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Introduction

We call upon the horse to perform for us in many ways, and he does so quite willingly. We ask him to jump and run as hard as he can, and even still to perform gracefully in the show ring. The tragedy that exists is that all too often we ask too much of the horse and a permanently disabling lameness is often the result. Our technology has provided us with drugs to ease his pain, and yet we are now finding that we are probably doing him more harm than good. The use of drugs such as phenylbutazone and corticosteroids is now under the scrutiny of the American public, for it has been discovered that their use may lead to an irreversible condition of degenerative joint disease (DJD). It is probably the most common condition with which a racetrack practitioner must deal. It is not only a medical problem, but is indeed an economic problem for the racing industry.

Anatomy and Physiology

The diarthrodial joint is composed of: (1) a joint capsule; (2) articular cartilage; (3) subchondral bone; (4) ligaments and tendons; (5) nerves, lymphatics, and blood vessels; and (6) synovial fluid.[6]

The joint capsule is made up of two layers. The outer layer consists mainly of dense fibrous tissue. It is designed to resist stretching and thus to protect the delicate synovial membrane which makes up the inner layer of the joint capsule. The synovial membrane is subdivided into an intimal and subintimal layer. The two predominant cell types which are present in the intimal layer are A cells, which are considered to be macrophages with phagocytic properties, and B cells, which are thought to produce hyaluronic acid.[5] The subintimal layer consists of fibrous, alveolar or adipose tissue which is richly supplied with blood vessels (especially veins) and lymphatics. The synovial membrane is well innervated for detecting pain and proprioception, and has good regenerative capacity. This is important with respect to DJD. In many areas the synovial membrane is folded into primary and secondary villi.

The articular cartilage is hyaline in nature and is composed of a mucopolysaccharide matrix, collagen fibers, and chondrocytes.[5] It lacks blood vessels, lymphatics, and nerves. Nutrition of articular cartilage is accomplished by diffusion of nutrients from synovial fluid and from the capillary beds of the subchondral bone below. It is arranged in three layers; (1) a thin calcified basal layer which is adhered to subchondral bone; (2) a thick intermediate layer which contains a great amount of water, accounting for the shock absorbing capabilities of articular cartilage, and (3) a thin tangential layer of collagen fibers.[6] The elasticity of articular cartilage is related to the fact that collagen fibers are parallel to the surface in the more superficial layers and perpendicular to the surface in the deeper layers of the cartilage.[5] Along with elasticity and shock absorbing functions, it provides a smooth gliding surface for joint motion. Of most importance with respect to degenerative joint disease is that cartilage has very poor regenerative characteristics.

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Synovial fluid is a protein-containing dialysate of blood plasma. It is normally transparent and pale yellow. It contains mucin, which is thought to be a hyaluronic acid protein complex. Hyaluronic acid content, degree of polymerization, and viscosity of the synovial fluid varies with range, speed, and kind of motion of the joint. Viscosity is highest during slow rate of motion and will decrease as the rate of motion increases, thus providing less resistance.[6] Synovial fluid is said to have four important properties; (1) constant load bearing; (2) lubrication or wetting of surfaces; (3) good heat conductivity; and (4) elasticity and the ability to become semisolid instantly on impact, which prevents squeezing out from the articular surfaces.[1] Also of importance is the hyaluronic acid-proteoglycan matrix which exists on the surface of articular cartilage and serves as a form of boundary lubrication.

Pathology

There are a number of terms for a condition that is actually a syndrome. Arthritis is probably the most common term, but degenerative joint disease is probably the most correct. Degenerative joint disease is characterized by degeneration of cartilage and hypertrophy of bone.[1] Soft tissues, however, are markedly affected. The synovial membrane becomes greatly thickened due to congestion and edema. Surrounding soft tissues are destined to become devitalized due to loss of blood supply, and eventually fibrosis and calcification of these tissues occurs. Characteristically, the synovial membrane responds by proliferating and invading the interior of the joint. This inflammatory tissue (pannus) grows from the synovium inward along the surface of the articular cartilage to which it is usually tightly adhered.[6] The result is an interference with the normal flow of synovial fluid through the pores of the articular cartilage, which results then in decreased nutrient diffusion. The cartilage becomes thin, weak and inelastic. The invading inflammatory tissues are then converted to tough fibrous tissue, which eventually being adhered to both opposing joint surfaces, greatly inhibits motion of the joint. This fibrous tissue may then become calcified resulting in firm bony union, and obliteration of the joint space. Radiographically, degenerative joint disease is characterized by uneven joint spaces, variable amounts of lipping, and hypertrophic new bone growth around the joint. Sclerosis of new bone may be evident. Then, as the disease progresses in long standing cases, ankylosis occurs.[6] Changes this advanced are irreversible.

The most significant factor in the initiation of degenerative joint disease, especially in the young Thoroughbred, is trauma. The joints that are most often involved are the carpus and the fetlock. There are many predisposing factors to trauma, including conformation, nutrition, and condition; but most importantly, use.[6] Degenerative joint disease in the case of the horse is a wear and tear phenomenon. The average Thoroughbred is probably the most underconditioned athlete in America. After a brief morning workout, the racehorse spends the rest of his day standing in the stall. Not only does he often race unprepared, which often leads to injury, but quite often he races in spite of injury. As mentioned, much of a racetrack veterinary practice is time spent relieving joint pain with drugs such as phenylbutazone and corticosteroids. It would not be far from the truth to say that, in fact, the widespread incidence of degenerative joint disease is due to the use of these drugs.

Often DJD begins with only mild trauma, damage occurring only to the joint capsule or periarticular ligaments. Adams refers to this condition as serous arthritis.[1] Clinical signs include joint swelling, heat, and pain evidenced by lameness. Inflammation of the synovial membrane is very painful. The most important aspect of this condition is that damage to the synovial membrane results in alteration of synovial fluid and changes in intra-articular metabolism. The viscosity of the synovial fluid is lowered due to decreased hyaluronic acid production by the inflammed synovial membrane, as well as increased hyaluronic acid destruction due to leukocytic enzymes. This is probably the most important aspect of DJD. Lubrication quality is reduced, and friction between articular surfaces increases substantially. Continued workouts eventually lead to cartilage damage. In the case of serous arthritis, fibrinogen, which is normally absent from synovial fluid, enters the joint resulting in the ability of the fluid to clot, which is a diagnostic characteristic. The number of leukocytes increases, producing a turbid color. The increased effusion results

from an increase in the osmotic pressure and also from a decrease in drainage from the joint as a result of the impaired synovial membrane.[6] Radiographically, the bony structures of the joint appear normal. However, if the inflammation of the joint persists, erosion of the joint cartilage and osteoarthritis may result. Serous arthritis is a reversible condition if treated properly. All too often it is not. The use of corticosteroids to mask the pain of serous arthritis will, and, often does, increase the possibility of development of degenerative joint disease.

Severe trauma may, of course, directly damage articular cartilage and/or subchondral bone leading to a more destructive and irreversible degenerative disease. Repair of cartilage depends to a great extent on the site of trauma. If it is central, repair is slow and often incomplete, resulting in an irregular joint surface.[6] Loss of elasticity of cartilage is thought to be the first physiological change leading degenerative joint disease. The loss of chondroitin sulfate and collagen is associated with loss of elasticity and can be attributed to interference with nutrition of the cartilage.[5] Enzymes from leukocytes and plasminogen are also responsible for articular damage. As cartilage damage continues, the subchondral bone is eventually exposed. Continued inflammation and wear leads to sclerosis of the bone followed by eburnation. As the central cartilage is lost, changes take place at the periphery in the form of osteophytes or spurs and lipping of joint margins. The chronically inflammed synovial membrane becomes hypertrophic and villi enlarge.

Treatment

There is really no satisfactory treatment for the pathological changes which have occurred in a long standing case of degenerative joint disease. However, proper management of the acute phase of joint injury as in the case of serous arthritis, can prevent further deterioration of a joint. My personal observation is that many trainers and veterinarians are negligent in this area. It is a difficult situation in that decisions in many cases are based on economics rather than what is best for the horse. Often it is more economical to offer temporary relief and to drop the horse in class, hoping to sell, rather than losing time and money with a proper course of therapy.

Therapy is based upon the pathological stages of trauma. Initially, there is devitalization of tissues, hyperemia and hemmorhages, swelling and edema. This stage is followed by clotting and organization of the area, fibrosis and decreased blood supply. Finally, adhesions and calcification of soft tissues occurs, caused by either the metamorphosis of fibroblasts into osteoblasts or the increased pH due to poor circulation and devitalized tissues.[6]

Initially then, therapy is aimed at reducing hemmorhage and edema, and to relieve pain. Cold in combination with compression and rest will achieve vasoconstriction and thus reduce swelling. It also provides some relief of pain. Cold is best applied for 20-40 minute applications, and at least one hour should elapse before it is reapplied. If cold is applied too long, there may be a reflex vasodilation.[1] At this time the joint can be injected with a corticosteroid to relieve inflammation, but absolute rest must follow.[5] It may be necessary to immobilize the joint with a cast for two to six weeks depending on the severity of the injury. Once the acute inflammatory phase is under control, heat, linaments, and exercise should be used to promote motion, the aim being to reduce or prevent adhesions. Heat causes vasodilation and increases the number of phagocytes in the area, as well as increases oxygen supply to the part.[1] In some chronic cases, it may be desireable to blister, fire, or apply radiation therapy. According to Adams, firing and radiation therapy promote an acute inflammation of the joint; when the acute inflammation subsides, the arthritis may heal and a sound joint result.[1] The most important part of therapy is the adequate rest prescribed. Anywhere from one to six months is necessary. Working the horse too soon may cause re-injury or may lead to osteoarthritis and ulceration of joint cartilage. The prognosis is usually favorable if bony changes have not occurred.

Once bony changes have occurred the prognosis becomes guarded. If new bone growth occurs on the articular surfaces, mechanical interference factors result in chronic lameness. The only conservative alternatives are firing, blistering, or radiation therapy. Firing has been acclaimed for its ability to remove osseous growth, however the inflamation it produces rarely causes any demineralization.[1] Soft tissue swelling is usually all that is reduced. The use of

counterirritation is indeed controversial. Mechanisms of action are not well understood. Some say it is barbaric, others swear by it. More often than not, the results of counterirritation are disappointing at best.

Surgery is always a consideration when extensive bony changes have occurred. Fresh chip fractures often heal if they have good blood supply and if there is minimum separation of the articular cartilage.[6] Chip fractures are often involved in the induction of degenerative joint disease, simply by mechanical disruption of joint surfaces with continued work. If small chips are going to be left alone, the horse should have complete rest and the healing process should be monitored radiographically at four to six week intervals. Separation of the fragment with possible disruption of blood supply is a good indication for removal or stabilization of the fracture fragment.[6] Of primary concern is the fact that one cannot see cartilage damage radiographically. Slight bone abnormalities may be indicative of more extensive cartilage damage. As a rule, osteophytes or fracture fragments which are removed will be much larger than the radiographs may indicate. The important thing to remember is that in addition to removal of a spur or fracture fragment, care must be taken to remove all the reactive underlying subchondral bone. This bone is usually found to be soft which is indicative of degenerative changes. If left undisturbed it will proliferate, therefore one should currette until solid, normal bone is reached. If properly done, follow up radiographs will show minimal bony proliferation. Also of surgical consideration is the removal of hypertrophic synovial villi which have extended into the joint. As stated, the synovial membrane is well supplied with many nerves and entrapment between articular surfaces results in pain. In any case, post-surgical aftercare should consist of at least four to eight months of rest to allow for proper healing.

The dissatisfaction with conservative methods of therapy has lead to the widespread use of intra-articular corticosteroids. Intra-articular injection of corticosteroids will allow a horse to use a joint which has extensive pathologic changes. The exact mechanism of anti-inflammatory action is unknown, however, some feel that they act as buffers between irritants and susceptible cells.[1] Somehow it alters damaged cells so they are less effective in producing substances responsible for the initiation of inflammation. Corticoids inhibit fibrocyte proliferation and healing. They are responsible for stabilizing intimal cells, protecting synovial vessels, and improving circulation by reducing edema.[5] Thus, the synovium in an injured joint is returned temporarily to a more normal state, enabling it to produce more synovial fluid which improves lubrication of the synovium. The immediate effects on synovial fluid are (1) increased viscosity (2) increased polymerization of hyaluronic acid (3) reduced synovial volume and (4) a reduction in the leukocyte count.[5] These effects are responsible for the clinical improvement seen as reduced joint stiffness and pain, which enables the horses to use the joint more freely.

There is widespread misuse of corticosteroids at the present. A joint should not be injected with a corticoid without prior radiologic examination. If a fracture is present, the joint should not be injected if any long-term working future is expected for the horse. The ill-advised injection of a corticosteroid during the time of degenerative arthritis or repairable fractures frequently causes a more rapid destruction of the joint with the production of extensive degenerative lesions that cannot be corrected by surgery. This causes many hopeless cases to be presented for surgery when the corticosteroids are no longer effective, whereas if they had been operated on early, a successful result could have been anticipated.[5] Although the immediate effect of corticosteroids are an increased production of hyaluronic acid due to decreased synovial membrane inflammation and stabilization of chondroblasts, the long term effects with continued use are a reduction in hyaluronic acid synthesis and a catabolism of chondroblasts.[4] Damage to chondroblasts results in cartilage thinning and a loss of cartilage elasticity. If a horse is allowed to continue working, extensive joint damage inevitably results. It must be stressed that intrarticular corticosteroids can be beneficial if used only for a short time and if followed by adequate rest, for they merely decrease inflammation while healing occurs. Too often, the corticoids are used to mask symptoms, and the horse is allowed to further damage the joint.[1]

There is a need, then, for a drug with the

positive action of corticosteroids which does not have it deleterious side effects. Rydell first reported on the prolongation of the anti-inflammatory effects of corticosteroids when they were injected along with hyaluronic acid.[7] Current research is investigating the use of intra-articular hyaluronic acid alone.

Hyaluronic acid is a non-sulfated polysaccharide from which synovial fluid derives it characteristic high viscosity.[3] It exists not only in the synovial fluid, but also is incorporated along with proteoglycans in a 1-10 micron thick protective layer which covers the articular surface.[5] Thus it forms a type of elastic cushion, serving to protect articular cartilage cells. Large concentrations of high molecular weight hyaluronic acid are required. In degenerative joint disease there is often a decrease in hyaluronic acid production as well as a breakdown of existing molecules. The protective function of hyaluronic acid is therefore lost. With continued use of the affected joint cartilage erosion results. Current research, however, is revealing that intra-articular injection of hyaluronic acid may serve as an adequate replacement.[2]

There are many sources of hyaluronic acid, but the molecule may vary with respect to size and its ability to form complexes with proteoglycans. Currently, hyaluronic acid from human umbilical cord seems to meet the requirements for replacement most satisfactorally. Hyaluronic acid derived from rooster combs has also served as a good source. That which has been derived from bovine vitreous has been shown to cause adverse reactions.[8]

In a study by Asheim and Lindblad, forty-five race horses with arthritis of non-infectious type (a total of 54 joints) were treated with hyaluronic acid intra-articularly.[2] These joints had previously undergone therapy with other methods (firing, blistering, steroids, etc.) without satisfactory results. In most cases only one 40 mg. injection was all that was required for satisfactory results. Evaluation of the joints was made on the basis of performance, and of the forty-five cases, thirty-eight were free from lameness, and thirty-two of them returned to the racetrack and went on to win races. They were observed over a period of one year. Only those cases with pronounced osteophyte outgrowth required re-injection. There were no side effects observed. In fact there were two cases in which horses with bone fractures extending into the joint showed marked improvement following intra-articular injection of hyaluronic acid. They concluded that hyaluronic acid acts directly on the surfaces within the joint, not only through its viscoelastic properties but also by a normalization of the hyaluronic acid synthesis.[2] The persisting effects of injection are explained by improving the character of the synovial membrane and fluid.

In order to study the effects of intra-articular hyaluronic acid more quantitatively a force plate study was designed by Auer et. al. on horses with both experimentally induced and naturally occurring osteoarthritis.[3] In both groups they demonstrated marked improvement of weight bearing capacity of the damaged joint as soon as two weeks following injection. In fact, three of the ten horses in the naturally occurring osteoarthritic group were racing again within two weeks.

The exact mechanism of functional improvement of a joint following intra-articular injection of hyaluronic acid is not well known. As mentioned, hyaluronic acid is known to exist freely within the synovial fluid and is also incorporated into the surface of articular cartilage in a proteoglycan matrix. Histological examination of an arthritic joint reveals a general loss of both proteoglycan and cartilage substance. Recall, also that there is decreased production of hyaluronic acid in arthritic joints. Aggregation of proteoglycans on the surface of articular cartilage is greatly reduced and these subunits are found floating freely within the joint fluid. It is believed that they are involved in the initiation of an inflammatory response. In vitro studies of arthritic human cartilage have shown that incubation of proteoglycan subunits with hyaluronic acid results in marked aggregation of these substances. It is therefore believed that hyaluronic acid serves to bind proteoglycans, and thus is of primary importance in the construction of the matrix which exists of the articular surface. Intra-articular injection of hyaluronic acid therefore improves the boundary lubrication of the joint surface by reconstructing the proteoglycan matrix, and reduces inflammation by removing proteoglycan subunits from joint fluid.[3]

It appears then that intra-articular injection of hyaluronic acid may be a successful means of dealing with degenerative joint disease. Its use is without the deleterious side defects of multiple corticosteroid injections.

Corticosteroids have an anti-inflammatory effect on diseased and damaged joints, masking clinical signs, rendering the joint pain free and permitting the horse to return to work.[6] The natural healing tendencies of the articular cartilage or joint capsule is therefore eliminated. Hyaluronic acid acts to lubricate synovial tissues and to improve the quality of the proteoglycan matrix of articular cartilage.[3] Natural healing tendencies are encouraged rather than depressed.

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