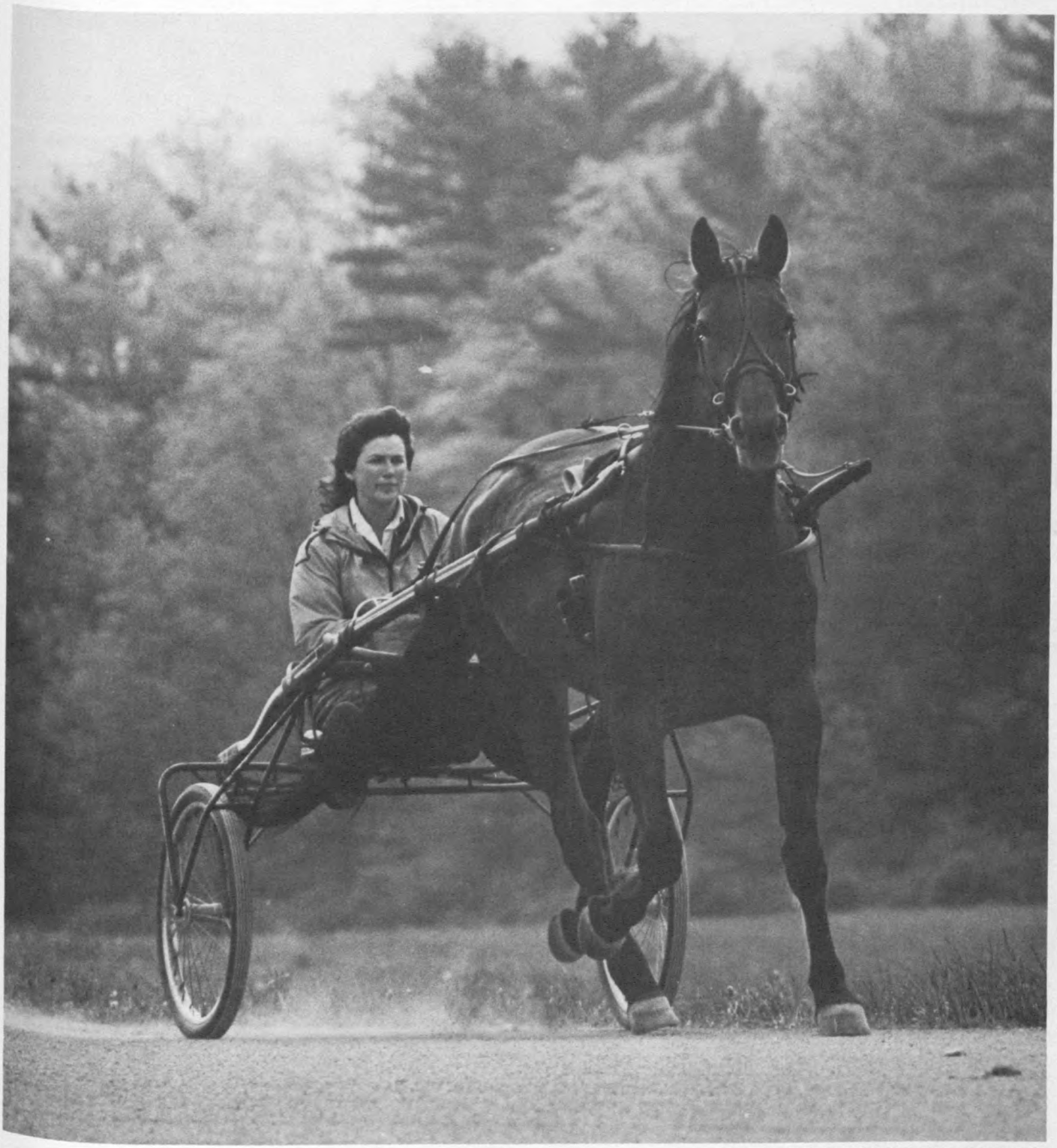
Number 8
September, 1985

EQUINE ROUNDS



SPECIAL LAMENESS EDITION

PREVENTIVE MEDICINE FOR LAMENESS

Part I: The Lameness Equation

By Darryl Millis '87

Recent progress in medicine has established that prevention of disease is both cost and time effective. The American Horse Council estimates millions of dollars are lost due to lameness each year. Included in these losses are not only the cost of treatment, but also losses associated with "lay-off" and the time and money needed to reconditon these animals. Of course intangible losses, such as missing a Grade I stakes race, cannot be assessed a dollar value, but the missed opportunity may have future financial repercussions such as lower breeding fees or reduced value of offspring. Unfortunately, lamenesses are traditionally thought of as unavoidable rather than preventable problems. Lamenesses result from a combination of many factors:

Lameness = Individual Variation + Nutrition + Conditioning + Training Program + Foot Care + Conformation + Use + Breed + Age.

Individual variations, such as bone and tendon quality, are the only factors that cannot be controlled through careful selection or environmental control.

Knowledge of equine nutrition has been applied as a means of preventing developmental deficiencies. For example, calcium and phosphorus imbalances result in a condition known as nutritional secondary hyperparathyroidism or "Bighead disease." Overnutrition may be equally as harmful. For example, excess dietary energy has been incriminated as a contributing factor in epiphysitis and contracted tendons in young horses. An adequate amount of a well-balanced diet is one key to prevention of lameness.

Conditioning, training surfaces, use, and training programs are closely related and may accentuate a horse's inherent weaknesses. Fortunately, our knowledge of these variables has expanded tremendously with the growing interest in Equine Sports Medicine and its applications patterned after human training techniques. Interval training is a new concept which can aid in the prevention of lameness by progressively stressing the horse's musculoskeletal system (as well as the cardiovascular system) without causing breakdown or injury. Although each horse requires an individual training prescription, the principles are the same for all horses. Several weeks of long/slow distance work are initiated to strengthen bones, ligaments and tendons. Speed work is added in small increments for short distances. Finally, the speed work is increased over longer distances until the horse is able to run a full race. This type of training and conditioning program is likely to form a solid foundation and minimize chances of fatigue and failure of the musculoskeletal system.

The degree of conditioning at the time of an event greatly influences the chances of breakdown. For instance, a horse early in a conditioning program is more likely to break down than is a horse farther along in a program.

To carry out such training programs safely, however, requires a proper training surface. Recent interest in this area indicates that perhaps the design of racetracks is not optimal for reducing stress. Theoretically, grass tracks with steeply banked curves would be ideal but turf is harder to maintain and there is not yet a suitable nor economical artificial surface. Similarly, good surfaces are imperative for hunters and jumpers, reining horses and other athletes. Hard surfaces increase stresses on the skeletal system and deep, soft surfaces increase stresses on tissues such as tendons and muscles. A firm,well-drained foundation covered with two to four inches of sandy loam is an excellent surface on which to train and perform.

Probably the most important factor in the lameness equation is the horse's intended use.

Some events place more stress, strain and concussion on the musculoskeletal system than others. Of course, it is the owner's prerogative and the horse's potential which determines use, but it should be recognized that as stress increases so does the risk of injury.

To help minimize stress, strain and concussion, particular attention should be paid to conformation, shoeing, breed and age. Excellent conformation does not guarantee a lameness-free horse, but it does minimize the risk. Of utmost importance are symmetry, straightness of legs with adequate angulation of shoulder and pastern, and well-proportioned bone. Shoeing, which initially weakens the hoof wall, is necessary to prevent the hoof from chipping and cracking and, if done properly, will greatly aid a horse and help minimize lameness. Too frequently, a horse is "overshod." Trailers that extend too far or caulks that are too long cause dramatic changes in the horse's natural way of going and are associated with additional torque and rotational forces in the limb which place extra strain on joints and long bones. It is important to maintain a balance between correction to prevent interference and correction to produce a perfectly straight way of going.

Breed has a minimal influence on the lameness equation, partly because of the wide variety of type and conformation in many breeds. It is unclear whether the genetic pool governing the musculoskeletal system differs among breeds, but it stands to reason that certain lines may have more bone and/or bone strength and it makes sense to breed animals that have good conformation and a low familial history of breakdown.

Age is one of the most important factors. The equine skeletal system continues to mature until 5 or 6 years of age and horses entered in high-stress events at a very young or very old age are at a much higher risk of developing lameness. Probably everyone has thought to himself that horses are raced at too early an age, but there is a dichotomy between maximizing soundness potential and economics. Many prestigious "big money" races are for 2- and 3-year olds. If trainers were to wait until their horses were 5 or 6, the cost of maintaining and training horses might become prohibitive. The early age at which horses are asked to perform will probably never change, but an awareness that young horses cannot tolerate the training and performance levels experienced by mature horses may help prevent some unnecessary lamenesses.

As is evident, the factors contributing to lameness are very complex and numbers cannot be added together to determine when and under what conditions a horse will become lame. The purpose of this article, however, has been to make owners more aware of the risk factors involved. Their consideration may help owners minimize or avoid certain risk factors while they receive the maximum benefits from their horses.

PREVENTIVE MEDICINE FOR LAMENESS

Part II:
Early Clinical and Pre-Clinical Detection of Lameness

With special thanks to
Dr. Francis Kallfelz & Dr. Patricia Tithof

Part I discussed many of the factors that contribute to lameness. While knowledge and proper management of these may help avoid many lamenesses, lameness is not 100% preventable. The next most logical step is the early detection and treatment of lameness since very early treatment results in the least permanent damage and the best prognosis for complete recovery and return to use.

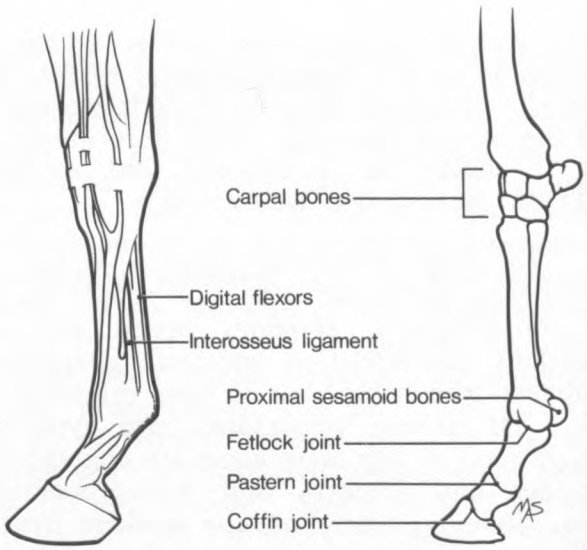
Unfortunately, there are few tests or procedures at present that are readily available and are sensitive and specific enough to predict or detect early lamenesses. Digital palpation,

ultrasound, thermography, scintigraphy and some new experimental procedures are among the most common, but each has its own limitations.

Certainly, the most readily available and least expensive method is digital palpation. It is most effective when performed daily by the same person both before and after training and performance. The best way to become proficient in palpation is to examine many legs with an experienced person, such as a veterinarian, and identify normal as well as abnormal structures. Always make note of any abnormalities, particularly the cardinal signs of inflammation: pain, heat, swelling and redness. Any of these may indicate injury to an area with subsequent attempts to repair the area by the inflammatory process. The exam must be thorough, complete and performed in the same manner each time to avoid missing structures. Subtle changes such as a swollen joint capsule, filling in a tendon sheath, or reluctance to flex a joint should be noted and monitored on a daily basis.

Structures which are especially important to monitor include the foot, fetlock joint, proximal sesamoid bones, pastern joint, coffin joint, deep and superficial digital flexor tendons, interosseous (suspensory) ligament, the carpal (knee) bones and joint capsules in the forelimbs and the stifle and hock joints (in addition to the tendons and ligaments) in the hindlimbs. Conscientious palpation and careful observation, combined with early treatment and rest at the first hint of lameness, could eliminate the majority of chronic lamenesses.

Ultrasound is becoming more frequently used, especially at race tracks. The principle is relatively simple. High velocity sound waves are directed through solid structures which slow the wave velocity to varying degrees, depending on the media encountered. In general, the more dense the media, the greater the velocity of the ultrasound waves. Conversely, damaged bone will slow the velocity of the waves. Experienced personnel are able to detect



changes in velocity along with asymmetry in velocities between legs as an aid in predicting breakdown. Although several groups of researchers have had much success in predicting weakening of bone and subsequent lameness problems, ultrasound's greatest usefulness appears to be in its ability to detect changes in soft tissue, such as tendons, ligaments and uterine tissue. When changes in tissue density and tissue interfaces are encountered by the ultrasound waves, they appear on an electronic screen as varying shades of gray. Very early changes, such as edema in the superficial digital flexor tendon, are able to be detected in this way. Many times, a person skilled in interpreting sonigrams is able to definitely diagnose a problem and begin early treatment to minimize or eliminate permanent damages.

Despite its promise, there are several drawbacks associated with ultrasound. First, there is much variability in the quality of equipment which can affect results tremendously. Also, people skilled in the use of such equipment and interpretation of results are essential if ultrasound is to be used as an aid in predicting breakdown. Ultrasound equipment is fairly expensive which may limit its use to large stables or those with valuable horses who can afford the investment. Finally, although many workers have achieved great

success using ultrasound, other researchers have not been able to reproduce these results and feel that ultrasound is useful in some individuals, but not all horses show characteristic patterns compatible with early breakdown. More research is needed about ultrasound and horsemen should be cautious of claims stating that ultrasound can predict all breakdowns before they occur.

Thermography detects slight changes in heat in the form of infrared radiation. The principle is similar to photos of homes losing heat as indicated by special infrared photography. Areas of inflammation in a horse's leg are detected by color changes in a special photo taken with an infrared camera. The beauty of thermography is that the principle is simple and physiologically sound in that heat is one of the major components of the inflammatory response. The equipment is simply a sensitive means of quantitatively detecting focal areas of increased heat. However, much like ultrasound equipment, thermographic equipment is costly. Furthermore, thermographs are sensitive but fairly nonspecific, meaning that they can indicate that a problem exists, but they usually do not indicate what the problem is and the exact structures involved.

Scintigraphy, or nuclear imaging, also exploits the horse's own inflammatory response by constructing a visual image of an area of increased bone metabolism. A radioactive material, usually technetium-99m labeled bone seeking material, is injected intravenously and allowed to accumulate in the skeleton, particularly in areas where new bone mineral is being laid down. A gamma camera then detects areas of increased radioisotope uptake and a polaroid picture is taken as a permanent record. The levels of radiation encountered are minimal, less than that of radiography, making this a safe procedure for both humans and horses. The technique is very sensitive in that it detects early changes in bone mineral metabolism, usually before changes can be seen on conventional radiographs. In some instances, this technique can easily detect hairline fractures that otherwise could only be detected by conventional radiography with great difficulty. Although this very sensitive technique is a valuable diagnostic aid and can detect very early bone changes, it is available only at teaching hospitals or very large private practices.

Several experimental procedures are being developed as aids in the early detection of lamenesses. One such procedure uses serum hydroxyproline as an indicator of changes in bone organic material. It measures changes in bone metabolism, specifically changes in the organic rather than the mineral portion of bone. Preliminary work indicates that hydroxyproline, a major amino acid of bone collagen, changes with certain skeletal alterations, such as growth, fracture healing, and early degenerative changes. Laboratory testing of blood samples may help identify horses prone to skeletal injury and allow early alteration in training schedules. The use of serum hydroxyproline in the determination of early skeletal changes is a relatively inexpensive procedure, requiring only a blood test. Unfortunately, it is still experimental and it cannot identify specific anatomic areas of early bone changes.

Although many lamenesses may be eliminated or the risk of lameness reduced by knowledge of risk factors, lameness and breakdown cannot be totally eliminated. The next best step is early detection and treatment. By using daily digital palpation and observation to determine early changes, many lamenesses may be averted by rest and therapy. For more refractory problems ultrasound, thermography, and scintigraphy may give much information and guide treatment to help prevent permanent damage.

Darryl Millis, is student co-editor of this issue. In his third year of the DVM program, he is particularly interested in equine lameness and orthopedics.

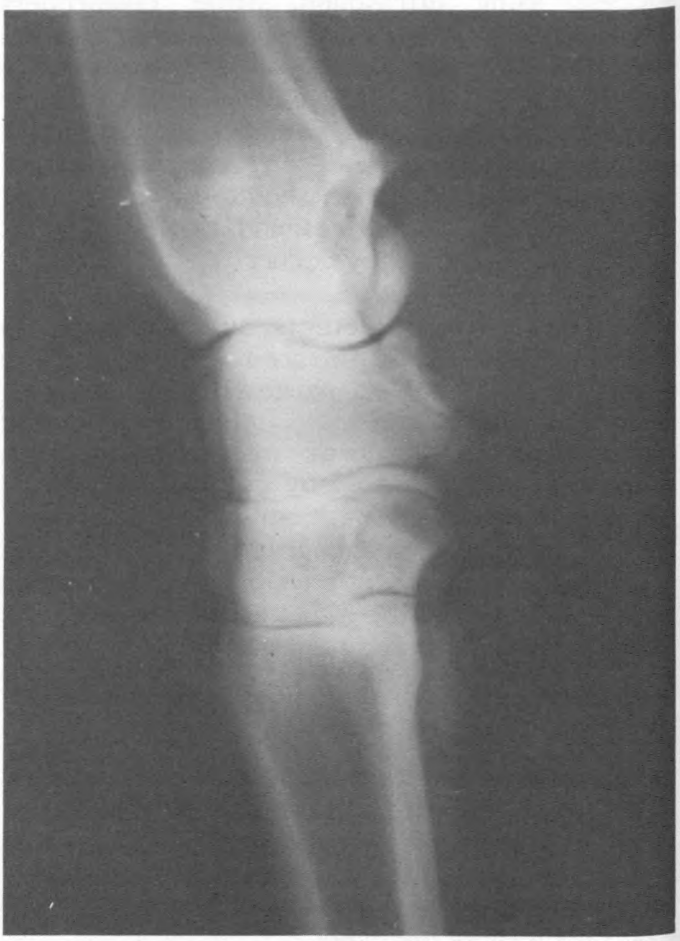
CARPITIS

By Lynne Swanson '86

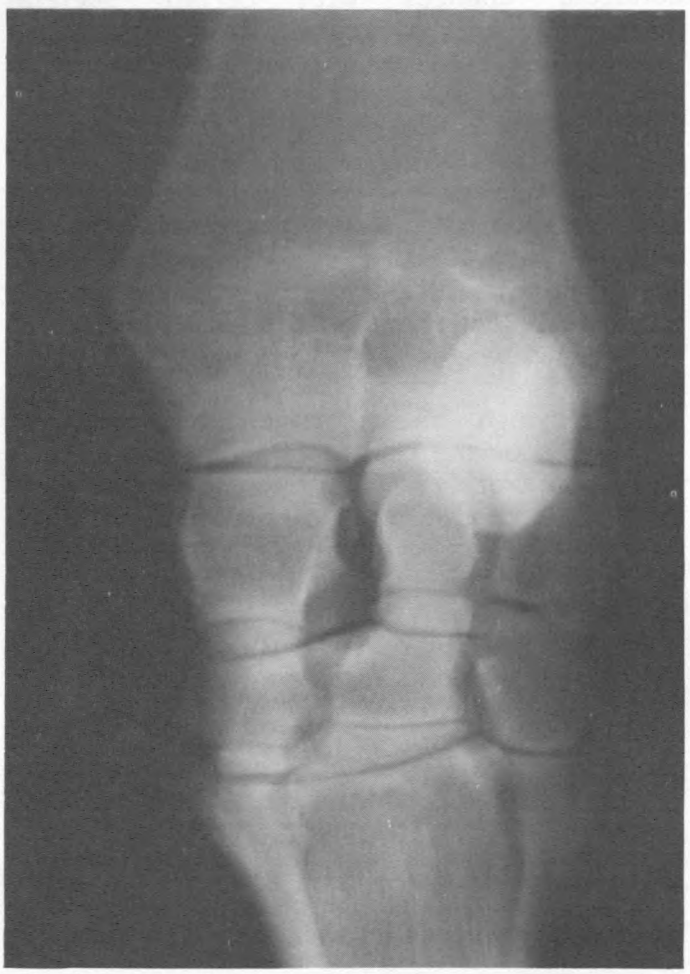
With thanks to Michael Collier, DVM

Carpitis, also known as "popped knee", carpal osteoarthritis and traumatic arthritis of the carpus, refers to inflammation of the carpal joint (commonly called the horse's "knee"). It is a serious condition even when its presenting signs are mild.

Carpitis is most commonly seen secondarily to one or any combination of four factors: forelimb trauma and concussion, stress due to excessive work, poor conformation, and abuse of intraarticular corticosteroids. Overwork of young or poorly conditioned horses predisposes to stress and fatigue with subsequent hyperextension of the carpus and concussion to bony and soft-tissue structures that make up the joint. Poor conformation, such as horses that are back at the knees or have bench-knees, knock-knees or bowed legs, is associated with poor alignment of the seven carpal bones which further predisposes the carpus to injury. Intraarticular corticosteroids, while often indicated in treatment of carpal problems including carpitis, may lead to symptoms of carpitis if overused or used without rest after therapy.



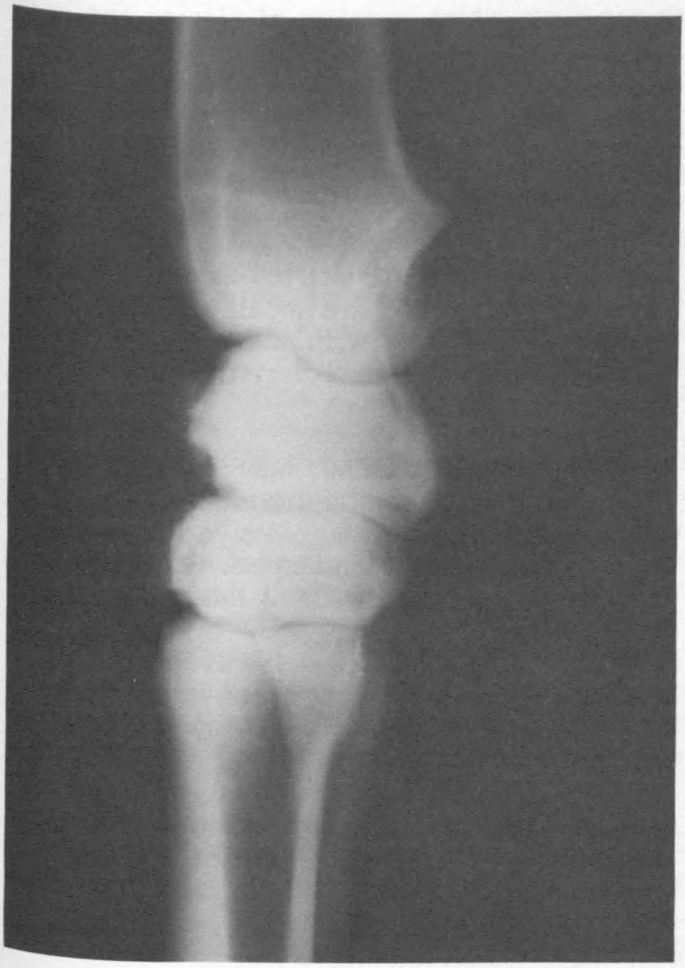
Lateral radiograph of a normal carpus.



Radiograph of a normal carpus

Signs of carpitis include joint swelling that may make the knee appear to be popping out to the side (hence the term "popped knee"), acute or insidious onset of lameness and a reduced ability to flex the carpus, with or without pain and heat in the joint. These signs are secondary to degenerative changes in the joint that may involve fractures, new bone growth in abnormal locations, tendon or ligament swelling or rupture and joint capsule inflammation.

The degree of damage to articular structures may be hard to assess from a clinical point of view. While some of the degenerative changes may be reversed with treatment, there is always the possibility that irreversible damage to the joint has occurred.



Lateral radiograph of a carpus with carpitis. Note soft-tissue changes and evidence of inflammation.

The most important factor in treating a horse with carpitis is rest. Six to ten or more weeks of rest is recommended. Less rest than this may lead to further degeneration of the carpal joint and a recurrence of signs. Adjunct therapy may include bandaging, ice packs, topical Dimethyl sulfoxide (DMSO), intraarticular injections of corticosteroids, hyaluronic acid, or polysulfated glycosaminoglycans in conjunction with phenylbutazone to alleviate pain. The prognosis for recovery following treatment varies with each case. Horses with good conformation that have suffered an acute injury have a better prognosis when compared to horses with poor conformation or a chronic history of carpal problems. Corrective shoeing or surgical repair may be required if fractures are present.

If carpitis is suspected the horse should be rested and a veterinarian consulted. The presence of slab or chip fractures needs to be ruled out. Other conditions such as carpal hematomas (subcutaneous bleeding over the joint) and hygromas (subcutaneous collection of fluid) must also be eliminated as possibilities. Rapid diagnosis and treatment will allow for optimum long-term results.

Lynne Swanson, is student co-editor of this issue. She hopes to practice in a mixed-animal or exclusively equine practice upon graduation.

BONE SPAVIN

By Amy Glaser '87

With thanks to Dr. Richard Pankowski for providing references and many helpful suggestions.

The hock is a complex joint that offers many potential lameness problems. One of the most therapeutically frustrating of these problems encountered by veterinarians and horse owners is bone spavin. Bone spavin can be defined as an osteoarthritis, or degenerative joint disease, of the distal hock joints that may eventually produce a bony fusion between the joints involved. The joints of the hock that are most commonly affected by this degenerative disease are the distal inter-tarsal joint and the tarsal-metatarsal joint. Degenerative changes may also occur in the tarso-crural joint and the proximal intertarsal joint. (See diagram.)

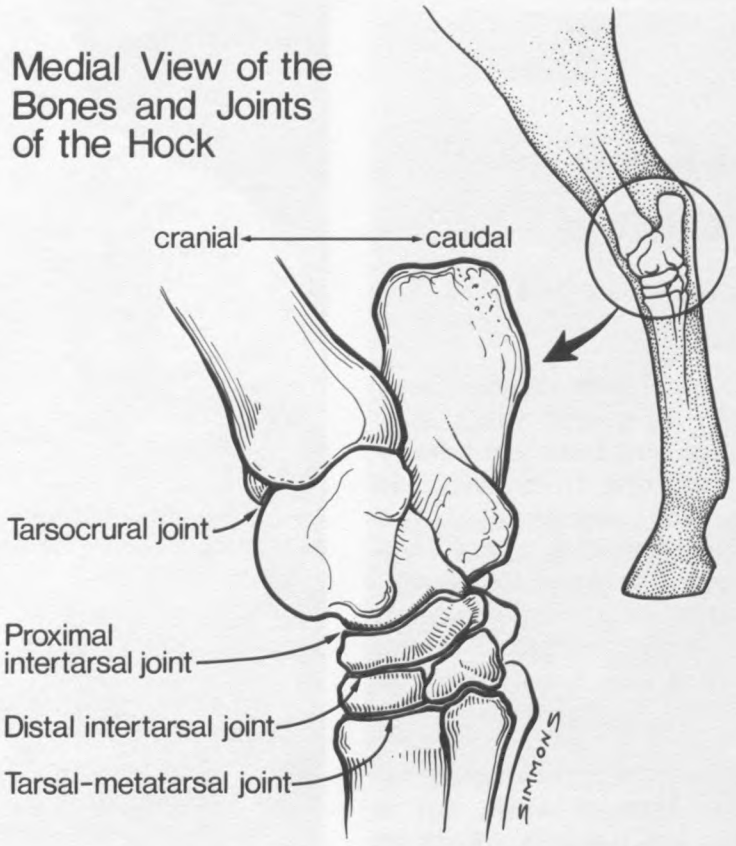
The arthritic changes present in the hock joints of an animal with bone spavin result from exposure to abnormal mechanical stresses for long periods. Horses with poor hock conformation, such as cow- or sickle-hocked animals, are especially prone to lameness originating in this joint. These types of conformations increase the compressive force placed on the inside and cranial (front) portions of the hock joints, respectively. In horses with poor hind limb conformation, the predisposition to develop degenerative joint disease in the hock could be a heritable trait. Other animals at risk are those whose joints are exposed to injury while working, during reining, roping, jumping, and racing. Many of the activities these horses perform create both shearing and compressive forces in the hock as the horse stops, turns, jumps, and runs or trots.

Signs suggestive of this problem result from pain which is greatest when the hock joints are flexed. An animal with an osteoarthritic problem in the hock will exhibit a gait characterized by decreased flexion of the hock in the cranial phase of the stride and a decreased arc height in the flight of the foot. As a result of these changes in the hoof flight, the toe tends to hit the ground first and will begin to wear. In a horse with early or mild degenerative changes, lameness may only be apparent at the beginning of a workout. In an animal with more advanced arthritic changes, exercise will frequently worsen the lameness.

Visually, a bone spavin may be apparent on the inside of the hock as a nonpainful, hard, bony swelling. In some animals, these gross changes are not visible, but more subtle alterations are apparent on radiographs.

Most animals with degenerative joint disease in the hock will react positively to the spavin test, which consists of flexing the hock for 90 seconds and then trotting the horse away briskly. A horse with osteoarthritis will be considerably more lame the first few steps after the hock has been flexed than it was before. Although other problems not related to the hock may cause a positive spavin test, a strongly positive response is suggestive of a hock problem. A definitive diagnosis can be made with injection of a local anesthetic into the affected joints with subsequent loss of lameness, and by characteristic radiographic changes.

Corrective shoeing may help alleviate pain and correct gait deficits early in some cases. However, more aggressive treatment of the problem is usually indicated. Treatment of degenerative osteoarthritis of the hock is aimed at bringing about the bony fusion of the affected joints. Conservatively, this is achieved by administering a non-steroidal anti-inflammatory drug, such as phenylbutazone, to combat pain while continuing to work the animal. The continued exercise increases the rate at which the diseased joints degenerate and speeds the fusion process, while the phenylbutazone allows the animal to remain reasonably comfortable. If the animal remains painful, corticosteroids such as



methylprednisolone can be injected into the involved hock joints. Decreased lameness should be evident two to three days post injection and will last two to three weeks. This may also speed joint fusion in more advanced cases. If conservative therapy does not result in ankylosis of the joints involved, surgical intervention may be initiated. Currently the most popular surgical approach is to use a hand drill to remove 60% or more of the remaining diseased articular surfaces in the affected joints. This is a very invasive technique which should only be used after more conservative therapy has failed to produce the desired joint fusion. Any recovery is slow, taking five or more months before ankylosis is achieved after surgical intervention; and it may never occur without surgery. Once fusion has occurred, the pain and thus the lameness usually disappear and the horse can return to normal function without the aid of pain medication.

The prognosis for horses with degenerative osteoarthritis of the hock joints is guarded. Joint fusion and complete recovery within a reasonable period of time without surgical intervention is rare and even if surgery is performed there is no guarantee that a functionally sound horse will result. Animals with only distal inter-tarsal joint and tarsal-metatarsal joint involvement usually have the best chance for functional recovery because these joints are only minimally involved in flexion and extension of the hock, and their eventual fusion may not compromise normal movement of the leg. Conversely, animals with tarso-crural and proximal inter-tarsal joint involvement have the worst prognosis because these joints are actively involved in flexion and extension of the leg.

Historically, osteoarthritis of the hock, or bone spavin, has been difficult to treat effectively. Perhaps the best way to treat this condition is to prevent its occurrence by breeding horses with sound hind limb conformation and by limiting externally imposed stresses on the hock joints.

Amy Glaser is a third year veterinary student with an interest in equine medicine.

NAVICULAR DISEASE

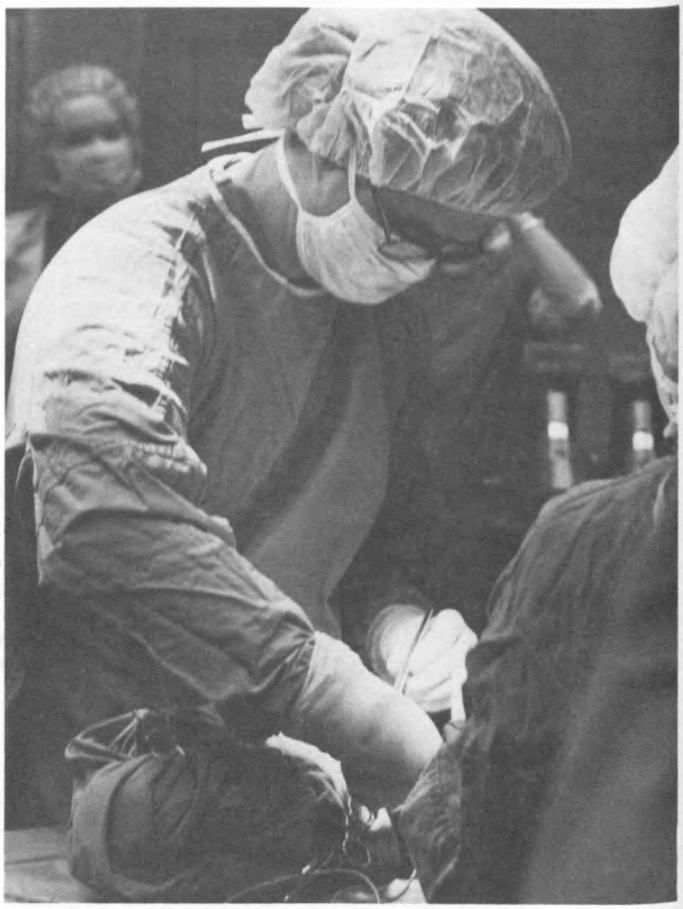
By Caroline Griffitts '88

With thanks to Dr. Lance Bell

Lameness due to navicular disease was first described clinically over 200 years ago. Classically, the lameness has been considered an arthritic condition resulting from navicular bursitis and subsequent degeneration of the flexor cartilage of the navicular bone and damage to the deep flexor tendon. Currently, the lameness is attributed to ischemia (oxygen deprivation) due to arteriosclerosis and thrombosis in the arteries supplying the navicular bone.

Navicular disease can occur in horses of any age performing any type of work, but is most common in hunters and jumpers. Work on hard ground, conformation characterized by an upright pastern or shoulder and feet too small for the size of the horse's body all predispose a horse to navicular disease. The horse develops a chronic progressive lameness, usually in the forelimbs, although it can also affect the hindlimbs. Afflicted horses show a short cranial (forward) phase of the stride, stumble often and tend to land on the toe first. At rest, the horse may point one foot or stand with both front feet extended farther than normal. The horse will go sound after a nerve block of the palmar (posterior) digital nerves. Radiographs of the navicular bone show areas of decreased bone density (appearing black on the film) corresponding to the distal nutrient foramina, or perforations in the bone, where the blood supply enters the bone. Normally these are cone-shaped, but in chronic navicular disease, they are larger and round or "lollipop" shaped.

One theory is that the disease appears to start with partial or complete obstruction of the digital or navicular arteries. This obstruction may be due either to proliferation of tissue in the arterial walls or to thrombosis (blood clot adhered to vessel wall).



This reduces the blood supply, causing ischemia of the bone, which is probably the cause of pain. Collateral circulation and supplementary circulation from the outer covering of the bone (periosteum) develop in response to the ischemia resulting in the enlarged nutrient foramina seen radiographically. If the collateral circulation is adequate, the ischemia will resolve and the lameness will disappear. However, if the secondary blood supply is also affected by thrombosis, the ischemia and its associated lameness will progress. If the obstruction to circulation is extensive, areas of bone may become necrotic (dead) and later the cartilage may be damaged. These necrotic areas are seen as radiolucent areas within the bone.

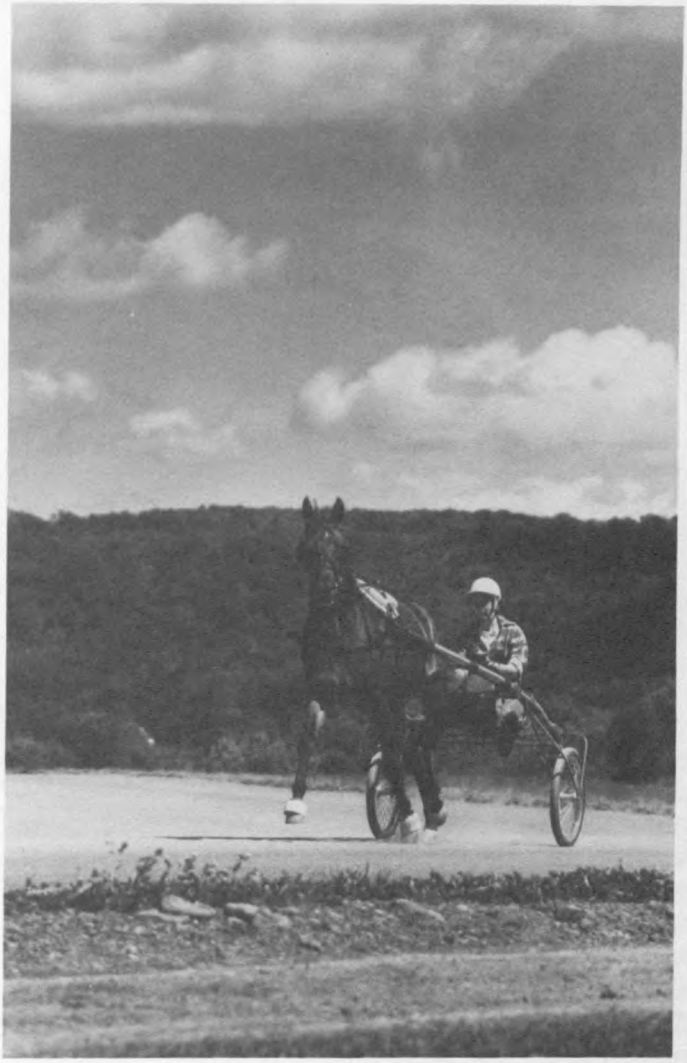
Many treatments have been suggested for navicular disease, but most have been successful only in temporarily eliminating clinical signs or delaying the progress of the disease. Many, such as the injection of cortisone, have been aimed at treating an arthritic condition. Palmar digital neurectomy -- cutting the nerves that supply sensation to

the area -- eliminates the signs of lameness, but does not alter the progress of the disease. Additionally the severed nerve endings may sprout new nerve parts which result in pain some time after surgery. Research at Cornell by Dr. John Cummings and Dr. Susan Fubini is addressing this problem through the use of a nerve toxin. In other research, anticoagulant therapy with warfarin seemed promising initially as a means of slowing the thrombosis. However, the difficulty in determining the proper dose, the continued daily treatments needed, and the possibility of hemorrhage make this drug unattractive to many owners and veterinarians.

At present, an alternative therapy is being tested. Isoxsuprine hydrochloride is a peripheral vasodilator which acts as a smooth muscle relaxant. It has been used in veterinary obstetrics and to treat human ischemic disorders. If it will cause vasodilation and thus increased blood flow in the lower limbs of horses, isoxsuprine would be useful to treat navicular disease by reducing ischemia. Clinical trials in Australia have been encouraging, with 83% of horses diagnosed with navicular disease becoming sound with isoxsuprine therapy. The improvement persisted for variable lengths of time after treatment ended.

Currently, studies are under way at Cornell to evaluate isoxsuprine for treatment of navicular disease. Dr. Lance Bell is studying the efficacy of oral isoxsuprine paste in the treatment of navicular disease. Drs. Nora Matthews, Charles Short and Robin Gleed are evaluating the cardiovascular effects of oral and injected isoxsuprine in horses. The results of these trials may indicate if this drug is promising as an effective treatment for navicular disease.

Caroline Griffitts is a second year veterinary student with an interest in equine medicine and nutrition.



EQUINE ROUNDS

A Newsletter for Horsepeople

New York State College of Veterinary Medicine
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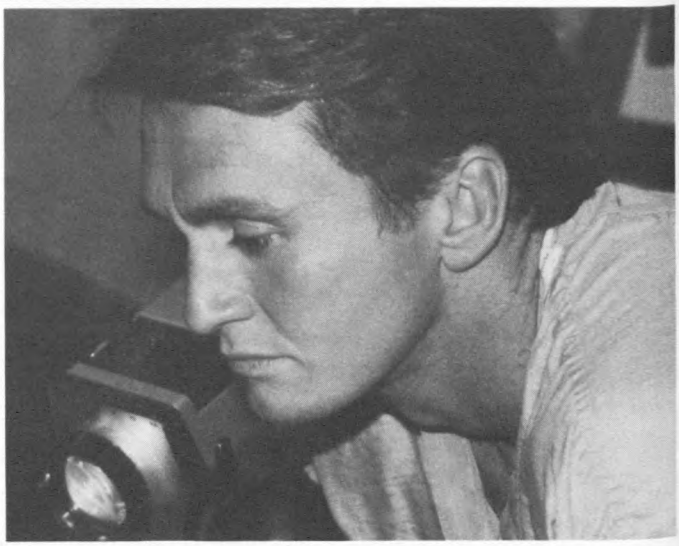

DR. RORY TODHUNTER

By Claire Tusch, '87

THE FACULTY EDITOR of this issue of Equine Rounds is **Dr. Rory Todhunter,** BVSc., MS, an assistant professor of surgery in the Department of Clinical Sciences. A native of Australia, Dr. Todhunter received his veterinary degree from the University of Sydney. After practicing for two and one-half years in Australia, the spirit of adventure captured him and he journeyed to Great Britain where he traveled extensively while working as a relief veterinarian. The myriad of experiences fueled a keen interest in equine surgery which brought him to the United States in 1980 to begin a three year residency in surgery at Michigan State University, culminating in a Master of Science degree in equine surgery. Although he developed expertise in many surgical procedures, he specialized in the delicate techniques of esophageal surgery such as the repair of constrictions and outpouchings of the esophagus.

In 1983, Dr. Todhunter joined the faculty of the New York State College of Veterinary Medicine. He has focused his interest on orthopedic surgery and is especially interested in procedures such as fracture repair, joint arthrodesis, arthroscopy and arthroscopic surgery. He is also leading or participating in several research projects including the evaluation of electrical stimulation in the healing of fractures, the prevention of post-neurectomy neuromas, the evaluation of hyaluronic acid degenerative joint disease and in collaboration with other veterinary schools, the assessment of the prophylactic use of antibiotics in surgery.

In addition to his busy clinical and research schedule, he teaches courses in large animal surgery, and plans to sit for the board examination of the American College of Veterinary Surgeons.

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