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The stifle is an important source of hindlimb pain causing lameness but lameness of the distal limb from the tarsus to the foot is much more common. Many clinicians will default to thinking pain causing lameness is originating from either the tarsus or the stifle and overlook other important regions such as the metatarsal region or metatarsophalangeal joint (MTPJ); importantly, diagnostic analgesia must be used whenever possible to sort out the authentic source of pain in horses with hindlimb lameness. In a yearling with hindlimb lameness and severe effusion of the femoropatellar joint (FPJ) in it not necessary to perform diagnostic analgesia. Radiography reveals the presence of osteochondritis dissecans (OCD) and the diagnosis is confirmed. However, in a mature, dressage horse with hindlimb lameness, a mildly positive response to upper limb flexion, and mild effusion of the FPJ diagnostic analgesia must be used to confirm pain is originating from the stifle since other sources of pain such as proximal suspensory desmitis, distal hock joint pain and pain arising from the MT-PJ are more common.

**CLINICAL CHARACTERISTICS OF HORSES WITH STIFLE PAIN**

There are no pathognomonic clinical signs defining the way horses move with stifle region pain. All horses are prone to develop stifle injuries, but horses negotiating fences may be predisposed. Horses moving fast over fences, such as timber horses and event horses, are prone to hit the stifle region and can sustain fractures of the patella and cranial tibial tuberosity. Horses working on uneven or slippery surfaces may be predisposed. In management of horses with stifle pain it is important to prevent slipping and quick sudden turns, movements that accompany unlimited turn out; therefore, turn out exercise is restricted. Palpation may reveal effusion of one or more joint compartments. Mild effusion of the FPJ is common and there are many false positives associated with this clinical sign. However, effusion of the medial femorotibial joint (MFTJ) is an important clinical sign; this joint is best palpated with the horse standing square or with the affected limb slightly cranial to the contralateral hindlimb. In normal horses there is a depression between the medial patellar ligament and the medial collateral ligament, but in horses with effusion there is a "bulge" in this location. In horses with long-standing osteoarthritis (OA) of the MFTJ there is often marked thickening of the joint capsule, an important clinical sign. The LFT joint capsule is much more difficult to palpate, and while, uncommon-to-rare, effusion of this joint capsule, palpated between the lateral patellar and lateral collateral ligaments is as close to a pathognomonic sign as it gets. Sometimes effusion of this joint can be seen if the horse is carefully examined visually. Horses often resent palpation of the stifle and discomfort during palpation should not be mistaken for authentic stifle region pain. However, sometimes careful deep palpation with the limb in a flexed position may elicit pain not detected when compared to the contralateral limb. Horses with fractures may have marked
soft tissue pain and severe effusion. Horses get enormous swellings with stifle hematomas. Horses with substantial injuries such as fractures or disruption of the cruciate ligaments and medial collateral ligament will be severely, often non-weight-bearing lame and have instability of the stifle joint; instability can be hard to detect since horses are so painful. Careful palpation may reveal lateral dislocation/luxation of the patella. I often see coexistent MTPJ (fetlock) and stifle pain, particularly in racehorses. OA of the MFTJ and mal-adaptive bone remodelling of the distal aspect of the third metatarsal bone is quite common in TB and STB racehorses. There appears to be a relationship between these two regions, an example of intra-limb compensatory or coexistent lameness. Some horses, particularly young horses with stifle instability will knuckle, likely making them prone to development of MTPJ pain. Never forget the MTPJ in horses with stifle pain (Loren Evans, personal communication, 1983)!

How do horses move with stifle region pain?! Shortened cranial phase of the stride is a hallmark of stifle lameness, but this clinical sign is not pathognomonic and present in many horses with hindlimb lameness. One of the best descriptions I have heard of how horses move while trotting with stifle pain, is “the limb just can’t quite keep up” (Dr. Kent Carter, Texas A&M University). In horses with severe OA, in young horses with OCD or other forms of osteochondrosis there can be marked shortening bilaterally, causing a gait described as “bunny hopping”. Only conditions of the tuber ischium or caudal thigh muscles cause more shortening of the cranial phase of the stride. Horse may appear to only partially load the affected hindlimb and are quick to unload the limb while trotting. From the side it appears the stifle region is more prominent than normal with the limb appearing to be held in mild external rotation. From behind the horse may swing, or abduct, the limb when pushing off during early protraction (the limb moves away from midline and then the foot is placed on the ground in the expected location, or just in front of the contralateral foot), or carry the affected limb straight; a “stabby” hindlimb gait is seldom seen but differentiation from other sources of hindlimb pain merely on the basis of limb flight is fraught with error. There are variable degrees of pain from subtle nearly non-detectable to severe grade 4-5 (on a scale of 0 being sound and 5 being non-weight-bearing). Lameness is more obvious while turning with the affected limb on the inside of a circle. There is often a positive response to upper limb flexion (the so-called spavin test) but horses with other sources of hindlimb pain respond positively as well. A stifle flexion test, hard flexion of the stifle without concomitant hard flexion of the hock, can be performed but this test is somewhat dangerous and there are many false positive results. I use other forms of manipulation such as forcing the proximal aspect of the tibial caudally or forcing the patella proximally, both done with the horse standing bearing weight and followed by trotting. A horse may manifest a marked positive response to this test before moving or, lameness grade may worsen considerably. Intra-articular analgesia is essential for diagnosis in mature horses, regardless of the presence of other clinical signs or imaging abnormalities. There is variable communication between the 3 joint compartments of the equine stifle joint and local anesthetic solution can either flow directly through communications or diffuse through synovial septae, but knowledge of the communications is essential to understanding results of diagnostic analgesia. Physical communication between the joint compartments was studied in cadaver specimens injected with latex; when the FPJ was injected there was communication with the MFTJ (60%) and with the LFTJ (3%). When the MFTJ was injected communication with the FPJ (80%) and LFTJ (3%) occurred. When the LFTJ was injected communication with the FPJ occurred in only 1 (3%) specimen; in no specimens did the MFTJ and LFTJ communicate. Functional communication between the FPJ and femorotibial joints was found in 74% of horses but authors recommended each compartment be blocked independently. Mepivacaine diffused between
synovial compartments of the stifle joint in 85-100% of specimens, often in concentrations high enough to elicit analgesia; authors concluded that diffusion of local anesthetic solution occurred more frequently than did latex or dye, and separate compartment injections were not as specific as once thought.4 What can be learned from these studies? To be safe all 3 compartments of the stifle joint should be blocked independently. The specificity of intra-articular blocks must be questioned. The LFTJ, anatomically, is closest to being a solitary joint cavity but could be blocked when the FPJ and MFTJ are blocked. In practice, I consider the LFTJ an independent synovial structure and have seen numerous horses in which analgesia of the other compartments failed to abolish pain originating from this joint; the LFTJ had to be blocked in order for horses to show improvement. So, to be safe all 3 compartments should be blocked; this can be done all at once or sequentially, but if done sequentially the results make lack specificity. I use 30 ml of mepivacaine and block independently all 3 compartments simultaneously in most horses. Importantly, the stifle joint is large and complex so horses should be given a minimum of 20-30 minutes before re-evaluated after the block. If improvement is noted, an additional 20-30 minutes is given before any other diagnostic analgesia procedure is performed. The clinician should not expect 100% improvement unless the horse is lame in another limb; 70-80% improvement is conclusive that the primary source of pain causing lameness is the stifle joint.

**DIAGNOSTIC IMAGING**

Radiography is the first step in diagnostic imaging of horses with confirmed or suspected stifle region pain. Digital radiography has greatly improved image quality but an occasional faint radiopacity in the intercondylar region, an artefact of digital imaging, should not be mistaken for fragmentation. Lateromedial, caudocranial, caudolateral-cranio medial oblique, and tangential (skyline) images should be obtained. Occasionally, cranialateral-caudomedial oblique images may be needed in horses with unusual fragmentation. Tangential images are not essential but or quite useful in the diagnosis of patellar fragmentation and to assess patellar position. Well-positioned and well-exposed caudocranial images are essential for evaluating the femorotibial joint space, narrowing of which is the earliest sign of OA, but must be repeated if there is any overlap or horses are mal-positioned. In young-to-middle aged racehorses, other signs of OA such as osteophyte formation will be subtle, but authentic narrowing of the MFT joint space is indicative of widespread cartilage damage. Ultrasonographic examination is quite useful in evaluating soft tissues of the stifle region and can be used to evaluate joint surface congruity. Examination of patellar ligaments, menisci, and collateral ligaments, and observing effusion, synovial proliferation, osteophytes, incongruity of the femoral condyle are all benefits of ultrasonography. There are false positive results, however, particularly when evaluating suspected meniscal injury. The cruciate ligaments cannot be examined completely, although disease of these ligaments is extremely rare. Magnetic resonance imaging and computed tomography, if available, can be hugely beneficial and will likely become more important in the near future. Scintigraphic examination is important but has low sensitivity. In fact, I suspect 2 sources of pain in horses in which bone scan images are negative or equivocal, the stifle and proximal suspensory ligament. The caudal scintigraphic image is essential to evaluate the MFTJ for the presence of increased radiopharmaceutical uptake (IRU) in subchondral bone, which occurs in horses with OA and subchondral bone cysts. Even subtle areas of IRU of the distal, medial femoral condyle should be investigated radiologically. Arthroscopic examination gives direct information of the integrity of cartilage and soft tissues and I believe has been “under sold” in the past. In horses with obvious radiologically or ultrasonographically apparent lesions arthroscopy can be therapeutic but the diagnostic value should not be underappreciated in horses in which pain causing lameness...
can be abolished, but in which few, if any, imaging abnormalities are present. In these horses, often early and important cartilage damage involving the medial femoral condyle suggestive of OA is often found.

**MANAGEMENT OF HORSES WITH STIFLE PAIN AND INJURIES – EMPHASIS ON SURGERY**

The FPJ and the cranial compartments of the MFTJ and LFTJ can be evaluated separately, if desired; the horse is most commonly positioned in dorsal recumbency. I routinely use fluid irrigation (gas insufflation can be used) and a single portal approach for routine evaluation of the stifle joint, with an initial arthroscopic portal between the middle and medial patellar ligament. Depending on the site of the lesion or if comprehensive examination is required, the septae between the FPJ and each the MFTJ and LFTJ are removed easily using a synovial resector (can be done manually) creating a single joint compartment. This approach is more versatile and consistent than using individual compartment approaches. I struggle in evaluating the caudal compartments of the FTJs, approaches to which have been recently refined. A cranial intercondylar approach to the caudal pouch of the MFTJ was described and can be performed without a switching stick using the single cranial approach and septectomy. Loose fragments in the FPJ most often accumulate in the extensive suprapatellar pouch and removal sometimes requires a long lavage cannula, long Ferris-Smith rongeurs, or a separate more proximally positioned instrument portal. Intra-operative radiographs are used if size and number of fragments retrieved differs from pre-operative assessment. A synovial resector facilitates removal of embedded fragments at the insertions of the cranial meniscotibial ligaments and various intra-articular blades can be useful to remove meniscal tears. I use microfracture sparingly since aggressive debridement and puncture through calcified cartilage into the subchondral bone can cause a subchondral cyst to occur when one did not exist pre-operatively and the technique was not associated with success. Impressive results were reported using mosaic arthroplasty and autologous fibrin laded with growth factors and chondrocytes.

Specific lesions – OCD lesions in the FPJ are debrided and loose fragments removed; excessive debridement and abrasion arthroplasty are avoided, if possible. Prognosis is poor with extensive lesions, of the lateral trochlear ridge of the femur (LTR), coexistent patellar chondromalacia and with intermittent patellar subluxation. Young horses with extensive OCD lesions of the LTR should be given 3-6 months rest to allow potential reattachment. Further debridement of the LTR in these severely affected horses will make them more prone to patellar luxation and should be avoided. Some consideration can be given to repair of these lesions using polydioxanone pins. In general, prognosis is quite good in young horses with OCD of the FPJ. If lesions are discovered in older horses conservative management including injections into the FPJ can be given. In some horses small lesions are found incidentally. Surgery is indicated given a willing owner since even small pieces, if they break off, can cause pronounced lameness and effusion, and the existence of lesions may complicate a future sale. Patellar fracture fragments, most common in horses negotiating fences (medial and proximal aspects) are removed; occasionally additional instrument portals are needed. Distal patellar fragments occur after medial patellar desmotomy and are easily removed using standard FPJ portals. Occasionally OA of the FPJ is seen in middle-aged and older horses and prognosis is poor. The relationship between the existence of OCD and the subsequent development of OA is compelling, but often not substantiated. For a long-term solution I still prefer to debride osseous cyst-like lesions of the medial femoral condyle. Reported success in horses 0-3 years of age (64%) was similar to that reported for injecting corticosteroids directly into the cyst lining (67%). In racehorses choice of technique is often directed by owner/trainer influence – if the horse must train and race soon after the procedure I use arthroscopi-
Pharmacologically assisted injection of corticosteroids (methylprednisolone acetate). Caution is used in operating older horses with subchondral lucent defects since they usually have substantial OA and prognosis is poor (only 35% of horses > 3 years of age returned to soundness). OA of the MFTJ (less commonly the LFTJ is a common problem causing lameness in older non-racehorses and racehorses. Horses with subchondral bone cysts can be managed conservatively and given rest and intra-articular injections; however, prognosis appears better with surgical management.

Arthroscopic evaluation in racehorses with severe OA is diagnostic but palliative and seldom curative; if racehorses have severe lameness, do not respond to intra-articular corticosteroids and have narrowing of the MFTJ space I discourage arthroscopic examination. Non-racehorses most commonly develop OA, with varying degrees of articular cartilage damage (early - soft, thin cartilage on the medial femoral condyle; later – full thickness cartilage loss and subchondral bone erosion, osteophyte formation). Coexistent meniscal fraying and tearing is seen in horses with OA, but I see very few horses with meniscal tears as the primary surgical lesion. I marvel at the extensive experience of Walmsley et al. Meniscal injuries most commonly occur in horses with other deterioration of the MFTJ or LFTJ and seem to be associated with rather than the cause OA or lameness. There are rare exceptions, however. Prognosis in non-racehorses is adversely affected by age, severity of lameness, the presence of a large meniscal tear, and the severity of radiological changes.

Few horses have solely a soft tissue lesion diagnosed at arthroscopy; lameness is usually caused by ongoing OA. Rarely, cranial meniscal ligament tears can cause substantial lameness.

Fractures involving the stifle occur most commonly in horses negotiating fences and usually involve the patella (see above) and cranial tibial tuberosity. Horses with non-displaced or mildly displaced tibial tuberosity fractures can, and should, be managed conservatively. These fractures are usually non-articular. Two of 17 horses with this type of fracture were retired for reasons unrelated to fracture and 12 of the remaining 15 (80%) returned to the previous level of competition. Horses with large, long and articular fractures are likely surgical candidates but tension band techniques such as application of a bone plate should be used. Simple screw fixation can fail. Given the fact that implants are in the tibial diaphysis special consideration for recovery from general anesthesia should be given; the tibial can fail catastrophically. Horses occasionally develop other unusual fractures or fragmentation, the origin of which can remain a mystery. Rarely a mid-body fracture of the patella is diagnosed and surgical management with screws and wires can be performed.

CONSERVATIVE MANAGEMENT, REST, INTRA-ARTICULAR INJECTIONS

Whether or not surgery is performed horses with stifle pain need rest to allow healing of articular surfaces, subchondral bone, soft tissues, or in most horses a combination of these tissues. Four to 6 months of rest is generally recommended particularly in older horses with substantial pain and in which surgery was performed. Many horses will require longer rest periods to become completely sound and are at risk for recurrent pain. Recurrence is a common problem in horses with stifle pain. I usually give a minimum of 4 weeks stall rest, then 4 weeks stall rest with hand walking, then 4 weeks walking with a rider up (in a jog cart if a STB racehorse, or a mechanical walker) and then 4 weeks walking and light trotting, if sound at this stage. I do not prescribe turn out exercise in the first 6 months after injury or surgery. Uneven surfaces, slipping, quick turning and stopping all predispose horses to recurrent injury and may be the reason stifle pain developed initially. How do you shoe a horse with stifle pain? There are no set rules, but some advocate raising the heels to allow easy breakover, a practice that makes sense. Since slipping is an important force predisposing to injury shoe additives (heel, toe caulks, borium) make some
sense to help limit slipping. Various intra-articular injections can be used and are commonly given to racehorses with OA. I avoid the use of methylprednisolone acetate if possible, not because of a direct deleterious effect, but because the drug is quite effective at reducing inflammation and may allow the horse with OA to overachieve, risking further development of OA. Hyaluronan (double dose) and triamcinolone acetonide are injected and are efficacious in the short-term but additional injections will be necessary and response in some horses is poor. Other injections such as IRAP and PRP are currently being given but efficacy is unknown.

REFERENCES