

Diagnosis and treatment of a subchondral bone cyst in the medial femoral condyle of a 1 year old Thoroughbred colt.

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INTRODUCTION:

This case report describes a 1 year old Thoroughbred colt diagnosed with a subchondral bone cyst in the medial femoral condyle of the left stifle. The colt presented with an insidious onset hindlimb lameness obvious at the trot (3/5 on AAEP 1-5 scale), with no other localizing clinical signs. The combination of a thorough lameness examination, regional anesthesia, radiography and ultrasonography allowed a diagnosis to be achieved. The cyst was treated successfully with corticosteroids injected via arthroscopy. This case report highlights the diagnostic challenge and therapeutic options available for horses with subchondral bone cysts in the medial femoral condyle. Periarticular subchondral bone cysts were first reported as a clinical entity in the horse in 1968.¹ Subchondral bone cysts in the stifle constitute a major cause of stifle lameness in young Thoroughbreds.² In one study of 86 horses presented for chronic stifle lameness, the most frequent diagnosis was a subchondral bone cyst of the medial femoral condyle; with 38% of horses having such a lesion.³ Subchondral cystic lesions nearly always develop at major weight bearing sites.⁴ The most common location for a subchondral bone cyst is the medial femoral condyle.⁵ Subchondral bone cysts can occur at joint articulations or in the metaphyseal area of growing bone, but articular cysts normally cause the most severe clinical signs.⁶ Grossly and histologically subchondral bone cysts have characteristic features.⁵

Subchondral bone cysts of the medial femoral condyle have been reported to range in size from shallow, less than 10 mm to much greater than 10 mm and can be conical, dome shaped or spherical with a thin radiolucent tract communicating with the articular surfaces.⁷ There appears to be little correlation between the size of the subchondral bone cyst and the degree of lameness.⁸

Grossly, subchondral bone cystic lesions present as a cartilage covered depression on the articular surface, associated with cracks and wrinkling in the cartilage.⁵ On cut section, the cartilage is found to be thicker over the cystic lesion.⁹ This cartilage overlying the cyst is usually degenerated but the remaining surface of the articular cartilage is normal.¹⁰ The articular cartilage at the margins of the subchondral cystic lesion consists of rounded and vacuolated chondrocytes, with the presence of chondromes (a nest of chondrocytes and their collective pericellular matrix).¹¹

The bone margins of the cyst are often sclerotic,⁹ with the bone at the edge of the lesion showing evidence of remodeling.¹² Bone sclerosis surrounding the cyst can range from mild to intense depending on the stage of development.¹⁰ The lining of the cyst is composed of fibrous connective tissue, with active fibroplasia and capillary proliferation present in the area of the cyst adjacent to the bone.¹² Some cyst cavities have been shown to be filled with dense, fibrous or myxomatous tissue, necrotic bone or calcified and mineralized tissue.^{12,13} The lining of these lesions has also been shown to secrete active inflammatory enzymes: interleukin-1 β and interleukin-6 which, along with prostaglandin E2, nitric oxide and neutral metalloproteinases, recruit osteoclasts and have been demonstrated to cause active resorption of bone in vitro. These inflammatory cytokines are thought to contribute to the pain, maintenance, slow healing

and expansion of these lesions.^{14,15} The cyst fluid is typically gelatinous, mucoid to serosanguinous in nature.⁵ Articular cysts are usually unicameral (single chambered), although bicameral and multicameral cysts do occur.¹⁶

Subchondral bone cysts are considered to have a multifactorial etiology which is not completely understood.^{5,17} An attempt has been made to correlate the pathogenesis of bone cysts in humans to the clinical condition found in horses. However, human subchondral bone cysts are usually associated with a primary joint disease,¹⁸ which is uncommon in horses who are typically young (< 3 years of age) and usually have little or no degenerative joint disease.⁶

Debate exists as to whether subchondral bone cysts are a manifestation of osteochondrosis,^{9,19} secondary to trauma^{11,12} or a combination of both.¹⁹

The pathogenesis for either an osteochondral or a traumatic origin can be confusing and an overlap may exist between these two etiologies. For example, the specific locations where subchondral bone cysts arise may be predisposed to by a delay in endochondral ossification at those particular sites, pointing to an osteochondral etiology.^{20,21} These locations, however, may also be subject to trauma due to excessive stress at those particular sites^{19,22} indicating a traumatic etiology.

Osteochondrosis has been cited by many authors as the primary etiology for the development of subchondral bone cysts and has been attributed to a disturbance in endochondral ossification.^{6,9,20,21,23} A disturbance of cellular differentiation in the growing cartilage leads to thickening of the epiphyseal cartilage due to the retention of the hypertrophied zone of cartilage cells with minimal calcification of the cartilage matrix. Endochondral ossification can not proceed normally, and necrosis subsequently

occurs in the thickened retained layers of cartilage. Subchondral cystic lesions have been described by some as resulting from an infolding of abnormal cartilage into the underlying bony spongiosa, with subsequent necrosis of the infolded cartilage.^{4,23,24} An alternative theory suggests osteochondrosis is a result of reduced structural (connective tissue) strength, rather than a failure of endochondral ossification.²⁵ Regardless of the etiology of osteochondrosis, the cartilage matrix remains non mineralized thus, blood vessels do not migrate into the defect to enable repair.^{9,23}

There are two possible outcomes in horses which develop osteochondrosis: either dissecting lesions develop leading to the formation of cartilagenous flaps (osteochondritis dissecans(OCD)); or local areas of degenerative cartilage persist deep within the subchondral bone and lead to the formation of subchondral bone cysts.^{9,23}

It is generally accepted that both OCD and subchondral bone cysts are manifestations of osteochondrosis.⁵ Whether osteochondrosis results in an OCD or a subchondral bone cyst is dependent on location. Subchondral cystic lesions tend to develop in weight bearing areas, whilst OCDs tend to develop in non weight bearing locations.⁴ For advocates of an osteochondral etiology for the development of subchondral bone cysts, the fact that subchondral bone cysts affect predominately a young population, are frequently bilateral, and are often associated with OCD lesions, is proposed as further evidence to support this theory.⁷ Bramlage (1993),⁶ amongst others, support this osteochondral theory but suggests an alternative pathogenesis to the classically reported 'cartilage plug' from failed endochondral ossification.⁶ They suggest that there is a disturbance in endochondral ossification and the cartilage does thicken, but instead of this leading to a non calcified lesion invaginated within the bone, it results in an

unstable cartilage surface and subsequent cracking of the articular cartilage. These fissures then allow synovial fluid to be pumped into the subchondral bone and, with each weight bearing step, this hydraulic pressure increases and causes bone resorption and the creation of a cyst.^{6,26}

Many factors have been suggested for the development of osteochondrosis, including trauma, endocrine abnormalities, nutritional factors and genetics.^{20,21} A familial basis has, for example, been reported in pigs²⁷ and the possibility for a familial tendency has been described in Standardbreds and Swedish Warmbloods,²⁸ although currently available evidence suggests that it is not yet possible to formulate a program of screening for osteochondrosis in horses.²⁹

High planes of nutrition along with rapid growth rates are associated with an increased incidence of osteochondrosis.³⁰ There is evidence that certain mineral imbalances can predispose to osteochondrosis. Low copper, calcium, or selenium levels and high phosphorous, zinc, or molybdenum levels have been shown to have an effect on the incidence of osteochondrosis.^{31,32}

It has been demonstrated that environmental conditions, such as exercise, can affect the development of osteochondrosis.³³ Foals confined to stalls had more lesions in the femoral condyles. In contrast, exercised foals had more lesions involving the lateral trochlear ridge of the femur.³³ Biomechanical forces appear, therefore, to play an important role in the location and development of osteochondral lesions.³³ Pool (1993) also questioned whether lesions, thought to be a result of osteochondrosis, were due to normal mechanical forces acting on abnormal cartilage or abnormal mechanical forces acting on normal articular cartilage.¹⁹

The other major, non osteochondral etiology, for the development of subchondral cystic lesions in the medial femoral condyle is trauma.¹¹

More recently, evidence for a traumatic etiology has been provided by studies where subchondral bone cysts in the medial femoral condyle were created subsequent to surgically induced full thickness lesions in either the cartilage and the subchondral bone,¹¹ or in the cartilage alone.³⁴

It has been proposed that the mechanism of action may involve the extrusion of synovial fluid into the subchondral bone, through an articular cartilage and subchondral bone defect¹¹ or through an articular cartilage defect alone,³⁴ during weight bearing which leads to pressure necrosis and subsequent resorption of bone at the point of maximal weight bearing.¹⁸ In many ways the pathogenesis is similar to the osteochondral theory for cyst development proposed by Bramlage, although the inciting cartilage damage is different. This traumatic pathogenesis may explain cystic enlargement following debridement of subchondral bone cysts.⁷

A traumatic etiology can be further divided into acute direct trauma³⁵ or prolonged minor trauma.³⁶

Support for an acute traumatic origin was proposed by Yovich et al (1989) who reported a case of a subchondral bone cyst secondary to an intra-articular fracture.³⁵ It was proposed that this trauma resulted in an articular cartilage defect which subsequently allowed synovial fluid to be forced through to the subchondral bone.³⁵ It has also been suggested that such direct trauma produces a focal area of subchondral bone ischaemia and necrosis with subsequent revascularization and resorption of the

necrotic bone.³⁷ This weakened subchondral architecture allows synovial fluid to be forced through an articular cartilage defect into the necrotic subchondral lesion during locomotion.³⁷ Where acute trauma has not occurred, researchers have attributed subchondral cystic lesions to be secondary to stress concentration in the subchondral bone in the region where the lesion develops.¹² This repetitive minor trauma is thought to produce trabecular microfractures, fatigue and segmental collapse, as a result of weight bearing on an uneven joint surface.¹² Variations in conformation could result in abnormal stress concentration and subsequent subchondral bone cyst development.³⁸

Traumatic etiologies have been used to explain the finding of subchondral bone cysts in older horses.⁷ However, the timing of predisposing trauma does not always correlate with the onset of signs attributable to subchondral bone cysts.³⁹

Proponents of an osteochondral etiology for subchondral bone cysts suggest that it is possible that older horses presenting with subchondral bone cysts may have developed the cyst at an early age and remained asymptomatic until a sufficient level of work is sustained.⁴⁰ Some authors believe that when cystic lesions are diagnosed as causing lameness in older horses, they should be considered recent exacerbations of an old problem rather than the development of a new condition.⁹

Subchondral bone cysts are of clinical significance due to the resulting lameness which often ensues. The lameness demonstrated in horses with subchondral cystic lesions is caused by pain.⁶ The pathophysiology of pain associated with subchondral bone cysts is currently unknown. It is thought to originate from 2 possible sources: 1) Hydraulic pulsations of synovial fluid through the articular defect into the subchondral bone creating a painful increase in intraosseous pressure in the bone surrounding the cystic

cavity: 2) Synovitis created by debris shed into the joint cavity.⁶ It has been suggested that subchondral lesions do not cause clinical signs until they 'rupture' into the involved joint.⁶

Most horses with subchondral cystic lesions are between one and three years of age and present to a veterinarian because of a unilateral lameness.²³ However, horses have been reported to range in age from a few months to fifteen years.²⁴ A small number of horses with bilateral lesions may be lame bilaterally.²³ All breeds of horses can be affected, although Quarter horses and Arabians appear to be particularly prone to the occurrence of subchondral bone cysts in the medial femoral condyle.¹³ The duration of lameness of horses presented in one study ranged from a few days to 3 years.⁷

The most typical history is of an intermittent lameness, appearing when a young horse begins training or is subjected to an increase in athletic activity²⁴ with partial resolution of clinical signs after periods of rest.⁵ In middle aged to older horses a traumatic event is usually associated with the onset of the lameness.¹⁰ The degree of lameness can vary from mild to severe and the onset can range from insidious to acute.¹⁰ In one report, for example, 15% of the horses with stifle bone cysts were lame at the walk, whilst 76% showed lameness when trotted in hand.² In another group of horses with medial femoral condylar cystic lesions, the majority of horses were only a grade 1 to 2 out of 5 lame (AAEP scale) in the affected limb.⁷ Synovial effusion may be present, but is not consistent in all cases and is not pathognomonic.⁵ Mild effusion of the femoropatellar joint was reported in only 15 of 41 cases of subchondral cystic lesions of the medial condyle of the femur in one study⁷ and effusion of the medial femorotibial

joint was even less common, occurring in only 2 out of the 41 horses diagnosed with subchondral bone cysts.^{5,7} Muscle atrophy of the affected quarter can occasionally be noted but again is not a consistent finding and is not specific to stifle lameness.² Often, no localizing signs are obvious on physical examination making the localization and diagnosis of the source of lameness challenging.⁴¹

The differential list for a hindlimb lameness varies depending on the severity and presentation of the lameness. If a horse presents with severe, acute lameness and concurrent palpable synovitis, the site of lameness is usually easily recognized and the area can then be imaged.²³ The diagnosis is much more difficult for the majority of horses with a subchondral bone cyst where no palpable abnormalities, and a mild, chronic hind limb lameness exist.²³ The list of differentials for this presentation of a non specific hindlimb lameness is considerable and must be refined.

A thorough lameness evaluation, including perineural and intra-articular diagnostic nerve blocks is required to rule out other causes of lameness and to localize the site of the pain to the stifle before a manageable list of differentials can be presented.²³

It is rarely possible by gait analysis alone to identify the source of a lameness. However, a combination of clinical signs and history can lead to a suspicion as to the source of the lameness.⁴² Some horses with stifle pain stand with the stifle rotated outwards, often having a reduced cranial phase to the stride.⁸ Most affected horses swing the leg medially and the lameness can often be accentuated when trotting in a circle with the affected limb on the inside.⁴³ Bilateral stifle pain may result in poor hindlimb impulsion rather than overt lameness.⁸ Proximal limb flexion often increases the lameness.

However, a negative response does not eliminate the stifle as the source of pain. It has

been generally reported that a very marked response to upper limb flexion is usually more likely to reflect stifle pain than hock pain.⁷ It has, however, also been stated that horses with subchondral bone cysts of the medial femoral condyle have a lesser degree of increase in lameness in response to hindlimb flexion compared with horses with lameness of tarsal origin.⁷ A more specific stifle flexion test can be performed by holding the limb behind the horse in the shoeing position.⁴⁴

If signs localizing the stifle as the source of pain are absent or equivocal, as is often the case, perineural anesthesia must be performed. This type of anesthesia is preferred over selective joint blocks as it more consistently abolishes pain from all aspects of the joint and surrounding soft tissue.⁴⁵ It can be used successfully to eliminate the distal limb as the source of pain, but its use is not possible in upper limb situations, for example, the stifle or pelvic areas.⁶

Perineural and intra-articular anesthesia are integral parts of many lameness investigations.⁴⁶ However, due to diffusion of local anesthetic the technique is not always specific or reliable.⁴⁶ False positives and negatives can be a confusing element of diagnostic nerve block interpretation. The speed of response to perineural anesthesia can also be variable. The anesthetic solution must diffuse into the nerve and this process takes longer with larger nerves. Distal limb blocks, those distal to the tarsus, should be assessed 10 minutes and up to 30 minutes post injection and proximal limb nerve blocks should be assessed at 20 minute intervals up to 1 hour post injection.^{45,46} Needle placement should be as accurate as possible to enhance the specificity of the anesthesia.⁴⁶ The volume of local anesthetic solution should also be

as small as possible to improve accuracy of the block by discouraging excessive diffusion.⁴⁶

Several local anesthetic products are available. All products block or inhibit nociceptive nerve conduction by preventing the increase in membrane permeability to sodium ions.⁴⁷ Mepivacaine is reported to be relatively fast acting and has a duration of action of 2-3 hours.⁴⁵ It is the least irritant of the short acting local anesthetic solutions and is the drug of choice in most situations.⁴⁶

Performing perineural and intra-articular analgesia of the hindlimbs is potentially dangerous.⁴⁸ Thus, although in principle nerve blocks should be performed sequentially working from the most distal proximally, the hazardous nature of hindlimb anesthesia permits exceptions to this rule. It is generally accepted that if the results of the clinical examination cannot localize the problem to the fetlock or distal limb, it is justifiable to begin with a low 6 plantar block.^{46,48} However, clinicians that begin with a low plantar block in order to limit the number of hindlimb injections in horses that lack clinical signs referable to the digit must be aware that performing blocks distal to this site at another time may be necessary if baseline lameness is alleviated using this strategy.⁴⁵

Restraint is important if hindlimb nerve blocks are to be performed safely.⁴⁶ Most horses respond well to the use of a twitch.^{45,46} However, sedation is sometimes required to perform the procedure safely.⁴⁶ Xylazine, detomidine and butorphanol can be used in a variety of combinations. The use of an alpha 2-agonist such as xylazine or detomidine may lower the horse's anxiety level but its tendency to kick may become less predictable.^{45,46} The use of sedation does not have a significant effect on the pain

causing the lameness, although it does usually mean that the horse cannot be safely trotted for around 20 minutes post sedation.⁴⁶

For hindlimb anesthesia the horse's tail should be tied and secured with tape.⁴⁶

Prior to performing perineural anesthesia the area should be thoroughly cleaned using an antiseptic solution. If there is a danger of penetrating a synovial cavity or if performing intra synovial anesthesia the area should be prepared using routine aseptic technique.⁴⁶

The smallest needle possible should be used, although there is a compromise between safety, speed of injection and the risk of breaking a very fine gauge needle.⁴⁶ Perineural injections are usually performed using needles ranging from 25 gauge to 20 gauge.⁴⁵

Eighteen to nineteen gauge needles are recommended for injections within the proximal metatarsal or plantar tarsal regions.⁴⁵ The needle should always be inserted detached from the syringe.^{45,46}

Efficacy of the block should be assessed before assessing whether or not lameness has improved. Perineural blocks must be assessed for the amelioration of deep and superficial pain. Extreme joint flexion can be used to assess deep pain, as can hoof testers when testing digit anesthesia.^{45,46} Complete elimination of skin sensation in the hindlimb is less predictable than in the forelimb due to the variable distribution of cutaneous innervation.⁴⁵ It must be appreciated that hindlimb lameness is rarely fully alleviated by local analgesia. An improvement in the degree of lameness greater than 70-80% after most perineural or intra-articular techniques should be considered a positive response in most horses.⁴⁵ The horse must always be reassessed under the same conditions as the lameness was first seen.⁴⁶

Several different methods exist for initiating diagnostic analgesia in a horse exhibiting hindlimb lameness, with the approach depending largely on the presentation and the clinician's preference. Effusion and the presence of localizing signs in any of the stifle joint compartments, for example, would lead to a high index of suspicion and an acceptable jump to intra-articular anesthesia of the stifle, bypassing more distal analgesia.⁴⁶ When presented with no localizing signs, however, perineural blocking should start distally or, alternatively, regional analgesia can be induced midway up the limb to first localize the lameness to either the distal or proximal aspect of the affected limb.⁴⁹

Working sequentially from distal to proximal the following blocks would be performed: plantar digital, dorsal ring of the pastern, abaxial sesamoid, low plantar, high plantar and tibial/peroneal.⁴⁵ The likelihood of lameness being abolished by plantar digital anesthesia is considerably lower in the hindlimb than the forelimb, but is still a possibility, thus it is considered by some as a good starting point.⁴⁵ The abaxial sesamoid block is avoided in racehorses, if possible, due to the high prevalence of lameness involving the metatarsophalangeal joint with the possibility for misdiagnosis.⁴⁵ Analgesia of the metatarsophalangeal joint region is achieved by using a low plantar block (low 6 point block). It is essential to anesthetize the medial and lateral plantar, the medial and lateral plantar metatarsal, and the dorsal metatarsal nerves in order to achieve a successful block.⁴⁵

A high plantar nerve block (high 6 point block) provides subtarsal analgesia by anesthetising the medial and lateral plantar nerves, medial and lateral plantar metatarsal nerves and the dorsal metatarsal nerves at the subtarsal level.⁴⁵ In this

proximal metatarsal region innervation to the suspensory ligament is provided by the plantar metatarsal nerves and the digital flexor tendons receive their innervation from the plantar nerves. Due to the infrequent nature of superficial and deep flexor tendon injuries in the hindlimb it has been suggested that the plantar metatarsal nerves be blocked alone initially.⁴⁶ Dyson(1997) believes that if the response to both a low plantar and subtarsal analgesia of the plantar metatarsal nerves is negative, subsequent additional blockade of the dorsal metatarsal and plantar nerves rarely produces any alteration in lameness.⁴⁶ Caution must be used when performing and interpreting a high plantar nerve block due to the possibility of inadvertent placement of anesthetic solution into the tarsometatarsal joint via its distoplantar outpouchings and of inadvertent placement in the tarsal sheath.⁴⁵ A single injection technique for diagnostic analgesia of the proximal suspensory ligament of the equine hindlimb has been reported.⁵⁰ The technique has been shown in vitro to provide a reliable method using a single needle puncture for perineural analgesia of the deep branch of the lateral plantar nerve, with minimal risk of inadvertently desensitizing structures within the tarsal sheath and the tarsometatarsal joint.⁵⁰ However, to provide complete distal desensitization further anesthesia of the medial plantar nerves would be necessary.

If results of subtarsal analgesia are negative, the next structure to be anesthetised is the tarsal region.

The hock is innervated by the deep and superficial peroneal nerves, the saphenous nerve and the tibial nerve. Analgesia of the hock can be performed by anesthetizing the peroneal and tibial nerves.⁴⁶ These nerves are large and, therefore, anesthesia can take longer to achieve than that of more distal nerves. As a result, these nerve blocks

can be difficult to interpret.⁴⁸ The efficacy of this block is assessed by reduced sensation at the heel bulbs and a reduced reaction to palpation of the body of the suspensory ligament.⁴⁸ It should be noted that there can be an increased toe drag, despite an improvement in lameness when using this type of anesthesia.⁴⁸

Intra-articular analgesia of the hock joints is in, theory, the most specific way to diagnose distal hock joint pain.⁴⁶ However, this intra-articular technique can result in false negative results and, because the plantar outpouchings of the tarsometatarsal joint are closely related to the plantar metatarsal nerves, there is also a possibility of false positive results due to alleviation of pain from the proximoplantar metatarsal region.⁴⁶

The peroneal and tibial blocks when completed successfully are more effective at eliminating pain from the hock joints than intra-articular analgesia. They are considered by many as the most reliable method of localizing pain to the tarsal region.⁴⁸

Additionally, these perineural blocks eliminate pain associated with subchondral trauma of the distal tibia and talus, distally located tibial stress fractures, pain associated with the tarsal sheath, the distal aspect of the common calcaneal tendon, the calcaneal bursa and the plantar aspect of the hock.⁴⁵ Therefore, to assist in the diagnosis of an ambiguous lameness these blocks may be superior. Perineural analgesia of the tibial nerve alone can be useful in certain cases of suspected proximal suspensory desmitis, which have failed to respond to subtarsal analgesia.⁴⁶ It should be cautioned that if a high plantar nerve block has not been performed the tibial/peroneal block will eliminate pain from a proximal suspensory desmitis.⁴⁵

If all limb analgesia distal to the stifle is negative, if there has been known trauma to the stifle region, or if there are obvious clinical signs that localize the stifle as the source of

lameness, it may be reasonable to move directly to intra-articular analgesia or imaging of the stifle joint.⁸ Such localizing signs would include distention of the medial femorotibial joint, outward rotation of the stifle, exaggerated lifting of the limb during flexion.⁸

It is recommended to block all three stifle compartments simultaneously, unless there is definitive clinical suspicion of a stifle problem involving a specific compartment.⁴⁸ It has been reported that blocking each joint independently may result in an overall false negative result or complication with interpreting results due to communication between compartments.⁴⁸ Results of diagnostic intra-articular anesthesia of the stifle can be equivocal when dealing with a subchondral bone cyst. The anesthesia will often improve the lameness, although it will not abolish it and occasionally there will be no improvement in lameness grade.¹⁷ In one report of eleven horses with medial femoral condylar cystic lesions, however, all eleven demonstrated reduced lameness following intra articular anesthesia.⁷ This variance in results is because the subchondral bone is often the source of pain and, therefore, local anesthetic solution must diffuse into that area. Subchondral bone receives its innervation from endosteal branches of peripheral nerves that enter the medullary cavity through a nutrient foramen and local anesthetic solutions may not penetrate the subchondral bone sufficiently to completely block these nerves.⁴¹ The results of the anesthesia can initially be negative, and it is, therefore, recommended that re-examination is performed one hour after injection, allowing the anesthetic time to diffuse into the cyst and surrounding bone.⁶ It has been suggested that a 50% improvement in lameness is enough to justify radiography of the stifle.⁴¹ If the region of lameness cannot be localized through clinical signs and perineural

analgesia, imaging studies should be undertaken with the aim of identifying the region of lameness. Depending on the clinical presentation and history this may involve survey radiographs or a scintigraphy examination.^{42,44}

With the stifle identified as the source of pain the list of differentials can be targeted.

This list of differentials for lameness originating in the stifle includes but is not limited to: subchondral bone cysts, osteochondrosis dissecans, osteoarthritis, epiphysitis, patellar fixation, fragmentation of the apex of the patella, patella fractures, ligamentous and meniscal damage and infection.⁸ The history and clinical signs can help eliminate some of the differentials from the list. For example, if the horse has never been seen to stand with the hindlimb in extension whilst the fetlock is flexed, patellar fixation can usually be removed from the list. Horses with osteochondrosis dissecans (OCD) are normally presented prior to training due to distention from synovial fluid effusion, whereas horses with subchondral bone cysts usually present following the commencement of training due to the development of lameness.⁵ Patella fractures are usually the result of a direct trauma, either a kick or hitting a fixed fence, resulting in sudden onset severe lameness.⁴¹

Sometimes, however, the stifle cannot be identified as the source of upper limb lameness due to negative or equivocal regional anesthetic results. In these cases diseases of the femur and pelvis must also be considered in the differential list for the hindlimb lameness. Conditions of the pelvic area that need to be ruled out of an upper limb hindlimb lameness differential list include stress fractures of the ileal wing, fractures of the tuber ischii, fractures of the sacrum, sacroiliac joint injury, aorto-iliaco femoral thrombosis, osteoarthritis of the coxofemoral joint, luxation of the coxofemoral joint.⁸ A

rectal examination may be indicated, when trying to rule out conditions of the pelvis.⁴²

Proximal tibial stress fractures will also not always be anesthetized by perineural analgesia and therefore must be considered in any lameness that has failed to be localized by analgesia.^{41,45} Finally, muscle conditions need to be eliminated from the list of differentials. Such conditions include recurrent exertional rhabdomyolysis and fibrotic myopathy. Diagnosis for this group, however, can usually be made by analyzing blood chemistry and hematology results and on clinical grounds.³

Although history, clinical signs, regional and intra-articular nerve blocks can help localize the stifle as the source of the lameness, the definitive diagnosis of a subchondral bone cyst is achieved by radiography.^{23,24}

Radiographic projections should include lateromedial, caudolateral-craniomedial oblique, caudocranial and a flexed lateromedial.⁷ Subchondral bone cysts are usually observed most clearly on the caudocranial view and they may be difficult to appreciate on the oblique view due to superimposition of the condyles or summation from the intercondylar eminences of the tibia. A number of caudocranial views with varying proximodistal inclination of the x ray beam are necessary to evaluate the subchondral trabecular pattern and the depth of the lesion.⁵¹ A flexed lateromedial view allows better visualization of the proximal tibia in the region of the intercondylar eminences and assessment of the proximal aspect of the trochlear ridges of the femur, without superimposition of the patella.⁵¹ Radiographs should be carefully assessed for evidence of degenerative joint disease, as the presence of osteoarthritis decreases the prognosis for resolution of the lameness in the affected horse.²³ Radiographic examination of horses with medial femoral condylar bone cysts should always include

the contralateral limb as bilateral involvement has been widely reported, even in horses with unilateral lameness.⁷ Radiographs should always be suitably exposed as underexposed radiographs can allow the lesion to go undetected.⁹

Radiographically, subchondral bone cysts in the medial femoral condyle are usually round or oval, with a variably sized base and communication with the joint. A sclerotic rim is seen in horses with older lesions.⁴¹

Subchondral bone cysts of the medial femoral condyle have been categorized by different authors over the years, based on radiographic appearance¹³ and location.²

The cases in one study could be divided into two groups according to radiographic features: Group A had a clearly defined bone cyst in the center of the medial femoral condyle, adjacent to and communicating with the femorotibial joint. Group B horses had one or more less clearly defined cysts in the region of the intercondyloid fossa of the femur and/or the proximal extremity of the tibia.² Another report categorized the medial femoral condyle lesions on the basis of their depth into the medial femoral condyle, which was then correlated with the lesion size and return to function after surgery.

Caudocranial views were used and measurements were taken from the articular surface to the most proximal extent of the lesion. The depth of the lesion was measured directly during arthroscopy. Type 1 lesions were 10 mm or less in depth, appearing as shallow, concave defects on the weight-bearing surface of the medial femoral condyle. Type 2 lesions were more than 10 mm in depth and typically conical. Type 3 lesions had a flattened contour of the subchondral bone at the distal aspect of the medial femoral condyle. This class of lesion was best observed on the flexed lateralmedial view, and did not extend to any measurable depth within the condyle.⁷ Yet another classification

system determined lesion size and type by using a modification of the above categorization by Howard, et al. (1995).⁷ Caudocranial radiographic views were used, measurements were taken of the height and width of the subchondral bone cyst, and the width of the medial femoral condyle. The height and width of the cyst were added and the total was divided by the medial femoral condyle width, compensating for differences in magnification between examinations.⁵² Type 1 lesions were defined as being <10 mm in depth and were usually dome shaped. Type 2A lesions were >10 mm in depth and had a lollipop shape with a narrow cloaca and a round cystic lucency. Type 2B lesions were > 10 mm in depth with a large dome shape extending down to a large articular defect. Type 3 lesions were defined as condylar flattening. Type 4 lesions had a lucency in the condyle with or without an articular defect, and had no radiographic evidence of a cloaca in the subchondral bone plate.⁵²

It has been reported that the flattening of the distal medial femoral condyle which is observed occasionally radiographically may be a precursor to a subchondral bone cyst.⁴¹ It has been suggested that horses with hindlimb lameness and even subtle radiographic lesions of the medial femoral condyle are likely to have arthroscopically apparent cartilage lesions and subchondral bone defects.⁵³

Regardless of the location, size, shape or degree of articular involvement of subchondral cystic lesions, the most consistent radiographic finding is a circumscribed area of subchondral lucency with surrounding sclerosis.⁹ Correlation between clinical and radiographic findings is necessary to confirm that the subchondral cystic lesion is the cause of lameness. This is particularly important as some of these lesions can be asymptomatic and sometimes can be incidental findings.^{23,24}

Occasionally, the cyst may not be obvious using conventional radiography and other modalities such as ultrasound, nuclear scintigraphy, or computed tomography (CT) may be helpful in confirming a diagnosis of a subchondral bone cyst.^{54,55,56}

Ultrasonographic examination of the stifle is regarded by some as an essential part of the assessment of stifle injuries.⁵⁶ Ultrasound examination is performed using a 7.5MHz linear transducer, with the stifle flexed in the standing, sedated patient. In this position, the medial femoral condyle becomes relatively superficial. On parasagittal and transverse views, the normal medial femoral condyle appears as a smooth convex surface. The subchondral bone surface is imaged as a hyperechogenic line, whilst the articular cartilage is imaged as an anechogenic layer that usually measures 2 mm thick in an adult horse. A subchondral bone cyst lesion of the medial femoral condyle is imaged as a defect in the smooth subchondral bone outline.^{56,57} The ultrasonographic depth of the defects in one study were shown to range from 2-10mm. The contents of the cyst were anechogenic in some cases but contained hyperechogenic material, indicative of mineralized or bony material, in other cases.⁵⁶

Ultrasonography appears to be the most sensitive diagnostic tool available to diagnose subchondral bone cysts of the medial femoral condyle.⁵⁶ In one study, 204 subchondral bone cysts that were diagnosed radiographically were also identified using ultrasound. However, some small cysts that were not diagnosed radiographically were identified during the ultrasound examination.⁵⁶ The main advantages of ultrasound include: the ability to assess the articular cartilage which is frequently thickened at the level of the lesion⁵⁴ and the ability to evaluate the soft tissues of the stifle, in particular the synovial fluid in the medial femorotibial recess, and the synovial membrane. Thickening of the

synovial membrane is indicative of chronic femorotibial arthropathy.⁵⁶ Ultrasound also allows evaluation of the medial and lateral menisci and collateral ligaments, which is of prognostic importance due to the reported association between subchondral bone cysts and medial meniscal injuries.⁵⁸ When a subchondral bone cyst is identified in the medial femoral condyle the medial meniscus should be examined carefully using ultrasound.⁵⁶ Finally, early detection of changes at the articular margins can be evaluated using ultrasound. These changes are not always detectable radiographically.⁵⁶ Complimentary investigation using these two imaging modalities is always indicated.⁵⁶ Many times only the open part of the cyst (cloaca) can be seen with the ultrasound: when the cyst extends further into the medial femoral condyle, the deeper part of the cyst cannot be imaged. Radiographic examination using a caudocranial projection is, therefore, required to completely assess the lesion in such instances. Like radiography, ultrasonography can easily be performed with a portable machine in the field.⁵⁶

Scintigraphic evaluation of horses with subchondral bone cysts is performed infrequently because radiographs and ultrasound examination usually confirm the diagnosis.²³ Its use, however, may be necessary in cases where no source of lameness has been identified.⁴⁴ Scintigraphy can be helpful to identify the location of the cyst only if the surrounding subchondral bone has had sufficient time to react. Scintigraphy may also be negative in the acute subchondral cystic lesion.⁶ Such lesions are termed scintigraphically silent.⁶ The ability of scintigraphy to diagnose or monitor subchondral bone cysts in the medial femoral condyle of horses is questionable.⁵⁹ One author demonstrated the inability of scintigraphy to demonstrate radiopharmaceutical

uptake in a horse, post surgical debridement, despite radiographic enlargement of the lesion. The ability to remain scintigraphically normal was postulated to be due to increased osteoclastic activity versus osteoblastic activity.⁵⁹ For these reasons, scintigraphy is usually indicated in cases with no localizing signs, to eliminate other potential diagnoses, rather than to specifically diagnose a subchondral bone cyst.⁴⁴ Scintigraphic examination would also be indicated in selected cases where the dangerous behavior of the horse prohibited the use of perineural or intra-articular anesthesia.⁴⁴

Other imaging modalities are noted but used very rarely for this diagnosis and include Computed Tomography (CT)⁶⁰ and Magnetic Resonance Imaging (MRI).⁶¹ CT of the equine stifle is possible in clinical cases and has been used to identify subchondral cystic lesions that could not be confirmed radiographically.⁶² Although CT has the advantage of being able to visualize both bony and soft tissue, not all CT gantries are suitable for examination of the equine stifle.⁶⁰

Although MRI can visualize soft tissue and cartilage and is the preferred technique for examination of the knee in humans,⁶³ it is only presently available for experimental diagnosis of the stifle in the horse.⁶⁴ Both MRI and CT, moreover, require general anesthesia to allow adequate imaging and, despite the advent of mobile units, these modalities are still not universally available.

There are two basic approaches for treatment of most subchondral cystic lesions: (1) conservative treatment with rest and medication and (2) surgical treatment. Factors that must be considered when choosing a treatment plan include: duration of the lameness, age of the horse, intended use of the horse, secondary articular changes such as

degenerative joint disease, additional lesions within the joint (meniscal tears) and financial constraints of the owner.²³

Conservative treatment, generally, is best reserved for young horses with non articular or small articular defects and no arthritic change.⁴¹ Surgery, however, is typically recommended whenever athletic soundness is required.²⁴

A successful outcome is considered as a return to soundness, as it has been reported that subchondral bone cysts may not change radiographically despite two years of rest.⁶⁵ Therefore, to equate radiographic disappearance of the cyst to a success may be misleading. It should be noted that radiographic appearance is not a reliable indicator of the degree of healing for any of the treatment modalities.²³ The definition of success as a return to soundness should also be cautioned as some horses will perform extreme forms of work before exhibiting lameness with this condition.^{9,66} Horses that do not respond to conservative treatment within 3-4 months or those that worsen should be treated surgically.²⁴

Conservative treatment can involve a rest period of at least 6 months.²³ This can be stall rest, small pasture turnout⁴¹ or continued light exercise.⁶⁶ McIlwraith(1982), however, cautions the use of continued light exercise, having observed breakdown of the medial femoral condyle in two cases with subchondral bone cysts of the medial femoral condyle.⁹

It has been suggested that conservative treatment alone may result in a temporary abatement of clinical signs, but lameness frequently returns once rigorous exercise resumes.⁷ There have been reports, however, of horses that have returned to racing following conservative treatment involving lesions of the stifle.² Adjunct treatment with

intra-articular hyaluronan and polysulfated glycosaminoglycan and systemically administered nonsteroidal anti-inflammatory drugs can be used in conjunction with a period of rest.³⁷ These medications decrease concurrent synovitis and possibly reduce intraosseous pain.⁶ Their effect on bone remodeling around the defect and rate of resolution of the lesion is unknown.²³

Management strategies involving surgical treatment include: debridement via arthrotomy,¹³ debridement via arthroscopy,⁷ debridement with cancellous bone grafting,^{67,68} debridement with bone substitute and chondrocyte or progenitor cell graft in fibrin glue,⁶⁹ or steroid injection into the fibrous lining under ultrasonographic⁷⁰ or arthroscopic guidance.⁵²

The goals of surgical debridement are enucleation of the cystic contents and removal of the damaged cartilage at the articular margin which lacks adequate subchondral bony support. Surgical debridement involves removing the abnormal tissue within the cyst including the cyst lining, and then scraping the subchondral bone until as much sclerotic subchondral bone as possible is removed. One author suggests this to be considered sufficient when normal cancellous bone is obtained from all sides of the cavity and subsequent bleeding from the opened cavity is observed.¹³ Debridement of the surrounding sclerotic rim down to normal healthy bone should provide an adequate stimulus for osteoblast migration into the organized blood clot formed following debridement.⁷¹ In summary, the goal of surgical debridement is to remove the fibrous cyst lining and adequately open up the cyst cavity. This eliminates the source of inflammatory mediators and permits the filling in of the defect with healthy tissue.¹³ Reports on the success of surgical debridement of subchondral cystic lesions in the

medial femoral condyle range from as high as 90 %¹³ to less than 50%.⁷ One study evaluating racehorse performance after arthroscopic debridement of the lesions demonstrated that by the time horses reached their four year old year, horses that had undergone surgery started an equivalent number of times and earned an equivalent amount for average earnings per start as their siblings.⁷²

These success rates can be analyzed further. One author reported that 64% of horses with cysts in the medial femoral condyle aged 0-3 years old returned to soundness following debridement, while only 35% of horses older than 3 years returned to soundness following debridement.⁴⁰ It is postulated that the coexistence of degenerative joint disease explains why the older population of horses had a lower success rate.⁴⁰ It was also noted that older horses take a longer time to return to work, with a median time of 24 months.⁴⁰

One of the greatest disadvantages to the debridement procedure is the fact that this technique leads to disruption of the articular surface and removal of variable portions of weight bearing cartilage. This finding is significant as it has been reported that the ability to start a race is directly influenced by the amount of cartilage surface disrupted at surgery.⁷² The need to preserve articular cartilage during surgery is illustrated by a report indicating a 70% return to soundness if the surface area of cartilage affected on the medial femoral condyle was <15 mm but only a 30% success rate if the affected cartilage was >15 mm.⁷² One additional complication of debridement of subchondral bone cysts in the medial femoral condyle is the association between debridement of subchondral bone cysts and subsequent formation of medial meniscal lesions. This has been documented in six horses subsequent to subchondral bone cyst debridement.⁵⁸ It

is suggested that the removal of cartilage and bone overlying the cyst cavity may alter the contour of the medial femoral condyle, precipitating damage to the underlying meniscus, or that possibly the rim of bone left at the edge of the debrided defect could act to damage the meniscus.⁵⁸ Although uncommon, injury to the meniscus subsequent to surgery should be considered as a complication of debridement and should be investigated in horses that remain lame or worsen after surgery.⁵⁸ One author has suggested that surgical debridement of the lesion should be performed with caution and possibly abandoned in favor of arthroscopic injection to avoid the risk of subsequent meniscal damage.⁵⁸

Surgical forage has been used following debridement of the subchondral cyst from the medial femoral condyle, to promote healing of the subchondral bone. It is now rarely performed as it has been shown to result in enlargement of the subchondral defect postoperatively.⁷ It is thought that drilling of the lesion may increase exposure of the epiphyseal bone to inflammatory mediators and degradative enzymes, therefore increasing bone resorption, rather than promoting filling in of the defect.⁷

Cancellous bone grafting has also been used to treat subchondral bone cysts of the medial femoral condyle following debridement of the cyst cavity.^{23,67,68} The cancellous bone graft can be harvested from the contralateral tuber coxae⁶⁷ or from the sternum.⁶⁸ The key to a successful graft is revascularization. The graft is packed tightly into the cyst cavity after extensive curettage of the cyst wall.⁶⁷ The benefits of cancellous bone grafting lie in the transfer of an autogenous material with osteoinductive, osteoconductive and osteogenic properties.⁶⁹ However, there are inherent problems associated with using bone grafts for the treatment of subchondral

bone cysts. The most pertinent problems are as follows: firstly, most cysts are at least 2 cm in diameter, which leads to poor vascularization and subsequently poor reossification of the core of the graft;⁶⁷ secondly, weight bearing and movement are counterproductive to graft revascularization and unfortunately it is impossible to immobilize the equine stifle joint;⁶⁷ thirdly, it has been reported that only 10% of grafted cells survive transplantation under ideal conditions⁷³ and finally, one author has suggested that grafting of the medial femoral condyle regardless of graft type is likely to result in secondary cyst formation.⁶⁸ It is suggested that cyst formation between the graft and the parent bone may result from synovial fluid being forced through spaces created during the compaction of the cancellous bone graft, which leads to the inhibition of neovascularization and graft incorporation.⁶⁸

Overall, no benefit has been observed by using cancellous bone grafts. One study demonstrated that cartilage healing was similar in grafted versus ungrafted surgical defects in the medial femoral condyle at six months post operatively, indicating that grafting has no advantages over surgical debridement alone.⁶⁸ Another author recommended reserving the use of cancellous bone grafts for very large lesions where the structural integrity of the bone was in question.⁶

Due to the unpredictable results found with cancellous bone grafting, numerous other forms of graft have been attempted.⁷⁴ Autologous osteochondral grafting has been proposed as an alternative to cancellous bone grafts.⁷⁴ Autologous osteochondral grafts are harvested from the abaxial aspect of the contralateral medial femoral trochlea through a small arthrotomy. Recipient holes are then drilled 8-10mm deeper than the cyst cavity. It has been suggested that drilling the recipient holes removes most of the

fibrous lining and sclerotic bone of the cystic lesion and that osteochondral grafts reconstruct the articular surface and load bearing capacity of the subchondral bone. One small study showed 3 out of 5 horses with a subchondral bone cysts in the medial femoral condyle returned to a previous level of activity following this procedure following a variable period of convalescence.⁷⁴

Other, relatively new techniques that are still in their infancy, include arthroscopic debridement and injection of fibrin glue/insulin-like growth factor-1(IGF-1) composite using gas distention of the joint, which has been reported to be a successful technique.⁶⁹ Seventy three percent of stifle subchondral cysts treated by this method returned to work for a minimum of 2 years post surgery.⁶⁹ To improve bone formation in the depth of the cyst, a two composite technique has been employed by these authors. Densely packed cancellous bone is used in the deeper regions of the cyst and a surface layer of fibrin laden with chondrocytes and IGF-1 is used to seal in the cancellous bone. Although no secondary cyst formation, such as described by Jackson, et al(2000), was observed, the initial bony filling was seen to develop stippling 4-8 months post surgery, which took up to 12 months to remodel and ossify.⁵⁸ It is for this reason that the same researchers have started to investigate the use of bone substitute materials to augment bone repair in the depth of cysts.⁶⁹

Studies to evaluate the use of bone substitute materials, such as the combination of osteoconductive tricalcium phosphate with osteoinductive materials, such as platelet rich plasma (PRP) and bone marrow aspirate concentrate (BMAC), are underway.⁶⁹

More recently, Wallis, et al(2008) reported a 67% success rate (return to soundness) with injection of corticosteroids, under arthroscopic guidance, into the fibrous lining of

the cyst of the medial femoral condyle.⁵² The aim of this procedure is to reduce the production of local inflammatory mediators located by the cyst lining. These mediators are thought to recruit osteoclasts which have been shown in vitro to cause active resorption of bone.¹⁴ It is suggested that prostaglandin E2 (PGE2) is a potent bone resorbing agent that is responsible for the osteolytic behavior of subchondral bone cysts. The beneficial effect of intralesional corticosteroid administration, therefore, can at least be partially explained by the inhibition of PGE2 synthesis by corticosteroids.¹⁴ In this technique, the needle is advanced into the cyst through and adjacent to the cloaca of the cyst. An effort is made to inject into the solid tissue within the cyst lining to avoid loss of the corticosteroid into the joint cavity. When the fibrous tissue is penetrated the cartilage overlying the cyst can be observed to bulge under pressure, upon steroid injection, and little or no corticosteroid is observed entering the joint cavity. The ability to visualize this 'bulging' is one of the advantages of this arthroscopic technique over an ultrasound guided method.⁵² This ability to visualize the cyst and joint allows observation of the injection process, whilst also allowing assessment of the health of the remaining joint. In one study, for example, seven of the horses treated had an associated osteochondral flap adjacent to the cloaca that required debridement. These flaps would not have been detected if injection had been performed under ultrasound guidance alone.⁵²

Corticosteroid injections (triamcinolone acetonide) are now considered by many as the preferred treatment option for treatment of subchondral bone cysts involving the medial femoral condyle.⁵² This technique offers equal success rates of between 67-77% to those found using debridement (67% Howard et al 1995 and 35-64% Smith et al.

2005)^{7,40} without any of the disadvantages of the debridement process. Unlike surgical debridement, moreover, the success rates with arthroscopic injection of corticosteroids do not seem to be influenced by age.⁵² Corticosteroid injection also seems to result in a lower amount of time for return to function: 2-4 months compared to 6-8 months post surgical debridement.^{40,52} One factor to note, however, is that the success of this procedure seems to be directly related to the surgeon who operated on the case.⁵²

One great advantage of this new technique over the other surgical techniques is that if the procedure is unsuccessful, the option to debride the lesion still remains.⁵²

The prognosis for subchondral bone cysts of the medial femoral condyle depends on a wide variety of factors and the determination of success depends on what criteria is used to judge success. It is widely accepted that radiography is not a sensitive way to evaluate the resolution of a subchondral bone cyst.²³ There does not seem to be any relationship between the radiographic density of a lesion and the eventual soundness of the horse,²³ regardless of treatment modality used. There is no association between a decrease in lesion size measured on radiographs and a successful outcome.^{24,52} A return to soundness, therefore, is the best indicator in determining success of a particular treatment modality.⁸ In most reports a successful outcome requires that horses perform soundly at their intended use and at a level equal to or above their performance prior to signs of subchondral bone disease. For young horses, success is considered if they perform soundly at their expected level.⁷

Factors thought to influence prognosis include the location and size of the lesion. Smaller lesions have a better prognosis than larger lesions when treated either

conservatively or with surgical debridement.^{6,66} For horses treated conservatively the location, shape and size of the cyst on the medial femoral condyle affects prognosis.² The age of the horse also affects prognosis. It has been demonstrated that older horses (>3 years old) carry a poorer prognosis for return to soundness following either conservative treatment⁶⁶ or arthroscopic debridement.⁴⁰ The amount of articular surface involved also influences outcome with cases with greater articular surface affected having a poorer prognosis.⁷² The presence of secondary degenerative joint disease is very important and seems to affect prognosis regardless of chosen treatment modality.^{6,13,40} The presence of concurrent meniscal or other soft tissue damage will negatively affect prognosis.⁵⁸ The intended use of the horse⁶ and the breed should also be considered: Quarter horses seem to have a poorer prognosis for soundness after surgical debridement, compared with Thoroughbreds and Arabians.⁷ Unilateral lesions seem to possess a better prognosis for return to work compared to bilateral lesions when treated conservatively or surgically.⁷⁵

Prognosis not only depends on the above factors, but also on the chosen treatment protocol as previously outlined. A success rate of 20%-50% has been documented for conservative treatment.^{24,66} In general, the success rate for treatment of subchondral bone cysts with debridement via arthrotomy and by way of arthroscopy is comparable.⁷ The success rate for arthroscopic corticosteroid injection into the fibrous lining of the cyst is equal to, and in some reports increased, compared to arthroscopy or arthrotomy.⁵² Arthroscopic debridement carries a 70% success rate for horses under 3 years of age⁴⁰ and one time intralesional injection of corticosteroids under arthroscopic guidance carries a 70% success rate regardless of age.

For intralesional injection by way of arthroscopic guidance, the age of the patient, number of joints and cyst size does not seem to affect prognosis.^{52,70} However, the presence of concurrent degenerative joint disease and in particular the pre surgical radiographic finding of osteophytes is an indicator of poor prognosis, as is the use of a single injection technique.⁵²

The prognosis for a horse with a subchondral bone cyst of the medial femoral condyle will, therefore, vary dramatically according to the above mentioned factors and the treatment modality chosen.

CLINICAL REPORT:

A 1 year old Thoroughbred colt presented with an insidious onset left hind limb lameness of approximately one week duration. The colt had recently started galloping exercise. At the onset of lameness the owner gave the horse 2.2mg/kg PO q12hrs phenylbutazone and stall rest for three days. The horse responded and the lameness subsided. The lameness returned, however, when the colt was next exercised.

This colt had previously received veterinary attention for routine herd health, but had no previous significant medical history and was fully vaccinated. At presentation, a physical examination revealed the colt to be bright, alert and responsive, with a body condition score of 5 (AAEP 1-9 scale). The horse was negative to hoof testers and had no obvious heat, pain, swelling or joint effusion in the left hind limb. He was a 3/5 lame left hindlimb (AAEP 1-5 scale) and had a mild positive response to upper hindlimb flexion in this limb.

The problem list for this horse included a mild left hindlimb lameness which responded to rest and anti inflammatories. There were no other clinical signs. The differential diagnoses for such a presentation are extensive.

Due to this horse's age, history and clinical signs osteochondrosis dissecans and subchondral bone cysts were high on the differential list for the cause of this hindlimb lameness. However, due to the lack of localizing signs, diagnostic nerve blocks were performed before a more definitive differential list could be composed, based on the region of pain. For all the nerve and joint blocks performed, the horse's tail was tied and secured with tape and the limb was prepared aseptically. Effectiveness of the anesthesia was assessed by evaluating deep pain through joint flexion combined with varus/valgus stress and hoof testers where applicable and by checking for skin sensation on the affected limb. In each case the normal limb was evaluated for comparison.

The horse was restrained using a twitch and a low plantar nerve block was performed on the left hindlimb. A 22 gauge 1 inch needle was used and 2 ml of mepivacaine hydrochloride^a was used at each site. The medial and lateral plantar nerves, the medial and lateral plantar metatarsal nerves and the dorsal branches of the plantar metatarsal nerves at the 2 and 10 o'clock positions subcutaneously on the cannon bone were anesthetized. The horse was evaluated at 5 minutes post injection, and at 10 minutes post injection. The horse's lameness grade was not improved following this block. A high plantar perineural block was subsequently performed. A 20 gauge 1 1/2 inch needle was used. This block was performed with the limb held off the ground. The needle was placed distal to the tarsometatarsal joint and axial to the fourth metatarsal

bone and was inserted until contact was made with the third metatarsal bone. Four ml of mepivacaine hydrochloride^a was deposited at this deep location to anesthetise the lateral plantar metatarsal nerve and another 4 ml was deposited as the needle was withdrawn to anesthetise the lateral plantar nerve. The procedure was repeated medially. For completeness a circumferential subcutaneous ring block was performed. Again, the horse was evaluated at 5 and 10 minutes post injection and there was no improvement in the lameness grade.

A tibial and peroneal perineural nerve block was then performed. The deep and superficial peroneal nerves were blocked at a site located laterally and 10 cm proximal to the tuber calcanei, in a groove between the long and lateral digital extensor muscles. At this location an 19 gauge 1 1/2 inch needle was buried to the hub and 10 ml of mepivacaine hydrochloride^a was injected beginning deep and continuing as the needle was withdrawn. The tibial nerve was blocked at a site 10 cm proximal to the tuber calcanei, cranial to the common calcaneal tendon, and caudal to the deep digital flexor tendon. A 20 gauge 1 1/2 inch needle was inserted laterally and 15 ml of mepivacaine hydrochloride^a was injected over the nerve. There was no improvement in lameness grade after evaluating the horse at 10, 15 and 30 minutes post injection. There was a delay of 2 hours between completing the tibial and peroneal block and commencement of stifle anesthesia. The horse's lameness grade was reassessed again at this stage before initiating the stifle anesthesia and was found to be unchanged.

Finally, all 3 compartments of the stifle joint were blocked simultaneously. The medial femorotibial joint was blocked using a 19 gauge 1 1/2 inch needle and 20 ml mepivacaine hydrochloride^a and the lateral femorotibial joint and the femoropatellar joints

were similarly blocked. Joint fluid was visualized from all 3 compartments and grossly appeared to be of a normal color and consistency. The horse was evaluated immediately post injection and at 5 and 10 minutes and 1 hour post injection. The horse became 50% improved when evaluated 1 hour post injection. Following diagnostic analgesia the horse's lower limb was covered in alcohol and bandaged for 24 hours. Intra-articular anesthesia was, therefore, considered successful at localizing the source of the lameness to the stifle and which allowed a refined differential diagnosis list to be compiled.

The differential diagnoses for stifle lameness in this young horse included the following; subchondral bone cyst, osteochondrosis dissecans, trauma, meniscal / ligamentous or other soft tissue injury, and infection. With the stifle identified as the region of pain it was subsequently imaged in an attempt to reach a definitive diagnosis.

Following diagnostic analgesia the horse was sedated with butorphanol tartate^b (0.05mg/kg IV) and detomidine^c (0.006mg/kg IV). Digital radiographs^d of the stifle were obtained: a lateromedial, caudolateral-craniomedial oblique, caudocranial and a flexed lateral views were obtained. A circular lucent area within the subchondral bone of the medial femoral condyle was observed on the caudo-cranial and flexed lateral views and determined to be a subchondral bone cyst. There appeared to be a visible communication between the cyst and the femorotibial joint (Figure 1). The cyst was surrounded by a sclerotic rim, but there was no evidence of concurrent degenerative joint disease (Figure 1, Figure 2). The defect measured 25mm proximo-distally, and 18 mm latero-medially. Using the Wallis (2008)⁵² scale this cyst was categorized as a

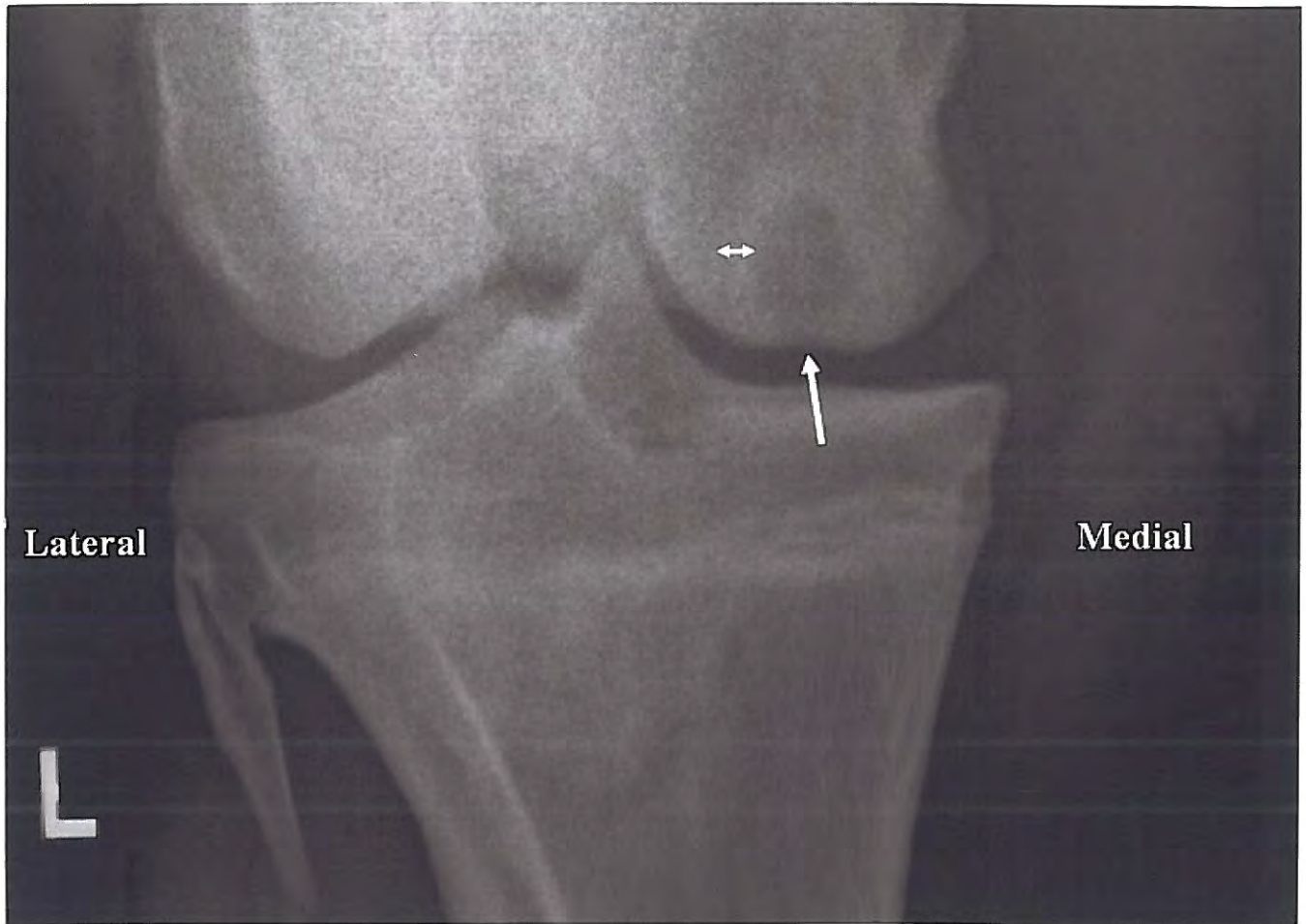


Figure 1: Caudocranial radiographic view of the left stifle. Note the circumscribed area of subchondral lucency with surrounding sclerosis (double ended arrow).

Communication between the cyst lumen and the femorotibial joint is also visible (white arrow).



Figure 2: Caudolateral-craniomedial oblique radiographic view of left stifle demonstrating sclerosis in the subchondral bone (arrow heads) and the defect at the articular margin (arrow) of the medial femoral condyle.

grade 2A (Figure 3). No abnormalities were seen on radiographic examination of the right stifle. Ultrasound^e examination of the stifle was subsequently performed. With the limb flexed a defect of the subchondral bone outline on the medial femoral condyle was imaged and was characteristic of a subchondral bone cyst (Figure 4, Figure 5). The depth of the defect measured 5.9 mm as assessed by ultrasound examination (Figure 6). The articular cartilage was thickened at the level of the lesion 3.1mm (normal <2 mm) and there was a mild synovial fluid distention of the medial femorotibial recess. No abnormalities were seen on examination of the medial meniscus or elsewhere in the joint. Ultrasound examination of the right stifle was within normal limits.

Following radiographic and ultrasonographic examination of the left stifle a definitive diagnosis of a subchondral bone cyst in the medial femoral condyle, without concurrent degenerative joint disease or meniscal damage, was made. It was decided to treat the subchondral bone cyst by injection of the lining with corticosteroids under arthroscopic guidance.

A complete blood count and serum chemistry were obtained pre operatively and performed in house. The results were all within normal limits (Table 1)

The left jugular vein was cannulated with a 14 gauge IV catheter.^f

Phenylbutazone^g (2.2mg/kg IV), Potassium Penicillin^h (22,000 IU IV) and Gentamicin Sulfateⁱ (6.6mg/kg IV) were administered pre operatively.



Figure 3: Caudocranial radiographic view of left stifle. Demonstrating the technique for obtaining measurements of the cyst as suggested by Wallis, et al (2008).⁵² This subchondral bone cyst measured 25mm proximo-distally, and 18mm latero-medially.

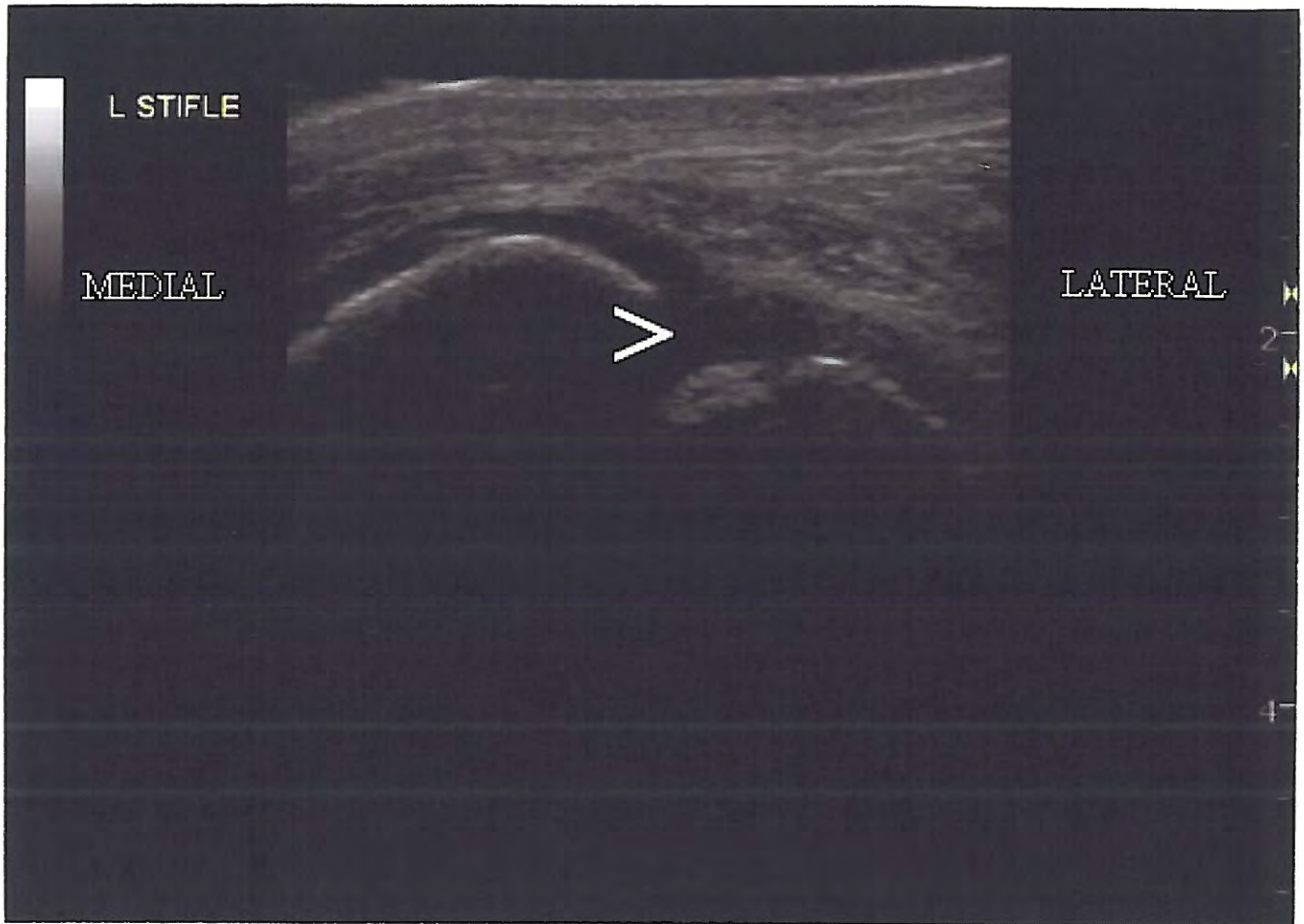


Figure 4: Transverse ultrasound view of the medial femoral condyle. There is a defect (arrowhead) in the medial femoral condyle, indicative of a subchondral bone cyst.

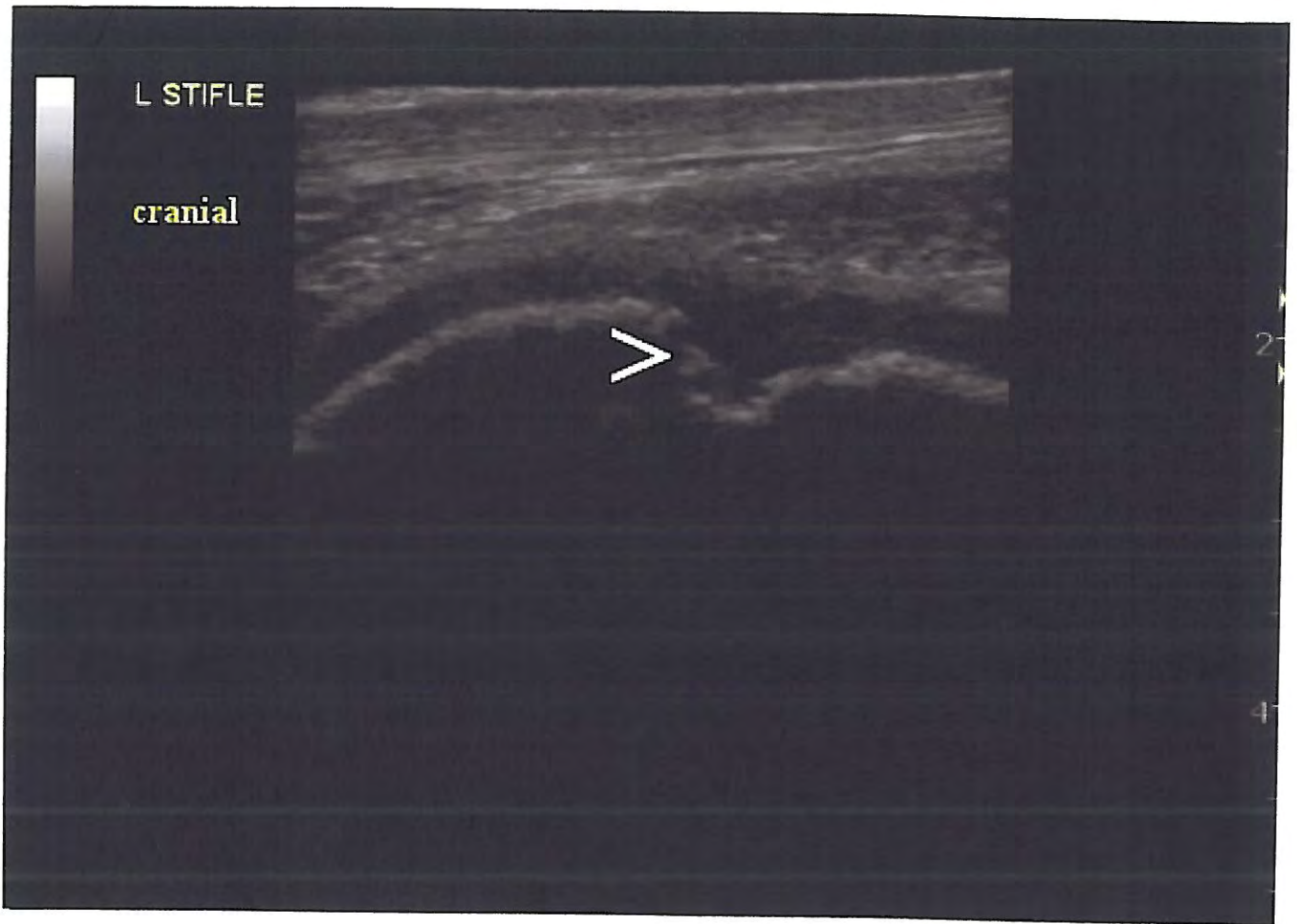


Figure 5: Parasagittal ultrasonographic view of the medial femoral condyle. The anechoic articular cartilage of the medial femoral condyle appears thickened at the level of the subchondral bone cyst (arrow head).

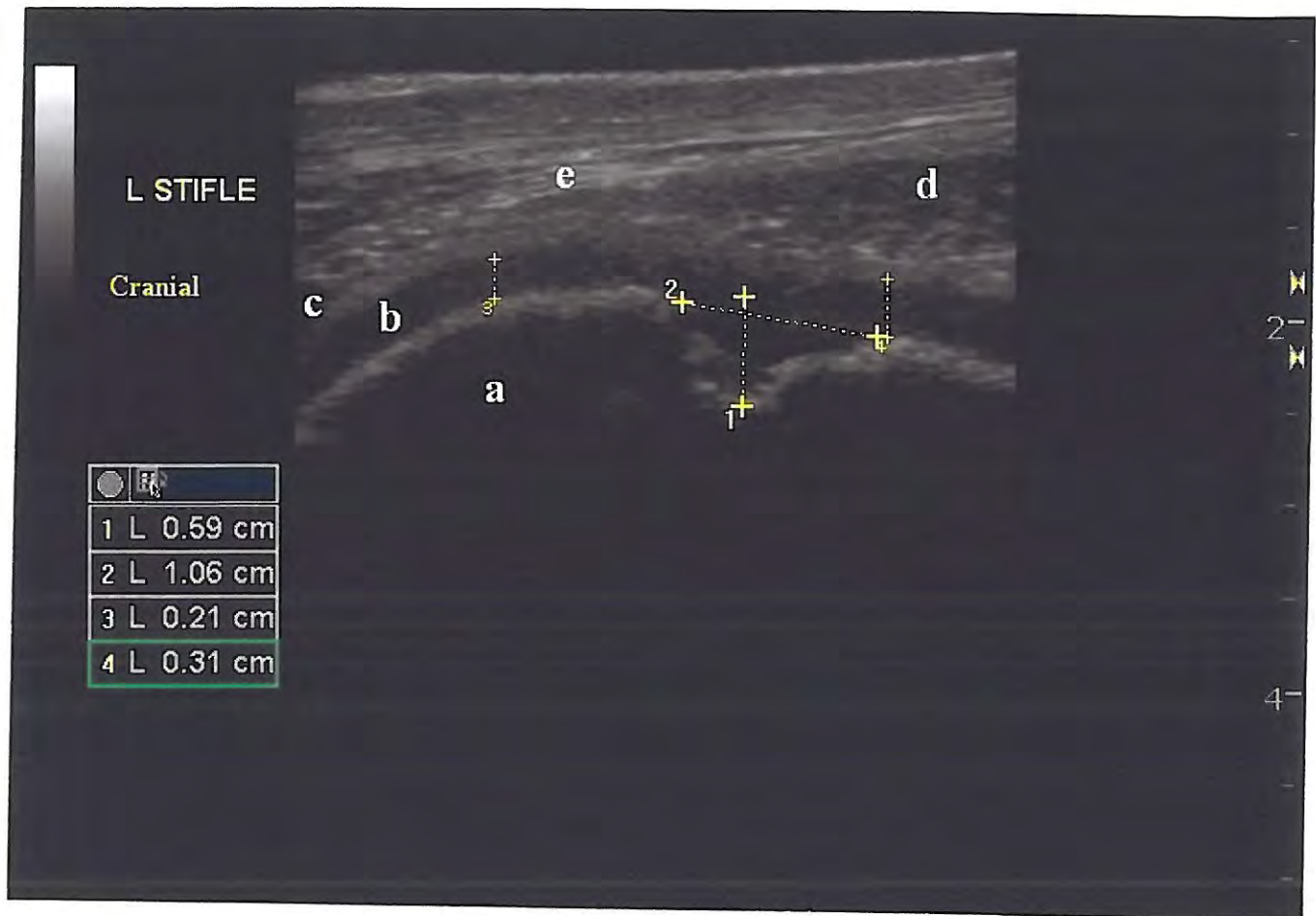


Figure 6: Parasagittal ultrasonographic view of the medial femoral condyle

demonstrating measurements indicative of a subchondral bone cyst:

a = Medial femoral condyle, b = Articular cartilage, c = Articular capsule,

d = Infrapatellar fat pad, e = Intermediate patellar ligament, f = skin.

1 = Depth of cyst (5.9mm), 2 = Width of cyst (10.6mm), 3 =Thickness of the normal articular cartilage (2.1mm), 4 =Thickness of the abnormal articular cartilage bordering the cyst (3.1mm)

TEST	RESULT	REFERENCE RANGE
White blood cell count	6.2	7.200-12.500/ UL
Red blood cell count	9.87	6.50-12.50 x 106/UL
Hemaglobin	13.5	11-17 gm/dl
Hematocrit	38	34-50%
Segmented Neutrophil	51	30-75%
Band Neutrophils	0	0-1%
% Lymphocytes	46	25-60%
Monocytes	3	1-8%
Eosinophils	0	1-2%
Platelet Estimate	183	200-400 x 103/UL
Total Protein	6.1	5.6-7.2g/dl
Fibrinogen	200	200-400mg/dl

TEST	RESULT	REFERENCE RANGE
Sodium	134	132-146 mmol/L
Potassium	3.2	2.4-4.7mmol/L
Chloride	97	95-104mmol/L
Aspartate Aminotransferase	301	226-365U/L
Creatinine Kinase	133	1-225U/L
Blood Urea Nitrogen	17	10-24mg/dL
Creatinine	1.0	0.7-2.0mg/dL
Alkaline Phosphatase	178	143-395U/L
Total Bilirubin	1.2	0-2.0mg/dL
Gamma-glutamyl transferase	15	0-36U/L
Calcium	11.2	10.7-13.5mg/dL
Albumin	3.1	2.3-3.9g/dL
Phosphorus	3.2	2.3-5.4mg/dL
Total Protein	5.8	5.6-7.9g/dL
Glucose	88	75-115mg/dL

Table 1: Complete blood count and serum chemistry values obtained prior to surgery were within normal range

The horse was premedicated with xylazine^j (0.8mg/kg IV). General anesthesia was induced with diazepam^k (0.1mg/kg IV) and ketamine^l (2mg/kg IV). The horse was intubated using a size 22 cuffed endotracheal tube^m. The horse was maintained on Isofluraneⁿ and oxygen using a semi closed circle system. Lactated ringers solution^o was administered during surgery at a rate of 10ml/kg/hr. The horse was positioned in dorsal recumbency. The left hindlimb was flexed such that the stifle and hock were at 90 degree angles (Figure 7). The leg was stabilized in this position with the use of ropes and a pulley system. The leg was surgically prepared and draped.

A lateral arthroscopic approach was used for examination of the medial femorotibial joint. The site for the arthroscopic portal was caudal to the lateral patellar ligament, cranial to the long digital extensor tendon and 2 cm proximal to the tibial spine. The arthroscopic cannula with conical obturator in place was directed medially and slightly caudad to penetrate the synovial membrane in the lateral aspect of the medial femorotibial joint. The obturator was removed and the arthroscope was inserted. The joint was distended with irrigating solution and an initial examination was performed. A characteristic dimple in the articular cartilage overlying the subchondral cystic lesion was visualized (Figure 8). An 18 gauge 3 1/2 inch spinal needle^p was then placed through the skin craniomedially, the location for the needle placement is best determined by trail placement (Figure 9). Optimum placement occurred with the needle perpendicular to the articular surface overlying the cyst (Figure 10). Once in position the needle was passed into the cyst until the bony margin of the cyst was contacted. 0.5ml triamcinolone acetonide^q was injected at each site. The needle was advanced

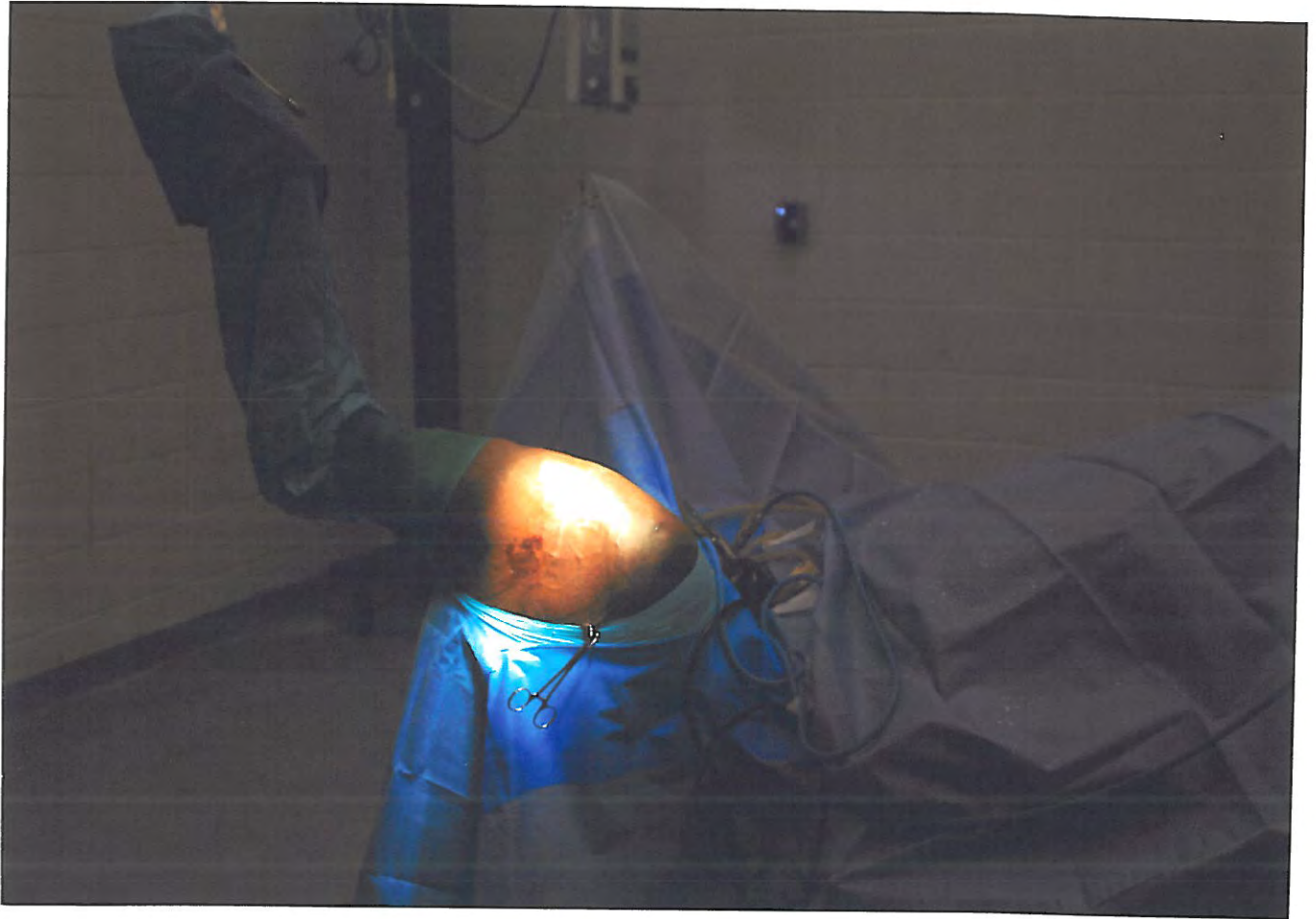


Figure 7: Intra-operative photograph depicting the limb position for surgery. Note the 90 degree angle of the stifle and hock, with the stifle placed in flexion.

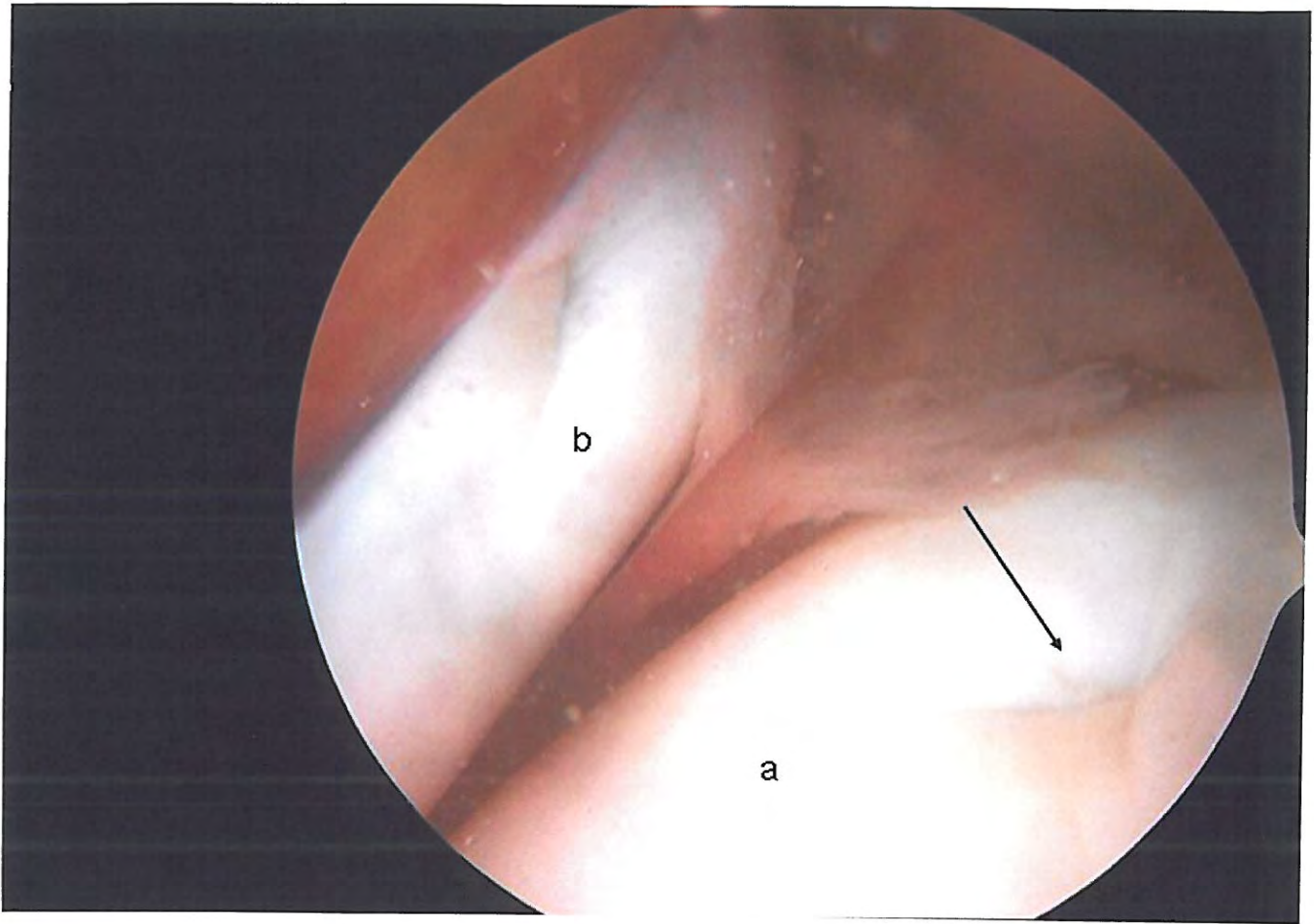


Figure 8: Arthroscopic view demonstrating a characteristic dimple in the articular cartilage overlying the subchondral cystic lesion on the medial femoral condyle (arrow).

a = Medial femoral condyle, b = Tibia

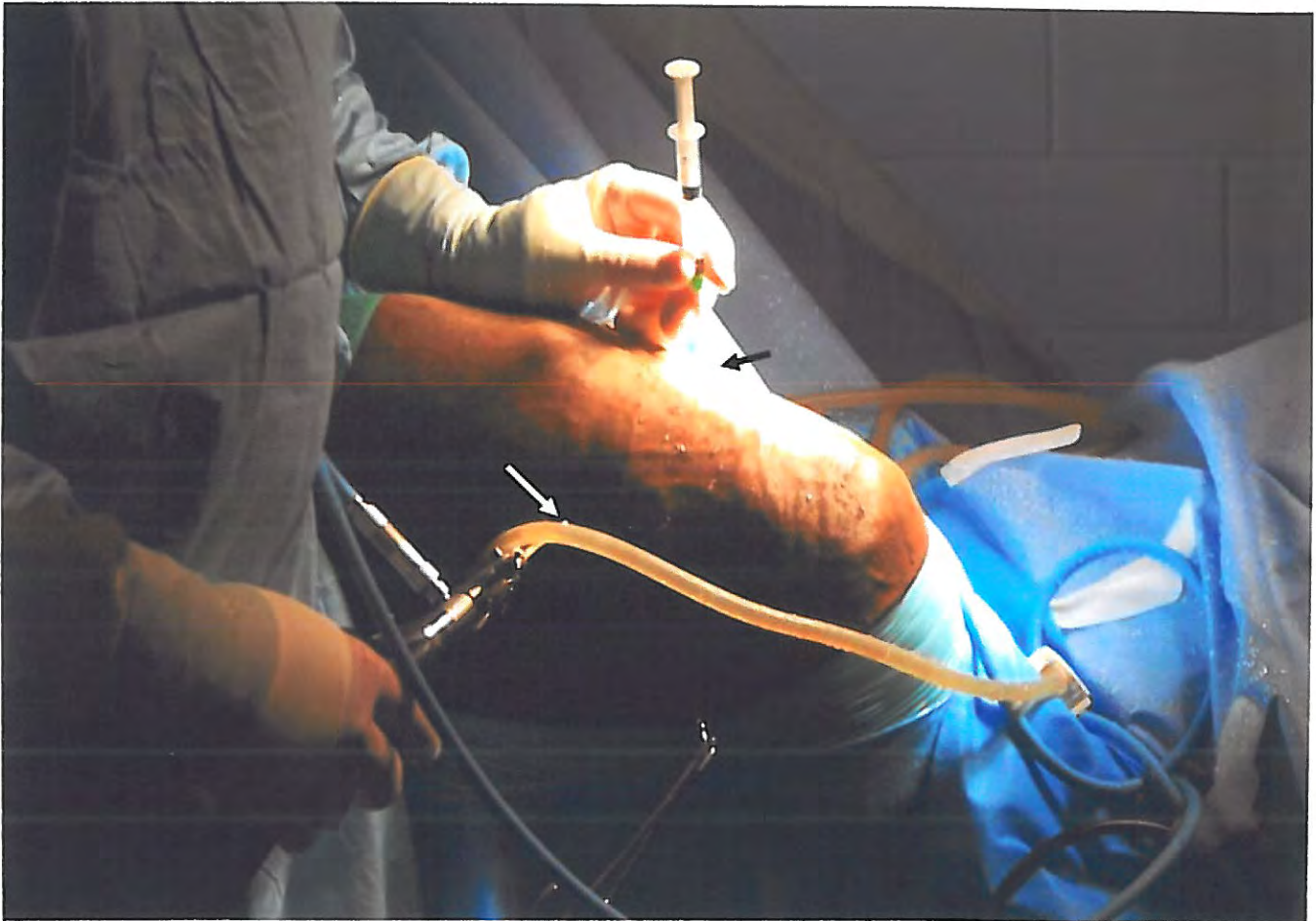


Figure 9: Intra-operative photograph depicting the arthroscope (white arrow) and needle (black arrow) in place for optimum visualization and treatment of the cyst with the stifle maintained in flexion.

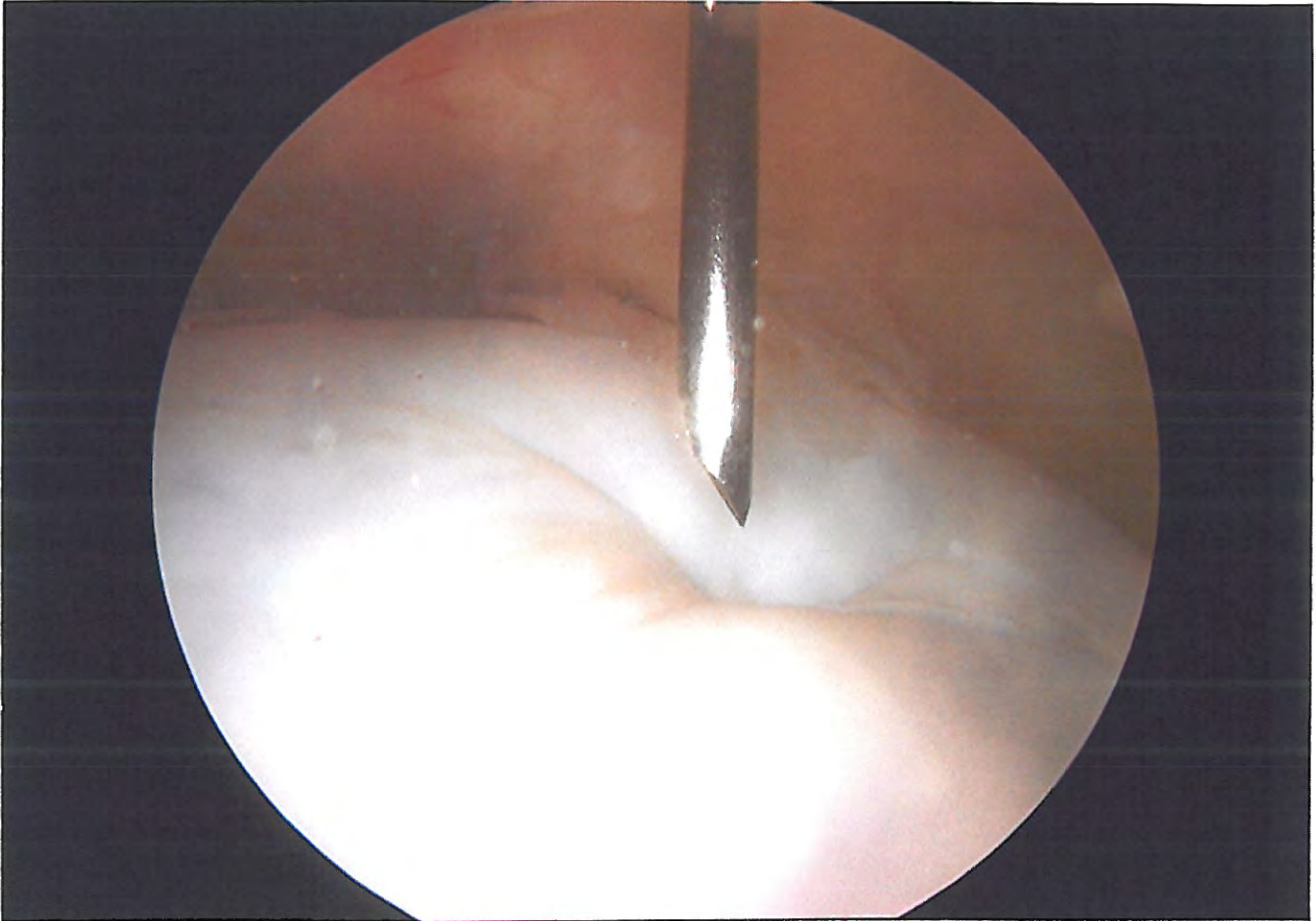


Figure 10: Arthroscopic view depicting ideal needle placement into the cyst cloaca. The needle was repositioned several times and triamcinolone acetonide injected into the cyst lining

into the cyst at 3-4 different angles through and adjacent to the cloaca and corticosteroid was injected at each location. The cartilage overlying the subchondral bone cyst was seen to bulge under pressure and with two of the injections no corticosteroid was seen to escape into the joint, indicating a successful injection. A total of 12 mg of triamcinalone acetonide^q was injected in to the cyst under arthroscopic guidance and 250 mg of sterile amikacin sulfate^r was injected into the joint prior to closure.

The skin portal was closed with 2-0 monofilament nylon^s in a simple interrupted pattern. The skin portals were subsequently covered with a non adherent dressing and adhesive tape.^t The anesthesia and recovery were smooth.

Post operatively, the horse was given phenylbutazone paste^u (2.2mg/kg PO q12hrs) for 7 days. The horse was placed on stall rest with hand walking only, for 30 days followed by turn out in a round pen for an additional 30 days. At 60 days post surgery the horse was reassessed for lameness and radiographs obtained as previously described. By this time the horse was sound, had no femoropatellar or medial femorotibial joint effusion, and follow up radiographs showed an increase in the opacity of the lesion, although they did not show a decrease in the size of the lesion.

The horse resumed training successfully at this time. At 90 days post injection the radiographic assessment demonstrated a decrease in the size of the lesion.

The horse has remained clinically sound for 7 months since the injection. Recheck physical and radiographic examinations have been carried out every 3 months.

It should be noted that a board certified surgeon was scrubbed in and assisted the author with the arthroscopic guided injection of the subchondral bone cyst.

DISCUSSION:

This case represents a yearling Thoroughbred colt diagnosed with a subchondral bone cyst in the medial femoral condyle of the left stifle that was treated successfully with arthroscopic injection of corticosteroids into the fibrous tissue of the lining of the cyst.

The history of this young horse presenting with an insidious onset lameness, that correlated with the commencement of training and that briefly responded to rest and anti-inflammatory medication was suggestive of a diagnosis of a subchondral bone cyst.

However, due to the lack of localizing signs, this horse presented a diagnostic challenge.

Following initial assessment and physical examination the possibility of formulating a meaningful differential list, based on a mild hindlimb lameness, was doubtful. The two important differentials at this time, based on age, stage of training and lack of clinical signs included subchondral bone cysts and osteochondrosis dissecans affecting some part of the limb.

Perineural nerve blocks were used in this case due to the lack of any physical findings along the limb. It was decided to block sequentially from distal to proximal in an attempt to objectively determine the area of pain. It is this author's preference to use perineural nerve blocks whenever possible, especially when dealing with an ambiguous lameness, due to the inconsistency of intra-articular anesthesia to abolish pain caused by many intra-articular problems. A decision was made to begin perineural anesthesia at the

level of a low plantar nerve block based on the lack of clinical findings in the distal limb. This horse was not sensitive to hoof testers, did not have a digital pulse and there was no heat, pain or effusion in this area. The horse's young age and the fact that this was a hindlimb lameness were also considerations when choosing where to begin the regional anesthesia. Reducing the number of injection sites was considered preferable. The decision to perform a high plantar block next was based on the need to desensitize all distal structures. There was no index of suspicion for this horse having a suspensory issue and, therefore, it was most important to rule out any problem at the subtarsal level rather than to try and specify a particular issue using individual blocks.

The decision to perform a tibial and peroneal nerve block to anesthetize the area distal to the stifle was made, and aided in establishing diagnosis of an upper limb lameness, by eliminating the region distal to the crus as the source of lameness. One concern when using this block after performing more distal analgesia is the subsequent inability to test if the nerve block has been successful by assessing skin sensation at the heel bulbs. Palpation of the high suspensory ligament is another acceptable method for determining success of the anesthesia. However, this horse had no reaction to palpation of the high suspensory ligament prior to the anesthesia, therefore, this manipulation was not thought useful in establishing the success of the block. The author, however, is experienced at performing this block and was confident as to needle placement and, therefore, the effectiveness of the anesthesia in this case.

An alternative approach, to alleviate this issue, would have been to anesthetize the tarsometatarsal and distal intertarsal joints directly to rule out the hock as the cause of

lameness, or to perform tibial and peroneal blocks independently, on a separate occasion.

Due to the unspecific nature of the lameness and clinical signs in this case, however, the author preferred to use perineural nerve blocks over intra-articular analgesia where possible. Thus by using the tibial and peroneal perineural block, not only would pain associated with the hock joint be eliminated, but also pain associated with subchondral trauma of the distal tibia and talus, distally located tibial stress fractures, the tarsal sheath, the distal aspect of the common calcaneal tendon, the calcaneal bursa and the plantar aspect of the hock. The negative response to this perineural analgesia in this case eliminated all these possibilities as the source of pain.

The time that elapsed between performing the tibial and peroneal block and performing the stifle anesthesia was approximately 2 hours. It has been suggested that at least 2 hours should elapse between these blocks to avoid misinterpretation, due to the potential for a delayed response to the tibial and peroneal block.⁴⁵ This is likely to be of greater significance where there appears to be a slight positive response to the tibial and peroneal block. There was no improvement over time in this case, thus the author was confident to proceed to stifle anesthesia. This author prefers to use 2% mepivacaine hydrochloride^a for all blocks as it is less irritating than lidocaine and has a shorter duration of action than bupivacaine. The author also likes to place alcohol and a bandage on the distal limb following the use of multiple blocks of the distal limb, to minimize swelling at the injection sites.

It is the author's protocol to block all three stifle joint compartments at one time, unless there are clinical signs or indications to block a particular compartment. It is the

author's experience that false negatives can be found by blocking the compartments separately; the response to anesthesia of a single compartment can often be less than the response to blocking all three. It is thought preferable to localize the problem to the stifle definitively by blocking all compartments and subsequently use imaging to identify the individual pathology.

The fact that there was no improvement in lameness until approximately an hour post anesthetic injection and then only a 50% improvement, is not surprising. In this author's experience, it is not uncommon for an intra articular block to take a prolonged period of time to become anesthetized. It is also documented that a 50% improvement in lameness following stifle joint anesthesia can be considered sufficient to justify further diagnostics of this area.⁴¹ This is reasonable considering subchondral bone pain is abolished inconsistently subsequent to intra-articular anesthesia and is, in fact, abolished much more consistently by perineural anesthesia. Subchondral bone receives its innervation from endosteal branches of peripheral nerves that enter the medullary cavity through a nutrient foramen and local anesthetic solutions may not penetrate the subchondral bone sufficiently to completely block these nerves. However, peripheral nerve analgesia is not possible at the level of the stifle.

The combination of perineural and intra-articular anesthesia allowed a differential list for a hindlimb lameness affecting the stifle to be established. The main differentials on this list included, a subchondral bone cyst, osteochondrosis dissecans (OCD) and ligamentous/meniscal injuries. The lack of femorotibial/femoropatellar joint effusion was not characteristic for an OCD and there was no history of trauma to indicate an initiating factor for a meniscal injury. However, neither condition could be ruled out conclusively

at this stage. A much less likely differential diagnosis was that of a joint infection, although the clinical signs and history were not supportive of this theory. At the time of intra-articular anesthesia, joint fluid was visualized and appeared to be of a normal color and consistency. Because the horse's history and clinical signs did not support a diagnosis of infection, cytology and culture and sensitivity were not performed. In the interests of completeness a fluid sample could have been obtained and analyzed at the time of arthrocentesis.

Radiographs, in this case, were diagnostic for the subchondral bone cyst.

Ultrasound examination was performed subsequent to radiographic identification of the subchondral bone cyst to aid prognostic and treatment making decisions. The combination of both imaging modalities is standard for this author. It is important to evaluate the stifle joint fully, assessing the stifle for concurrent degenerative joint disease and medial meniscal injuries so that the most appropriate form of treatment can be instituted. The main advantage of ultrasound is the possibility of evaluating the soft tissues of the stifle and the ability to detect early changes at the articular margins, not identified by radiography. Intra articular nerve anesthesia in this case defined the region of pain and radiographs subsequently identified the cause and therefore the diagnosis of the lameness. It should be noted, however, that if a subchondral bone cyst is identified radiographically but there is no corresponding positive perineural/intra-articular anesthesia, further diagnostic tests must be performed to identify other sources of pain and rule out the possibility of the bone cyst being an incidental finding. If perineural and intra-articular anesthesia have been negative along the entirety of the limb, ultrasonography of the pelvic area both transcutaneously and per rectum may be

an appropriate next step. In such cases where localization of the source of pain is not possible, scintigraphic examination may ultimately be required with the aim of demonstrating a problem area which can be further investigated. Due to this horse's young age and fractious nature it would also have been reasonable to have performed a scintigraphic examination as a first line to identify a general region of pain. However, the author felt this colt was amenable to regional anesthesia and is always concerned about the possibility of a scintigraphically silent osteochondral source of pain.

In this particular case, the cyst was diagnosed radiographically and the diagnosis confirmed using ultrasound. There was no need for the use of any advanced imaging techniques such as CT. However, it should be recognized that both radiography and ultrasonography can underestimate the pathology present which can lead to difficulties in establishing an accurate prognosis. CT could have been a useful additional modality in which to assess the joint, by permitting 3D imaging of the cyst. In this case, however, these modalities would not have affected the decision making process or treatment plan and would have needlessly exposed the patient to two general anesthetics.

This horse was a good candidate for any of the available treatment modalities, due to his young age and lack of other lesions detectable within the stifle joint.

Conservative treatment was dismissed by the author and the owner, due to the tendency for lameness to return once the period of rest is over and due to the fact that this horse was scheduled to attend a Thoroughbred sale as a 2 year old. Consequently, the amount of time off from training was a major factor in the decision making process.

The decision to treat this subchondral bone cyst with an arthroscopic guided intralesional corticosteroid injection instead of arthroscopic debridement, was made

based on personal experience with this technique and due to the slightly increased success rate and decreased recovery time of, 2-4 months as opposed to 6-8 months following cyst debridement.^{40,52} The decrease in complications observed with intralesional injection of corticosteroids compared to cyst debridement, such as enlargement of the cyst post operatively and the potential for subsequent medial meniscal injuries, influenced the decision making. The minimal disruption to the articular cartilage compared to that with surgical debridement was a major factor in choosing this treatment modality. One further advantage of the intralesional injection was the option to undertake further treatment (arthroscopic debridement) should the horse have not responded to this therapy in a favorable manner.

Three approaches have been used for diagnostic arthroscopy of the medial femorotibial joint: cranial, lateral and craniolateral. Despite the cranial approach allowing a more consistent examination of the intercondylar area, the lateral approach was chosen due to the ability of this approach to leave a clear area cranially for instrument placement which is desirable when working on the medial femoral condyle.⁴³

This author has performed the intralesional injection under ultrasound guidance when financial constraints dictate. The disadvantage of not being able to assess the joint with the arthroscope, however, and thus the potential for missing concurrent cartilage and soft tissue damage in the joint make this option less desirable than the arthroscopic guided injection.

The author is aware that, although initial published results for the technique of arthroscopic injection of corticosteroids into the cyst lining look favorable, the technique is still in its infancy and there are no long term follow up studies demonstrating long

term successes.⁷¹ It should also be noted that although initial published results suggest that the age of the horse is not an important prognostic factor, this finding should be interpreted with caution since only 25% of the horses in this published study were over 3 years old.⁵²

Despite the evidence demonstrating the lack of association between follow up radiographic examination and success, radiographs were taken 60 days post surgery in this case. The main reason was to observe for any signs of osteoarthritis and to ensure that the cyst was not enlarging as this would have affected both time off and prognosis. A follow up lameness examination was performed to assess the soundness of the horse at the trot. The horse was monitored once he resumed training and for seven months afterwards.

The rationale behind returning the horse to light training at 60 days post surgery is based on data published by Wallis, et al (2008). Their results suggest that if the horse is sound at 30 or 60 days, returning them to light exercise at this time is advantageous.⁵² This horse was sound at the trot in hand at 60 days post surgery, had no femoropatellar or medial femorotibial effusion and no deteriorating radiographic signs and therefore was allowed to begin light training. Had this horse become lame following the commencement of training he would have been removed from training and re evaluated.

SUMMARY:

A 1 year old Thoroughbred colt was diagnosed with a subchondral bone cyst in the medial femoral condyle of the left stifle. The horse presented with a 3/5 left hind limb lameness (AAEP 1/5 scale) after commencement of galloping exercise. There were no

other clinical signs. Perineural and intra-articular anesthesia identified the stifle as the source of lameness. The cause of the lameness was definitively diagnosed radiographically. Subsequent ultrasound examination allowed evaluation of the soft tissue and cartilage within the joint and enabled a treatment plan and prognosis to be established.

The horse was treated by intralesional corticosteroid injection performed by arthroscopic guidance under general anesthetic. Post operative care consisted of confinement to a box stall with hand walking for 30 days, followed by 30 days turn out in a round pen. The colt successfully reentered race training and has remained sound for the last 8 months.

Endnotes:

- a. 2% Mepivacaine hydrochloride, Carbocaine, Pfizer, New York, NY.
- b. Butorphanol Tartate, Torbugesic, Fort Dodge Animal Health, Iowa.
- c. Detomidine Hydrochloride, Dormosedan, Pfizer Animal Health, NY
- d. Digital x ray machine, Eklin Mark III, Sound-Eklin, Carlsbad, Ca
- e. Digital ultrasound machine, Logic E, GE Healthcare, Wauwatosa, WI
- f. 14 gauge 5 1/2 inch Abbocath-T, Hospira, Inc, Lake Forest, IL.
- g. Phenylbutazone injection 20%, Phoenix pharmaceutical, Inc, St Joseph, MO.
- h. Penicillin G Potassium, Pfizerpen, Pfizer, New York, NY.
- i. Gentamicin sulfate solution, Sparhawk Laboratories Inc, KY
- j. Xylazine, Tranqved. Vedco, St Joseph, MO
- k. Diazepam, Hospira, Inc, Lake Forest, IL.
- l. Ketamine Hydrochloride, Ketaset, Fort Dodge Animal Health, Iowa.
- m. Endotracheal tube-22m cuffed, Jorgensen Laboratories, Loveland, CO.
- n. Isoflurane, Abbot Animal Health, Chicago, IL
- o. Lactated Ringers Solution, Baxter Healthcare Corporation, Deerfield, Illinois.
- p. 18 gauge 3 1/2 inch spinal needles, Kendall, Tyco health care group, Mansfield, Ma.
- q. Triamcinalone acetone suspension, Vetalog, Fort Dodge, Iowa.
- r. Amikacin Sulfate Inj (500mg/ml), TEVA Parental medications, Irvine, Ca.
- s. Monofilament Nylon, Ethilon Suture, Ethicon, Johnson and Johnson, New Jersey.
- t. Adhesive tape, Microfoam 3 inch, 3M Health Care, St Paul, MN
- u. Phenylbutazone paste, Vedco, St Joseph, MO

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