Use of sodium monooiodoacetate to fuse the distal hock joints in horses

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SUMMARY: Intra-articular injection of sodium monooiodoacetate (MIA) was investigated as an agent for chemical arthrodesis of the distal hock joints in the horse. Five horses diagnosed with either spavin (three horses), a small tarsal bone fracture or a failed surgical arthrodesis, had 150 mg of MIA injected into the tarsometatarsal (TMT) joint of the affected hock(s). Eight joints were treated in the five horses. Follow-up evaluation by clinical and radiological examination took place over 9 to 14 months. Two of the five horses were sound at the conclusion of the study and one horse, although lame after flexion, was considered by the owner to have been treated successfully. One of eight TMT joints showed complete radiographic fusion. Complications after treatment included pain, chronic lameness and swelling. It was concluded that chemical arthrodesis using this technique can not be recommended as being a superior treatment as compared with surgical arthrodesis at this time but is deserving of further clinical evaluation.

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Introduction
Osteoarthritis or bone spavin of the equine hock is a frequent cause of hind limb lameness (McIlwraith and Turner 1987; Edwards 1982; Wynn-Jones and May 1986). The tarsometatarsal (TMT) and distal intertarsal (DIT) joints (the latter currently known as the centrodistal joint, Schaller 1992) are most commonly affected, but occasionally the disease extends into the proximal intertarsal (PIT) joint. The PIT joint is currently referred to as the talocalcaneal central and calcaneal quadrilateral joints (Schaller 1992), but as the former term is more readily understood and is in widespread use it shall be referred to as such in this paper. Figure 1 illustrates the joint spaces in the equine hock joint. Bone spavin is a degenerative and progressive disease that can ultimately result in ankylosis of the affected joint(s). Bone ankylosis brings joint immobility with an associated reduction in pain and subsequent soundness. Unfortunately, the natural ankylosing process is slow and unreliable often resulting in only partial fusion of the joint(s) (Edwards 1982). This has prompted the development of arthrodesis techniques in an attempt to enhance the chances of a successful ankylosis.

Surgical techniques of arthrodesis involve removing varying amounts of the articular cartilage using a drill bit (Adams 1970; Edwards 1982, Wynn-Jones and May 1986; McIlwraith and Turner 1987) and/or internal fixation (Mackay and Liddell 1972; Wynn-Jones and May 1986; Archer et al 1989). Surgical arthrodesis has an overall success rate of about 80% (Auer 1992). The current recommended surgical arthrodesis technique, which involves placement of three drill holes across each joint, is associated with minimal post-operative complications and pain (McIlwraith and Turner 1987). Nevertheless, surgical arthrodesis is a major procedure that requires general anaesthesia, is relatively expensive and has a convalescence period of up to 12 months (McIlwraith and Turner 1987).

More recently, chemical arthrodesis of the distal hock joints in the horse using intra-articular sodium monooiodoacetate (MIA) has been attempted but, to date, this has only been used experimentally (Bohanon et al 1991). The technique has been suggested as a simple and cheap alternative to surgical arthrodesis.

The purpose of this study was to investigate the clinical application of MIA as an agent for arthrodesis of the distal hock joints in the horse. Five horses suffering from spavin, tarsal bone fracture or failed surgical arthrodesis were given a single injection of 150 mg of MIA into the TMT joint. They were assessed clinically and radiographically over a period of 9 to 14 months for signs of ankylosis. A successful result was achieved in three of the five horses, although one of the horses was still lame after a hock flexion test.

Materials and Methods
Criteria for the selection of cases for this study were diagnosis of severe advanced bone spavin, fracture of a small tarsal bone(s) or failure of surgical arthrodesis. The diagnosis was based on clinical appraisal of lameness, response to hock flexion test, response to intra-articular analgesia or cortisone administration and radiographic changes. The joint disease could involve either or both the TMT or DIT joints but not the PIT or talocalcaneal (TC) joints. Chemical arthrodesis was offered as a treatment option to the client when surgical arthrodesis could not be financially justified.

A single injection of 150 mg (50 mg/mL) MIA was made into the TMT joint of each affected hock. After sterile skin preparation, a 21 G 26 mm needle was inserted between the fourth metatarsal bone and the fourth tarsal bone on the plantarolateral aspect of the hock as described by Sack and Orsini (1981). Before injection, synovial
fluid was retrieved to confirm the needle's position within the joint. If it was thought that the temperament of the horse would interfere with the administration of the intra-articular injection then short-term general anaesthesia was given using xylazine (1.1 mg/kg IV) followed by ketamine (2.2 mg/kg IV).

Management after treatment involved box rest for 4 weeks. After this time the clients were advised to begin a controlled graded exercise regimen starting with 4 weeks of riding at the walk. Phenylbutazone (2.2-4.4 mg/kg once or twice daily) was used to control exercise regimen starting with 4 weeks of riding at the walk. Phenylbutazone therapy was used to control pain for the first 2 to 3 weeks after injection. Phenylbutazone therapy was continued if it was deemed necessary to enable the horse to exercise.

Follow-up assessment was performed by the authors and referring veterinarians. This involved clinical evaluation of lameness, swelling, response to hock flexion test and radiography.

Lameness was graded on a scale of 0 to 4 where 0 = sound, 1 = lameness just detectable, 2 = lameness easily detectable, 3 = lameness pronounced but still weight-bearing, and 4 = non-weight-bearing lameness. Swelling was graded on a scale of 0 to 3 where 0 = no swelling, 1 = mild swelling limited to the injection site and joint capsule, 2 = moderate swelling involving periarticular tissue and/or proximal tarsal sheath, and 3 = severe swelling involving the whole joint and some or all of the distal limb.

Radiographs were taken at various times after injection and at the conclusion of the study. Four standard radiographic views were used; dorso-plantar, dorso 60° lateral-plantar medial oblique, dorso 60° medial-plantar lateral oblique and latero-medial.

Criteria for successful response to treatment were soundness, return of the horse to a level of performance equal to or better than at presentation, and client satisfaction. Only horses satisfying all three criteria were deemed to have been treated successfully.

Results

Five horses were treated with chemical arthrodesis and a total of eight TMT joints were injected. The case details are listed in Table 1.

Chemical arthrodesis using MIA was successful in two of the five cases in this study (Table 2) and the owner of one other horse was satisfied with the results as the horse was performing well, although it was still slightly lame after a hock flexion test.

Cases 2 and 5 were treated standing with a nose twitch applied for restraint. Cases 3, 4 and 6 were injected under general anaesthesia. Intra-articular injection of the TMT joints of cases 1 and 2 was difficult to perform. In all horses, except case 5, deliverance of the full 3 mL of MIA required administration against joint resistance.

Pain after treatment was evident in all horses for up to 2 weeks. Pain was shown by increased lameness in the treated limb although the pain was easily controlled with phenylbutazone. Phenylbutazone therapy needed to be continued for 8 to 12 weeks in cases 1 and 2 due to chronic lameness. These cases remained lame at long-term follow-up. Case 3 was in full work 10 months after treatment; it was sound at the trot and the owner was very satisfied with the treatment. However, we felt that the horse could not be considered a success due to mild lameness after a hock flexion test. Soundness was achieved 7 to 8 months after treatment for cases 4 and 5.

Grade 3 swelling was seen in all the treated hocks immediately after injection. The swelling showed gradual reduction over a period of several months, but cases 1 and 2 still had hock swelling at the end of the study. Palpation of the swollen areas did not elicit pain at any stage of the study.

There was a general lack of owner compliance with regard to exercise after treatment. Case 2 was the only horse to be exercised according to the prescribed regimen. Case 1 was placed in a paddock after treatment and did not receive any exercise until 6 months later. Case 3 and 5 began walking and lunging exercise at three months after injection and this was gradually increased to full work at the conclusion of this study. The exercise regimen of case 4 was unknown.

Radiography of the treated joints revealed that only one joint, case 5 TMT joint, showed evidence of total bony fusion. The seven other partially fused joints showed areas of radiographically normal looking joint surface. In all horses the TMT joint showed more fusion than the DIT joint of the same tarsus. Areas of fusion in the partially ankylosed joints were on the plantarolateral aspect of the joint with the dorsal joint margins still distinct. There was no obvious difference in amount of TMT joint fusion between cases 1 to 4. No radiographic evidence of osteoarthritis was evident elsewhere in the tarsus.

Discussion

MIA is reported to produce a reliable, diffuse and severe insult to the articular cartilage after intra-articular injection (Kalbhen 1980; Williams and Brandt 1984; Yovich et al 1987; Gustafson et al 1992). MIA is a potent inhibitor of glycolysis and has been used extensively to produce experimental models of osteoarthritis (Kalbhen 1980; Williams and Brandt 1984; Yovich et al 1987; Gustafson et al 1992). When administered intra-articularly, MIA will reliably produce widespread destruction of the glycolysis-dependent chondrocytes and begin a self-perpetuating osteoarthritic process in the joint (Kalbhen 1980), which will ultimately result in ankylosis (Bohanon et al 1991). A further possible mechanism by which MIA may enhance arthrodesis is that it results in reduced cartilage chondrone formation (Bohanon et al 1991; Gustafson et al 1992). Chondrones are thought to delay the ankylosing process by forming persistent cartilage bridges between the joint surfaces (Auer 1992). Therefore reduced chondrone formation may lead to a potentially more effective arthrodesis.

The only published report of MIA-induced arthrodesis in horses has been by Bohanon et al (1991), and this work was done in horses free of lameness. These authors found that a series of three injections of 100 mg of MIA made 3 weeks apart into both the TMT and DIT joints of horses would produce an average of 70.5% joint fusion in 6 months with unfused areas of the joints showing potential for fusion. Lack of complete bony fusion was attributed to incomplete distribution of MIA across the joint surface or inconsistent load distribution across the distal tarsal joints. The radiographically observed partial fusion in our study was thought to be due to a
combination of poor MIA distribution through the joint space, reduced MIA dosage and lack of exercise after treatment. Poor diffusion of the MIA throughout the joint might have been a result of chronic degeneration of the TMT joint(s) as all the horses (except case 5) had disturbance of the joint space, and this might have inhibited the even distribution of MIA. The joint with the least joint surface disturbance (case 5) was the only case that showed complete TMT joint fusion. The bony fusion seen in the partially fused joints was mainly in the plantarolateral area of the joint. This would suggest that the MIA was having its greatest effect close to the injection site.

The difficulty with intra-articular injection experienced with some of the cases might have resulted in less than the entire dose of MIA reaching the articular surface. The severity of the osteoarthritic change seen after MIA administration is dose-dependent (Gustafson et al 1992), therefore reduced MIA dosage would lead to less articular cartilage damage, which may affect the joints’ potential for arthrodesis.

Exercise has been emphasised as an important part of management after treatment of all surgical and chemical arthrodesis cases (Adams 1970; McIlraith and Turner 1987; Bohanon et al 1991). Intra-articular MIA produces more diffuse and severe osteoarthritic changes in exercised guinea pigs compared with those with immobolised MIA injected joints (Williams and Brandt 1984). Exercise is thought to perpetuate osteoarthritis by increasing the mechanical forces acting on the chemically damaged cartilage. It is therefore indicated to enhance the rate of bony fusion. The overall lack of compliance with the prescribed exercise regimen seen in our study was thought to contribute significantly to the lack of total fusion seen in some of our cases.

The TMT joint was used as the only site of MIA injection for three reasons. Firstly, each horse showed clinical improvement after injection of local anaesthetic or corticosteroids into this joint only. Therefore it was considered that an approximately equivalent volume of MIA would have a similar distribution and effect. The slightly higher volume of local anaesthetic used in some cases may have resulted in more extensive diffusion through the joint than occurred with 3 mL of MIA. This may have contributed to the poor results achieved after subsequent injection of MIA in some cases. Additionally, it was a concern that the difficult medial approach for access to the PIT joint would increase the possibility of peri-articular leakage of the tissue-irritant MIA, which may have resulted in further complications. Finally, it was thought that a single injection into the most distal joint would reduce the chances of MIA diffusing into the more proximal PIT and TC joints. In our study we depended on communication between the TMT and DIT joint to allow passage of the MIA into the latter joint. Initial reports suggested the two distal tarsal joints communicated in most cases (Gabel 1979). Joint communication was thought to increase if the injection was made under pressure (Sack and Orsini 1981). Communication between the TMT and DIT joints ranging from 8.3% to 35% has been reported (Sack and Orsini 1981; Kraus-Hansen et al 1992; Bell et al 1993; Dyson and Romero 1993). Furthermore, injection pressure is no longer thought to have an effect on the degree of joint communication (Kraus-Hansen et al 1992). Therefore, in our study, it is expected that MIA injected into the TMT joint might not have extended into the DIT joint in all cases. Intra-articular MIA has been shown to be capable of producing comparable fusion in both the TMT and DIT joints when the joints are injected separately (Bohanon et al 1991).

This, the reduced amount of joint fusion seen in the DIT joint compared with the TMT joint in this study can be explained by reduced MIA access. Intra-articular contrast studies (Dyson and Romero 1993) could be performed to determine whether or not communication exists between DIT and TMT joints. However, even if communication did exist, poor diffusion of the MIA through the joint would still suggest that separate injection sites be used. We would therefore recommend that, for future chemical arthrodeses, the TMT and DIT joint be injected separately, although there is some risk of diffusion of MIA occurring into the PIT joint. This risk could be reduced by the use of contrast studies before injection.

The source of continued lameness in some horses in our study was not specifically defined. Lameness after chemical arthrodesis can arise from soft tissue inflammation, partial joint fusion or from unrelated areas of the limb (Bohanon et al 1991). Complete fusion of the joint is not necessary to eliminate joint movement so it is difficult to draw conclusions as to the contribution of partial joint fusion to the lameness seen in these cases.

Potential sources of lameness after chemical arthrodesis, not previously examined, is damage to the PIT and TC joints. Communication of the TMT and DIT joints with these more proximal joints has been well documented in the past (Sack and Orsini 1981; Kraus-Hansen et al 1992; Bell et al 1993; Dyson and Romero 1993). Dyson and Romero (1993) found that the TMT joint communicates with the PIT joint in 20% of horses. If MIA were to diffuse into the more proximal joints it could potentially cause osteoarthritis and subsequent lameness. No radiographic evidence of osteoarthritis of the PIT joint was seen in any of our cases, but this does not preclude cartilage damage occurring within the joint. Radiographic findings have been found to correlate poorly with lesions observed arthroscopically in other joints (Dyson 1987; Kannegieter and Burbridge 1990). Gustafson et al (1992) found horses given intra-articular MIA (0.09 mg/kg) exhibited mild lameness where gross pathological abnormalities of the joint were rare and histological changes mild. Bohanon et al (1991) reported unexplained lameness in 2 of 6 horses after intra-articular MIA administration. Radiographic examination of the PIT joint in these two horses showed no changes but unfortunately, neither this joint nor the TC joint were examined post mortem for evidence of osteoarthritis. Therefore, diffusion of MIA to the more proximal joints of the hock might have been a cause of lameness.

Severe peri-articular swelling after injection of MIA has not been a problem previously associated with this treatment, although a mild, transient swelling of the joint region has been reported (Yovich et al 1987; Bohanon et al 1991; Gustafson et al 1992). The extensive swelling observed in our study was considered to be due to damaging effects of MIA on soft-tissue. It is anticipated that MIA could escape from the joint in several ways. Firstly, peri-articular leakage of MIA may occur when intra-articular injections were made under pressure (Kraus-Hansen et al 1992). Bohanon et al (1991) found that injections into joints suffering from MIA-induced osteoarthritic changes were made under pressure and associated with increased swelling. The disturbed joint spaces present in many of our cases meant the intra-articular injection was usually made under pressure and therefore leakage of MIA into the surrounding soft tissue could be expected. The undesirable effects of peri-articular leakage of MIA may be reduced if the technique was modified to use MIA at a lower concentration. Also, it would appear likely that some intra-articular MIA had also diffused into the adjacent soft tissue structures of the tarsus. Studies show that the TMT joint communicates with the tarsal sheath and the tendons of the tibialis cranialis and peroneus tertius of the horse (Sack and Orsini 1981; Kraus-Hansen et al 1992; Dyson and Romero 1993). No specific studies have documented the effects of MIA on soft tissue, so it is not known whether MIA has a permanently damaging effect on soft tissue structures.

The cause of intense pain after MIA administration is not known but is more likely to be due to tissue irritation than actual joint degeneration as the changes associated with MIA osteoarthritis appear at around 4 weeks after treatment (Kalbhen 1980; Bohanon et al 1991; Gustafson et al 1992). Aside from the previously mentioned soft tissue structures, MIA can also reach the plantar metatarsal...
nerves (Dyson and Romero 1993). If MIA could damage these nerves, the subsequent neuritis may explain, at least in part, the pain seen in these cases. Bohanon et al (1991) found no significant association between swelling and lameness but suggested that soft tissue damage is still a potential source of pain. In our study those horses still chronically lame (cases 1 and 2) also had residual hock swelling. Further studies are needed to determine the nature and extent of the tissue irritant properties of MIA.

In conclusion, arthrodesis of the distal hock joints using intra-articular MIA is not more reliable than the current surgical techniques (McIlwraith and Turner 1987). Partial joint fusion, chronic lameness, pain and swelling were significant complications of this technique and were seen more often than those reported after surgical arthrodesis (Adams 1970; Edwards 1982; Wynn-Jones and May 1986; McIlwraith and Turner 1987). These complications could probably be reduced if the technique were modified as specified above and the regimen of exercise after treatment was adhered to. We believe that the technique has potential as a cheap alternative for those people unable to afford surgery, but cannot be routinely recommended nor should it be considered superior to surgery at this time.

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Pathologic lesions: they're the worst kind

"... From this limited, ad hoc survey (of 20 journals and eight books), I came up with the following picture of them.

"Like the plain ones I know about, pathologic lesions may be macroscopic or microscopic, the more common kind. Mild or severe, localised or widespread, they appear in both natural and experimental diseases. Of the several possible variants that come to mind, only neurohistopathological lesions and immunopathologic lesions are mentioned, the first much more often than the last. As far as I can tell, pathologic lesions occur in most organs (including placenta), typically in more than one at a time. Sometimes even their absence makes them notable. They are found in various domestic, laboratory, and wild animals, namely, mammals, birds, and fishes, and an occasional amphibian. I came across only one report of them in modern reptiles, but they have been seen often in dinosaurs and other prehistoric animals. Quite possibly invertebrates suffer from them as well; yet all I saw in my brief look was pathological damage in mussels. Although not my concern here, human beings also have pathologic lesions, usually for the same reasons animals do."

Hadlow WJ (1994) Vet Pathol 31:290

however, the remedy is alternative medicine

"... But the doctors were hardly wiser than they are now, and after prescribing rest and exercise, starvation and nourishment, society and solitude, that he should lie in bed all day and ride forty miles between lunch and dinner, together with the usual sedatives and irritants, diversified, as the fancy took them, with possets of newt's slobber on rising, and draughts of peacock's gall on going to bed, they left him to himself ..."

Virginia Woolf (1928) Orlando