as the stretching or tearing of a supporting ligament of a joint by forced movement beyond its normal range. In its simplest form, there is minimal disruption of fibers and minimal swelling and dysfunction (similar to a mild sprain that we might do to an ankle). On the other hand, severe sprains may cause total rupture of ligaments with marked swelling, hemorrhage and joint instability leading to dislocation. The cause of such problems is having an abnormal force applied to the ligament which becomes tense and then gives away at one of its attachments or at some point in the substance of the ligament. If the attachment pulls loose with a fragment of bone, it is called a sprain fracture or an avulsion fracture.

A mild sprain is one in which a few fibers of the ligament have been torn with some hemorrhage into the ligament but there is no loss of strength. This type of problem will present clinically as an inflammation in the joint capsule (capsulitis) or if an intra-articular ligament is involved, there will be fluid filling in the joint. Rest and a support bandage are the appropriate treatment if it is not intra-articular.

A moderate sprain is one in which a portion of the ligament is torn and some degree of functional loss is obtained. The amount of damage could vary from a tear that can be visualized of a relatively small portion of the ligament or almost complete disruption with retraction of the torn ends. It is possible to heal these injuries with fibrosis. If there is instability, a cast is indicated. Surgery is not necessary unless there is complete separation of the ligament (severe sprain). In the past, we have found it difficult to diagnose some problems. However more recently we have observed partial tears in cruciate ligaments during diagnostic arthroscopy of a problem located in the femorotibial joint. In such cases, debridement has been done and successful results obtained.

A severe sprain is one in which a portion of the ligament is torn and some degree of functional loss is obtained. The usual result is partial or complete dislocation of the joint. Most dislocations involve complete loss of integrity to one or more joint ligaments, as well as damage to other joint structures such as fibrous joint capsule and surrounding tendons. In some instances, dislocations can be treated with restoration of ligamentous integrity and preserving the normal joint. This is usually done by a combination of surgery and cast formation. In other joints, we know that severe osteoarthritis will be the result of the dislocation and we go immediately ahead and treat with arthrodesis (surgical fusion). The best example is the pastern joint. It is common in working Quarter Horses particularly (but other breeds as well) to dislocate their pastern by rupture of the joint capsule or fracturing off the back part of the short pastern bone. These cases can be treated effectively with surgical fusion and over 80% come back to athletic soundness.

TEARING OF INTRA-ARTICULAR LIGAMENTS

With the development of our arthroscopic surgery techniques, we now recognize tearing of intra-articular ligaments in the absence of obvious destabilization. A good example is tearing of the medial palmar intercarpal ligament of the carpus that we initially reported from CSU. The typical situation is a racehorse with a history of carpal problems. Arthroscopic examination reveals various degrees of tearing of the ligament in the back of the joint. Our follow-up study has shown that if 30% or less of the ligament is torn, the prognosis is good. However if 50% or more is torn, the prognosis is much more guarded.

Partial tearing of the cranial cruciate ligament has been mentioned previously and is another entity diagnosed on arthroscopic examination of femorotibial joints. Whereas it was previously believed that all tears of the cruciate ligament were hopeless, "discovery" of this condition means that matters change considerably. A number of such conditions including this and meniscal tears (described below) have only been recognized in the horse with the use of diagnostic arthroscopy. Now that veterinarians are aware of it, cases of stifle lameness with no x-ray changes are blocked to ensure that the femorotibial joint is the site of the problem and if so, are referred for arthroscopy.
MENISCAL TEARS

The menisci are two semicircular pieces of fibrocartilage in the stifle joint. They are very important to the femorotibial joints in terms of stability and function. They were the first condition treated arthroscopically in humans. Severe tears have been diagnosed with other clinical means in the horse but no such cases have ever been treated successfully. With the advent of diagnostic arthroscopy, we now recognize a syndrome on meniscal tearing that can be treated. If the cruciate ligaments are intact, only a small amount of the meniscus is visible arthroscopically. However, it is fortunate that this portion is close to the center of the joint and can be reached. Such injuries are treated with surgical resection of the torn portion.

INTRA-ARTICULAR FRACTURES AMENABLE TO TREATMENT AND IN WHICH THE HORSE CAN BE RETURNED TO ATHLETIC ACTIVITY

Intra-articular Chip Fractures

These are the most common reason for arthroscopic surgery on adult horses (the other common use for arthroscopic surgery is to treat osteochondritis dissecans, which is primarily in the young horse and is discussed later). Chip fractures occur most commonly in the carpus (commonly called knee) and fetlock joints. Cases also occur in the pastern, coffin and hock joints but are much less common.

Why Chip Fractures Cause Problems and Need Treatment

Chip fractures involve the articular surface, which means that they pass through the articular cartilage and disrupt the smooth surface. Figure 14 demonstrates typical examples of carpal chip fractures. The physical disruption of the articular surface provides a physical defect in the joint. The fracture line also releases bone debris that causes synovial inflammation (synovitis). The chip fragments are usually still attached to synovial membrane and their movement within the joint causes direct tugging on the synovial membrane, which has been demonstrated as very painful. The horse therefore has pain directly from the site of the fracture (both synovial membrane tugging and nerve endings in the fractured bone), as well as pain due to inflammation of the synovial membrane. The presence of a physically disrupted surface causes direct damage to the opposing articular surface as well as release of inflammatory mediators, which provides two different pathways that both can contribute to the development of permanent osteoarthritis. Some chip fractures are treated with intra-articular medication to take away the inflammation and pain but this kind of treatment is a compromise because the chip fracture still exists. Arthroscopic removal of the fracture is the preferred technique.

Clinical Signs, Diagnosis and Treatment of Chip Fractures

The diagnostic features, treatment and the results of treatment will be discussed for the commonly encountered chip fractures.

Figure 14. Diagram of two types of carpal chip fractures. Click to enlarge.
Carpal chip fractures - These are the most commonly encountered. The fragments chip off the front of the carpal bones and there are seven different sites where these chip fragments can occur. The signs the horse usually shows is some degree of fluid filling in the knees and varying degrees of lameness. If the case is a simple fresh chip fragment, the lameness is not very obvious and the main feature is the horse traveling with that leg (or both legs when both knees have chips) more widely apart. The diagnosis can be confirmed with x-rays. As mentioned previously, arthroscopic surgery is recommended to both prevent the progression of osteoarthritis as well as treat the immediate clinical lameness and pain for the horse. The surgical techniques enable quick effective surgical treatment. The horse is left with single sutures in two holes in the front of the knee. Hand walking can be commenced two weeks after surgery. Swimming as well as water treadmill exercise are used in the postoperative period by many trainers and owners. Depending on the amount of cartilage damage and the size of the fractures, training may be commenced any time from six weeks to six months after surgery. Based on follow-up studies with carpal chip fragments, 75% come back and race successfully at the same or better level than before. Not all the 25% in the unsuccessful group fail because of carpal problems. Other problems happen to the horses that contribute to this figure. The success rate does vary depending on the amount of articular cartilage damage in the joint. However, it was a pleasant surprise to find that we can tolerate up to 30% articular cartilage loss off a bone that has fractured without lowering the success rate. Once we get to 50% cartilage loss or a significant amount of subchondral bone loss, then the success rate comes down to 50%. In addition, with grade 4 lesions (significant loss of subchondral bone), we probably have some degree of microinstability and this contributes to the horses chipping again when the return to racing.

Proximodorsal P1 chip fractures - These chip fractures occur at the front of the fetlock (they need to be distinguished from fragments in the back of the joint and in particular sesamoid bone chip fractures). They are the most common chip fragment of the fetlock seen in racehorses. They occur in both Thoroughbred and Quarter Horse racehorses but more commonly in Thoroughbreds. However, when they occur in Quarter Horses they tend to be more severe with a larger component of bone loss. They present clinically with filling in the joint and various degrees of lameness. When the fetlock is flexed and the horse jogged off, then the lameness is obviously increased.

The treatment for this condition is arthroscopic surgery and our follow-up data shows that the surgery should be done as soon as possible after the fragment has occurred. Because of the complications that we’ve seen with the old surgical technique for removal (arthrotomy), both veterinarians and trainers have been reluctant to operate these cases. In many instances, training is continued even though a chip fragment is known to be present and this does cause secondary osteoarthritic change. The surgery is straightforward involving an insertion of the arthroscope and instrument through small holes in the front of the joint. Fragments are elevated and removed. A recent study showed that the success rate with these cases is over 80% when the chip is fresh and a little under 70% when the chip has secondary changes on the articular cartilage of the distal cannon bone along with it.

Plantar first phalanx chip fragments - It is somewhat controversial whether these fragments are a fracture or due to developmental orthopedic disease. However, they are seen causing lameness problems in racehorses. They are extremely common in the Standardbred racehorse but are seen regularly in other horses. They are also particularly common in the Warmblood breeds.

The clinical signs are a little different than most other chip fragments in that they generally don't show up until advanced levels of training or exercise. On the other hand it has been shown, at least in the Standardbred, that the fragments are present as yearlings. It is presumed that the fragment becomes a problem because it lodges between the sesamoid bone and the first phalanx and with increased flexion associated with increased exercise, a mechanical problem results that in turn causes pain. The problem is dealt with using arthroscopic surgery but it is a rather difficult technique to learn. There is very little room to move between the sesamoid and first phalanx and the fragment needs to be found and its attachment severed within the joint capsule. Horses receive two to four months out of training after surgery and the results are good.
Sesamoid fractures - Chip fractures of the sesamoid bone can occur on the apex, abaxial surface or base of the sesamoid and more severe fractures can also occur (Fig 15). They are usually seen in racehorses and are presumably associated with trauma. They all occur in the area of attachment of the suspensory ligament. Horses will present with lameness and synovial effusion. There is commonly thickening over the branch of the suspensory that attaches to the sesamoid fracture. The diagnosis is confirmed by radiography.

These cases can now be operated arthroscopically and it is a subjective feeling that it improves the prognosis compared to the previously used open surgery (arthrotomy) technique. The fragments are carefully dissected out of the suspensory ligament and joint capsule attachments and removed. The time of convalescence may be up to six months because of the involvement of suspensory attachments and the need for healing. In the simple fracture of the carpus, we do not have to worry about reattachment of any vital soft tissue structures but it is a different situation with sesamoid fractures.

Carpal slab fractures - These occur most commonly in racehorses but are seen in barrel racing horses and occasionally in other horses. A slab fracture is a fracture that extends from one articular surface to the opposite articular surface. They are most commonly encountered in the carpus (knee) but are also seen in the lower bones of the tarsus (hock). The horses show more lameness than with chip fractures and the diagnosis is confirmed with radiographs. Small slab fractures can be removed but most are treated with lag screw fixation. The principle of lag screw fixation is to drill a hole larger than the diameter of the threads in the slab and then have a threaded hole in the main body of the bone. Once the screw is tightened, the fracture is compressed and this facilitates healing. The prognosis for this injury often depends on the amount of defective bone at the articular surface. This often takes the form of a wedge of bone. Approximately 50% of horses may compete athletically again after such a fracture. Slab fractures can also cause collapse of the joint and are discussed under severe injuries later.

OSTEOARTHRITIS (DEGENERATIVE JOINT DISEASE)

Both osteoarthritis (OA) and degenerative joint disease have been used synonymously. Convention dictates that osteoarthritis is the preferred term. It refers to the stage of progressive and permanent loss of articular cartilage. Much of the treatments discussed previously and further on in this course are aimed at minimizing or preventing osteoarthritis. Conditions that may lead to osteoarthritis if they are too severe or treated inadequately include synovitis and capsulitis, sprain and joint luxations, intra-articular chip fractures, more severe intra-articular fractures, osteochondritis dissecans, subchondral bone cysts, and septic arthritis. Possible pathways for joint degeneration were previously illustrated in Figures 7A and 7B. The signs of OA are progressive dysfunction in the joint clinically. There is swelling and lameness and progressive stiffness develops in the soft tissue. In advanced stages, there is loss of joint space on the radiographs and formation of bone spurs (osteophytes) as well as mineralization within the joint capsule (enthesophytes). The extent of cartilage damage can be confirmed arthroscopically.
Treatment of Osteoarthritis
There are three principles of treatment of osteoarthritis: 1) prevention and/or treatment of the primary cause (e.g. synovitis and capsulitis, intra-articular chip fracture), 2) treatment of any active synovitis in the joint to minimize progressive deterioration of the articular cartilage due to inflammatory mediators, 3) treatment of articular cartilage damage. The treatments for the second group have been previously discussed. In the third group, we are very inadequate in our ability to treat articular cartilage damage. That fact is evidenced by the number of people who have hips as well as knees replaced. It is because the articular cartilage has worn down to bare bone and this causes extreme pain and dysfunction. The principles of treatment of equine osteoarthritis is similar to those of humans--treat the pain and dysfunction palliatively as long as possible. The definitive treatments in man are joint replacement or joint fusion. In the horse, the only definitive treatment available is joint fusion (arthrodesis).

What Do We Know About Articular Cartilage Healing and What Have We Done to Try to Achieve It?
As mentioned before, articular cartilage does not replace itself with the normal tissue--this has been recognized for a long time. We have spent considerable time and effort in researching this area. New treatments come along and we have investigated them. Some of the principles of articular cartilage healing that we have developed based on this research are:

1. Partial thickness defects do not heal. However, if the cartilage remaining is attached to the bone it should be left alone.
2. Full thickness defects heal with fibrous tissue and fibrocartilage. Past convention has been to aggressively curet or debride every cartilage defect so we could get down to bleeding bone and "healing". We know this is no longer true and therefore are conservative about debridement.
3. All separated fragments of cartilage and/or bone need to be removed because they continue to cause irritation to the joint as well as pain to the patient. Debridement removes all loose tissue and then we have copious flushing of the joint to remove any more fragments.
4. The use of subchondral bone drilling does not seem to help satisfactorily. We have recognized that we need to try to maintain the subchondral bone plate because this acts as a foundation for overall articular function. We are currently investigating a technique with microfractures (small pick holes) in the bone to retain subchondral bone but get blood supply into the defect to help healing.
5. We have tried periosteal grafts (some early work in rabbits had shown they could produce hyaline cartilage) and these have failed.
6. We have taken hyaline cartilage from the sternebrae and implanted it into defects with absorbable pins. In four months, this tissue looked like normal articular cartilage. However, at 12 months some deterioration takes place between the graft tissue and the bone so that cracks appear and breakdown follows.
7. It is felt at the present time that our hopes lie in a combination of preserving bone support as much as possible, using growth factors to promote the cells to produce cartilage, and also implantation of chondrocytes that are grown and multiplied in the laboratory to help seed these defects.

It is because of our inability to heal this cartilage that it is so important to treat correctly and early all the other disease processes discussed in this course.

SEVERE TRAUMATIC INJURIES TO JOINTS

Introduction
Obviously the line between severe and not severe injuries is very hard to draw. This section discusses injuries that cause joint instability and without effective treatment lead to a need to euthanize the horse. In some instances, when treated early athletic function can be achieved (distal metacarpal and metatarsal condylar fractures, first phalanx sagittal fractures) but in others, surgery is aimed at salvaging the horse to save its life.

Collapsing Slab Fractures
These are a more severe injury than the routine carpal slab fractures previously described. With displacement of a frontal fracture of the third carpal bone, for instance, the radial carpal bone can drop down leading to loss of support of the joint. Either immediately or over a period of time the joint collapses and cannot bear weight. As has been previously discussed, when we have one limb unable to bear weight we have immediate risk of laminitis developing in the opposite
hoof. The treatment for such injuries is stabilization with screws as much as possible. In some severely comminuted (multiple pieces), a plate and screws may be applied to fuse the joint.

**Distal Metacarpal and Metatarsal Condylar Fractures**
These are a regular racing injury of Thoroughbreds. A fracture progresses from the fetlock joint up for variable distances. In most times the fractures are lateral (outside) of the center of the bone and in others they are medial. The lateral ones usually exit and the fracture is well defined. The medial one has hidden lines, does not usually exit and there is always concern in recovery of these cases because of the potential for an undefined fracture line. All these fractures are treated with lag screw fixation. In undisplaced fractures, the prognosis for racing is very good. In displaced fractures, the prognosis is poor but surgery is still indicated to salvage the horse for breeding.

**Fetlock Destabilization (Failure of Suspensory Apparatus)**
The suspensory apparatus of the limb consists of a suspensory ligament, sesamoid bones and distal sesamoidean ligaments (see diagram). If there is a traumatic disruption of any parts of the suspensory apparatus, loss of support from the fetlock results. This injury is the most common catastrophic injury encountered in racehorses. The most common site of failure is in the sesamoid bones whereby both sesamoid bones fracture.

Conservative and surgical treatments have been used for this condition. Much of the success depends on the condition of the horse at the time of surgery. In some instances, the horse has made the fracture open and there is heavy contamination at the site before veterinary attention can be given. In these instances, euthanasia is recommended. If the injury is not open, our preferred treatment is the use of fetlock arthrodesis. It is recognized that the advent of the Kimsey splint and its use by veterinarians to support the limb as soon as it is injured has helped success rate as well.

**First Phalanx Fractures**
The first phalanx can fracture from the fetlock joint to the pastern joint in a frontal or sagittal plane. When single, these fractures typically have a gap, are not displaced and can be treated with lag screw fixation. In these instances, the prognosis is good.

Unfortunately, more severe comminuted fractures occur in the first phalanx and treatment for salvage is problematical.