DISSERTATION FOR THE DIPLOMA IN EQUINE ORTHOPAEDICS

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CLINICAL APPLICATIONS OF ENDOSCOPIC SURGICAL TECHNIQUES IN EQUINE ORTHOPAEDICS

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WORD COUNT: 3509
<table>
<thead>
<tr>
<th>Chapter</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>ENDOSCOPIC TREATMENT OF CONTAMINATED AND INFECTED SYNOVIAL CAVITIES</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>NON-INFECTED TENOSYNOVITIS OF THE DIGITAL FLEXOR TENDON SHEATH; A RETROSPECTIVE ANALYSIS OF 76 CASES</td>
<td>22</td>
</tr>
<tr>
<td>4</td>
<td>ENDOSCOPIC ASSESSMENT AND TREATMENT OF LESIONS OF THE DEEP DIGITAL FLEXOR TENDON IN THE NAVICULAR BURSAE OF 20 LAME HORSES</td>
<td>30</td>
</tr>
<tr>
<td>5</td>
<td>ARTHROSCOPIC TREATMENT OF FRACTURES OF THE LATERAL MALLEOLUS OF THE TIBIA</td>
<td>37</td>
</tr>
<tr>
<td>6</td>
<td>THE USE OF SMALL (2.7MM) SCREWS FOR ARTHROSCOPICALLY ASSISTED REPAIR OF CARPAL CHIP FRACTURES</td>
<td>45</td>
</tr>
<tr>
<td>7</td>
<td>CONCLUSIONS</td>
<td>55</td>
</tr>
<tr>
<td>Appendix 1</td>
<td>CURRENT DATA FOR DIGITAL FLEXOR TENDON SHEATH TENOSCOPY</td>
<td>67</td>
</tr>
<tr>
<td>Appendix 2</td>
<td>ENDOSCOPIC EVALUATION OF THE NAVICULAR BURSA; OBSERVATIONS, TREATMENT AND OUTCOME IN 92 CASES</td>
<td>69</td>
</tr>
<tr>
<td>Appendix 3</td>
<td>ENDOSCOPIC EVALUATION OF THE PREDICTIVE VALUE OF STANDING LOW-FIELD MRI FOR INJURIES WITHIN THE NAVICULAR BURSA</td>
<td>90</td>
</tr>
</tbody>
</table>
INTRODUCTION

In the brief period of time since arthroscopy was first described in the horse (Watanabe 1949), the technique has become firmly established as the standard for intra-articular surgery. The advantages of arthroscopy over arthrotomy are similar in the horse to those recognized in man; arthroscopy enables superior visualization, and access to a greater surface area of the synovial cavity; reduced patient morbidity and periods of hospitalization; earlier mobilization; reduced scar tissue formation and restrictive fibrosis; improved functional outcome in patients (Jackson 1974; O'Connor 1974). There is no longer debate over the relative merits of arthroscopy vs. arthrotomy. The question of whether a procedure should be performed arthroscopically has been replaced by “is it possible to perform the procedure arthroscopically?” This change in mindset has led to the development of increasingly sophisticated techniques, and the applications of arthroscopy thus have grown enormously.

Development of arthroscopic surgical techniques: The first surgical application for arthroscopy in the horse was removal of osteochondral fragmentation in the carpus (Ommert 1981; Valdez et al. 1983). This was followed by further description of techniques in the fetlock, carpal, tarsocrural and femoropatellar joints (Selway 1983; McIlwraith 1984; McIlwraith and Martin 1984). Description of the technique for removal of fragments from the apical and abaxial surfaces of the proximal sesamoid bones was published subsequently (Palmer 1989); this was the first description of a technique which required significant soft tissue dissection. Gradually over time, arthroscopy replaced
arthrotomy for removal of almost all intra-articular fractures, including those with extensive soft tissue attachments. These include the extensor process of the distal phalanx (Boening et al. 1989; Dechant et al. 2000; Ter Braake 2005; Crowe et al. 2010), and basilar sesamoid fractures (Parente et al. 1993; Southwood and McIlwraith 2000). In current surgical practice, even when fragment size necessitates an arthrotomy for removal, evaluation of the joint, and dissection from soft tissue attachments is first performed arthroscopically. For example, with fractures of the medial pole of the patella, the fragment location can be marked with percutaneous needles and a short-targeted arthrotomy performed for removal (Dyson et al. 1992; Marble and Sullins 2000). Limited exceptions where arthrotomy is still the accepted surgical method for removal exist, such as the supraglenoid tubercle of the scapula (Wagner et al. 1985; Pankowski et al. 1986).

Prior to inception of this dissertation, the tibia malleoli were also included on this list (Wright 1992), and the potential for arthroscopic removal had not been explored. Since then, reference has been made to cases described herein, in the standard arthroscopy text book (McIlWraith et al. 2005c), and following this by another group with an additional, limited number of cases (O'Neil and Bladon 2010).

Intra-articular soft tissue injuries: It has long been recognized that injured soft tissues also can be primary causes of joint disease. Prior to routine use of arthroscopic techniques, villonodular synovitis was a recognized condition in the dorsal fetlock joint and had been managed by excision of the enlarged synovial pad via arthrotomy (Nickels et al. 1976; van Veenendaal and Moffatt 1980). A decade passed before an arthroscopic technique was described for excision (Kannegieter 1990), and a larger number of cases
were subsequently reported (Dabareiner et al. 1996). Later, it was observed that quite commonly less severe synovial pad enlargement is noted during diagnostic arthroscopy, often in joints with concurrent osteochondral fragmentation (McIlwraith 2002).

Soft tissue injuries have also been observed in other joints. In the middle carpal joint, when using arthroscopy for removal of osteochondral fragments, surgeons noted concurrent soft tissue injuries, namely to the dorsomedial intercarpal ligament, and the lateral and medial palmar intercarpal ligaments (Kannegieter 1990; Kannegieter and Burbidge 1990; McIlwraith 1990; Whitton et al. 1997). The latter received significant further investigation, and the importance of injury to the ligament as a cause of joint disease in its own right was reported (McIlwraith 1992; Phillips and Wright 1994; Whitton et al. 1997). Debridement was recommended as a means of treatment and results following surgery were published. The rationale proposed was that debridement may facilitate healing (McIlwraith 1992). This was given support by follow-up arthroscopy in a limited number of cases, where healing was observed (Firth et al. 1991; Kannegieter and Colgan 1993). Experience with a large number of cases allowed development of prognostic values according to the severity of injury (McIlwraith 1992).

Since then there have been reports of soft tissue injuries as an important cause of lameness in other joints. In the stifle, lesions of the menisci and their cranial ligaments were described (Walmsley 1995), and further information followed from a series of 80 cases reported by the same group (Walmsley et al. 2003). Other injuries identified arthroscopically in the femorotibial joints include tears of the cranial cruciate ligament (Prades et al. 1989). In the tarsocrural joint there has been brief reference made to
arthroscopically identified tearing of the short collateral ligaments (McIlWraith et al. 2005c), and in the coxofemoral joint to lesions of the round ligament (Nixon 1994).

**Tendon sheaths and bursae:** Unsurprisingly, the advantages of minimally invasive techniques to other synovial cavities were quickly recognized and endoscopic techniques have been developed for examination of tendon sheaths and bursae. The first description was for the digital flexor tendon sheath (Nixon 1990). This was followed by the carpal sheath of the digital flexor tendons (Cauvin et al. 1997; Southwood et al. 1998), the calcaneal bursa (Ingle-Fehr and Baxter 1998), the tarsal sheath of the lateral (deep) digital flexor tendon (Cauvin et al. 1999), the navicular bursa (Wright et al. 1999) and the bicipital bursa (Adams and Turner 1999). Many of the first clinical applications (and reports) involved synovial contamination and infection (Magee et al. 1997; Ingle-Fehr and Baxter 1998; Tudor et al. 1998; Cauvin et al. 1999; Wright et al. 1999). However, as confidence with the techniques grew, application to first the investigation and then the treatment of aseptic causes of lameness followed.

A technique for tenoscopic annular ligament desmotomy, for management of annular ligament desmitis and tenosynovitis was published in 1993 (Nixon et al. 1993). In 1999, the same group from the USA published tenoscopic observations in 25 horses, which underwent desmotomy for management of lameness referable to the digital flexor tendon sheath (Fortier et al. 1999). In the same year authors in Europe reported tenoscopic observations and treatment of 20 cases with tenosynovitis of the digital flexor tendon sheath (Wright and McMahon 1999). There were differences in observations between the USA and Europe; tenosynovial masses were reported from the USA, whereas in Europe
tears of the flexor tendons predominated. Treatment also differed; all cases underwent tenoscopic debridement of injured structures, but those cases reported from Europe did not undergo concurrent desmotomy of the palmar/plantar annular ligament. Despite the similarities between cases, the differences in interpretation left a degree of confusion. Development of useful prognostic indices was limited, and the role of desmotomy in case management required clarification. Experience in a greater number of cases was needed. In 2001, the diagnostic potential of navicular bursa endoscopy was evaluated in cadaver limbs, and it was concluded a potentially useful technique for investigation of clinical cases with early podotrochleosis (Cruz et al. 2001). In 2003, an endoscopic approach through the digital flexor tendon sheath (performed blindly) was reported (Rossignol and Perrin 2003), along with endoscopic findings in 6 clinical cases with navicular disease. A transthecal approach where a window was created in the T-ligament under endoscopic guidance was latterly described (McIlWraith et al. 2005b). As for the digital flexor tendon sheath, reports of greater numbers of cases was needed to better define lesions, determine appropriate treatment protocols, aid in case selection and to define prognostic indices.

Arthroscopically guided reconstruction: A further development of arthroscopic surgical technique involved guiding internal fixation of fractures involving the articular surfaces. This was first reported with slab fractures of the third carpal bone (Richardson 1986). Arthroscopy enabled accurate reconstruction of the articular surfaces without the need for an arthrotomy. In addition, further benefits to case management were seen, by providing a superior means of assessing the entire joint and facilitating management of other
concurrent or secondary injuries (Richardson 1986, 2002). Once the benefits of arthroscopically guided internal fixation became accepted, the principles were transferred to fractures in other locations, including the condyles of the third metacarpal and metatarsal bones (Richardson 2002) and recently the proximal sesamoid bones (Busschers et al. 2008).

Until now, removal has been the treatment of choice for intra-articular chip fractures. It has been recognized for some time that the prognosis following removal of larger fragments is inferior to that following removal of smaller fragments, as demonstrated in the middle carpal joint (McIlwraith et al. 1987). Using small AO/ASIF implants it was considered possible to transfer the techniques developed for repair of intra-articular fractures to large intra-articular fragments (McIlwraith et al. 2005a). It was considered logical that conservation of articular surfaces would be beneficial, but the potential for this in clinical practice was unknown.

*Endoscopic surgery for the management of synovial contamination and infection:* The objectives in treating synovial contamination and infection are; the removal of foreign material, debridement of contaminated/infected and devitalized tissue, elimination of microorganisms, removal of destructive enzymes and radicals, promotion of tissue healing and restoration of a normal synovial environment. In man, the advantages arthroscopy offered over arthrotomy, resulted in the technique becoming popular for management of contaminated and infected synovial cavities. These advantages included superior visualization of the entire synovial cavity, identification of foreign material and infected or devitalized tissue, and access to a larger area of synovial surfaces (Dory and
Wautelet 1985; Parisien and Shaffer 1992). Experience using the technique clinically demonstrated that arthroscopy enabled efficient evaluation, debridement and decompression of the joint, with minimal morbidity, reduced periods of hospitalization and maximal functional recovery compared to other treatments (Jarrett et al. 1981; Ivey and Clark 1985; Smith 1986; Skyhar and Mubarak 1987; Thiery 1989; Parisien and Shaffer 1992; Stutz et al. 2000). In the horse, arthroscopy was first mentioned in the literature for the management of infectious arthritis in 1983 (McIlwraith 1983), and it was later compared to arthrotomy in an experimental model (Bertone et al. 1992). This demonstrated arthroscopy and partial synovial resection to be inferior to arthrotomy and open drainage. However, this experiment failed to reproduce the features typically seen in clinical cases, and arthrotomy was also associated with increased risk of secondary infection, post-operative fibrosis and requirements for greater post-operative care. In 1999, the first author of that report, in a review article, recommended arthroscopy as the primary line of therapy (Bertone 1999). Subsequently there were a number of clinical reports in equines which concluded that arthroscopy could be used effectively in the management of synovial infection, with potentially the same benefits as seen in man (Gibson et al. 1989; Ross et al. 1991; Lapointe et al. 1992; Schneider et al. 1992; Steel et al. 1999). However, these reports were limited by low case numbers and the variety of medical and surgical treatment protocols used, such that the relative benefits of arthroscopy could not be critically evaluated.

In 1999, Wright et al. described an endoscopic technique for evaluation of the navicular bursa, and documented its use in management of contamination and infection in this structure. Results compared favorably to the traditional open surgical technique.
(Richardson 1986). Later good results were reported with tenoscopic surgery in the management of infected digital flexor tendon sheaths (Frees et al. 2002). Although, the latter article combined arthroscopic treatment variably with suction drains and open drainage.

These reports all suggested arthroscopic surgery offered similar advantages in the horse to those seen in man, but still required more critical evaluation with greater numbers of cases and with a broader spectrum of synovial cavities.

The information presented in this dissertation illustrates the authors’ involvement and contributions to developing and evaluating clinical applications of endoscopic surgical techniques in the horse.
References


Endoscopic surgery in the treatment of contaminated and infected synovial cavities

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Keywords: horse; synovial; contamination; infection; endoscopy

Summary

Reasons for performing study: Contamination and infection of synovial cavities are a common occurrence in clinical practice and, if inadequately treated, may have career or life threatening consequences for affected horses.

Hypothesis: The objectives in treating contamination and infection of joints, tendon sheaths and bursae are most effectively met by endoscopic surgery.

Methods: Over a 6 year period, cases of synovial contamination and infection admitted to a referral clinic were evaluated and treated endoscopically. The horses received local and systemic antimicrobial drugs with minimal nonsteroidal anti-inflammatory medication but no other medical or surgical treatment. All arthroscopy and instrument portals and, whenever possible, all traumatic wounds were closed. Diagnostic information, endoscopic observations and results of treatment were evaluated retrospectively.

Results: A total of 140 affected animals were referred and 121 cases were treated endoscopically. These involved 70 joints, 29 tendon sheaths, 10 bursae and in 12 cases a combination of synovial cavities. The most common aetiologies were open wounds (n = 54) and self-sealing punctures (n = 41). Foreign material was identified endoscopically in 41 but predicted prior to surgery in only 6 cases. Osteochondral lesions were evident at surgery in 51 and recognised before surgery in 25 cases; 32 horses had intrahecal tendon or ligament defects.

Follow-up information was obtained for 118 animals; 106 (90%) survived and 96 (81%) returned to their preoperative level of performance. The presence of osteitis/osteomyelitis, other osteochondral lesions and marked deposits of pannus were associated with nonsurvival. For those animals which survived, non-Thorougbred horses, a combination of synovial structure involvement and regional i.v. antimicrobial administration were associated with reduced post operative performance. Marked pannus, regional i.v. antimicrobial administration and duration of systemic antimicrobial administration were associated with a group combining nonsurviving animals and those with reduced post operative performance.

Conclusions: Endoscopic surgery makes a valuable contribution to the management of synovial contamination and infection.

Potential relevance: The information obtained from and therapeutic options offered by endoscopy justify its early use in cases of synovial contamination and infection.

Introduction

Diarthrodial joints, tendon sheaths and bursae are closed spaces with a similar mesenchymal synovial lining that produces and maintains a selective physical, cellular and biochemical environment (Dyce et al. 1996). The principles of synovial contamination and infection are similar for each of these cavities. Contamination results from the introduction of micro-organisms and can occur through open wounds or self-sealing punctures, via haematogenous spread, local extension of a perisynovial infection or iatrogenically (Bertone 1996). Open wounds and self-sealing punctures also may introduce foreign material. Infection follows when the micro-organisms reproduce and colonise the synovial cavity. The principal potentiating factors for establishing infection are considered to be the presence of foreign material (Reginato et al. 1990) and/or devitalised tissue, the nature and number of contaminating organisms (Bertone 1996) and immunological compromise particularly in young animals (Firth 1983; Martins et al. 1986). Following colonisation of the synovium, a combination of bacterial pathogenicity and host immune response lead to the release of a variety of enzymes and free radicals which result in substantial inflammation and ultimately destruction of the tissues in the synovial cavity (Bertone 1996). Established infection frequently results in the production of an intrasynovial fibrinocellular conglomerate (pannus) (McIlwraith 2002). This may cover foreign material and devitalised tissue, act as a nidus for bacterial multiplication and is rich in inflammatory cells, degradative enzymes and radicals. It also is a barrier to synovial membrane diffusion therefore compromising further intrasynovial nutrition and limiting access for circulating antimicrobial drugs (Bertone 1996).

The objectives in treating contamination and infection are similar for all synovial structures: the removal of foreign material, debridement of contaminated/infected and devitalised tissue, elimination of micro-organisms, removal of destructive enzymes and radicals, promotion of tissue healing and restoration of a normal synovial environment. A number of techniques have been described which include use of drains (Jackman et al. 1989), through and through lavage (Koch 1979), open surgery (Rose and...
Love 1979; Honnas et al. 1991a; Bertone et al. 1992; Schneider et al. 1992a; Baxter 1996) and endoscopy (McIlwraith 1983; Bertone et al. 1992; Wright et al. 1999; Frees et al. 2002). Variations of these techniques have also been described, including open surgery followed by insertion of closed suction (McIlwraith 1983) or open passive (Santschi et al. 1997) drains or by open drainage (Bertone et al. 1992; Schneider et al. 1992a) and endoscopy followed by closed suction drainage (Ross et al. 1991; LaPointe et al. 1992), fenestrated drains (Honnas et al. 1991b) or creation of an open draining wound (Bertone 1999).

In treating joint infection in man, arthroscopy is considered to offer several advantages over lavage and arthrotomy including improved visualisation, identification of foreign material and infected or devitalised tissue and access to a larger area of synovial surfaces (Dory and Wantelet 1985; Parisien and Shaffer 1990). Arthroscopy ensures an efficiently evaluated, cleaned, debrided and decompressed joint with minimal morbidity, reduced period of hospitalisation and maximal functional recovery compared to other treatments (Jarrett et al. 1981; Ivey and Clark 1985; Smith 1986; Skyhar and Mubarak 1987; Thiry 1989; Parisien and Shaffer 1990; Stutz et al. 2000).

The authors hypothesised that the objectives in treating synovial contamination and infection in horses are most effectively met by endoscopic surgery. This paper describes a retrospective analysis of 121 cases treated endoscopically with no adjunctive surgical interference and, whenever possible with primary wound closure.

Materials and methods

All cases of synovial contamination and infection admitted to a referral clinic between 1 January 1996 and 31 December 2001 were identified. A synovial cavity was considered contaminated or infected if communication with an open wound could be demonstrated and/or if synoviocentesis yielded samples consistent, on the basis of gross appearance, protein content and cellularity with contamination or infection. Wounds were described as open if synovial drainage was evident or could be demonstrated on synovial inflation and as self-sealed punctures if these features were absent.

TABLE 1: Age, gender and breed of 121 cases of contaminated or infected synovial cavities

<table>
<thead>
<tr>
<th>Details</th>
<th>No. cases</th>
</tr>
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<tr>
<td>Age (years)</td>
<td></td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>12</td>
</tr>
<tr>
<td>0.5–1.0</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>5–10</td>
<td>47</td>
</tr>
<tr>
<td>11–20</td>
<td>13</td>
</tr>
<tr>
<td>&gt;20</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>3</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>28</td>
</tr>
<tr>
<td>Gelding</td>
<td>43</td>
</tr>
<tr>
<td>Female</td>
<td>50</td>
</tr>
<tr>
<td>Breed</td>
<td></td>
</tr>
<tr>
<td>Thoroughbred</td>
<td>72</td>
</tr>
<tr>
<td>Thoroughbred-cross</td>
<td>31</td>
</tr>
<tr>
<td>Pony</td>
<td>5</td>
</tr>
<tr>
<td>Other</td>
<td>13</td>
</tr>
</tbody>
</table>

Of 140 cases identified, 19 did not receive endoscopic treatment. Wound size precluded synovial inflation in 11, extensive intrasynovial fibrosis and adhesion formation resulted in multi loculation in 2, infected acquired bursae were excised in toto in 3 and the primary foci of infection were extra synovial in 3 horses.

The remaining 121 cases received endoscopic evaluation and treatment. Clinical case notes were reviewed and data collected. Individual clinical and surgical variables were compared using the Chi-squared test with a significance level of P<0.05. Follow-up information was obtained via a telephone questionnaire with owners and, for racing Thoroughbreds, race records also were evaluated. Horses were classified as survivors or nonsurvivors. Those surviving were further classified as reaching a level of performance equal or better than that achieved before treatment or having a reduced level of performance.

Comparisons were made between survivors and nonsurvivors; between animals whose level of performance was equal or better than that achieved before treatment with those with reduced post operative performance and between animals which returned to a pretreatment level of performance with those that had reduced performance together with nonsurvivors. Continuous variables were inspected and analysis performed in their continuous state and with division into statistically and biologically plausible categories. Statistical analysis was performed using logistical regression analysis with a significance level of P<0.05.

Results

Case details

The age, gender and breed of cases are summarised in Table 1. A total of 140 synovial cavities were contaminated or infected, including 11 horses with 2 and 4 horses with 3 structures involved. Eighty-five (70%) cases involved hindlimbs and 36 (30%) forelimbs. The individual structures affected are detailed in Table 2. Joints were affected in 70 (58%), tendon sheaths in 29 (24%), bursae in 10 (8%) and a combination of synovial structures in 12 (10%) horses. The distribution of aetiologies is shown in Figure 1.

Fig 1: Aetiologies of 121 cases of synovial contamination or infection. W = wound, 54 cases (45%); P = puncture, 41 cases (34%); H = haematogenous, 14 cases (11%); I = local erosion, 8 cases (7%); L = iatrogenic, 4 cases (3%).

History

Treatment prior to presentation included only antimicrobial and anti-inflammatory drugs immediately before referral (n = 69), medical regimens (n = 33) or surgery (n = 14). There was no information for 5 animals. Of those treated medically, 15 received
I. M. Wright et al.

Synovial structures affected in 121 cases of contamination and infection

<table>
<thead>
<tr>
<th>Synovial structure</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digital flexor tendon sheath</td>
<td>27</td>
</tr>
<tr>
<td>Tarsocrural joint</td>
<td>22</td>
</tr>
<tr>
<td>Metatarsophalangeal joint</td>
<td>15</td>
</tr>
<tr>
<td>Navicular bursa</td>
<td>10</td>
</tr>
<tr>
<td>Distal interphalangeal joint</td>
<td>9</td>
</tr>
<tr>
<td>Femoropatellar joint</td>
<td>9</td>
</tr>
<tr>
<td>Middle carpal joint</td>
<td>6</td>
</tr>
<tr>
<td>Medial femorotibial joint</td>
<td>6</td>
</tr>
<tr>
<td>Metacarpophalangeal joint</td>
<td>6</td>
</tr>
<tr>
<td>Antebrachiocarpal joint</td>
<td>5</td>
</tr>
<tr>
<td>Lateral femorotibial joint</td>
<td>5</td>
</tr>
<tr>
<td>Extensor carpi radialis tendon sheath</td>
<td>4</td>
</tr>
<tr>
<td>Calcaneal bursa</td>
<td>3</td>
</tr>
<tr>
<td>Proximal interphalangeal joint</td>
<td>3</td>
</tr>
<tr>
<td>Tarsal sheath of the lateral digital flexor tendon</td>
<td>2</td>
</tr>
<tr>
<td>Common digital extensor tendon sheath</td>
<td>1</td>
</tr>
<tr>
<td>Long digital extensor tendon sheath</td>
<td>1</td>
</tr>
<tr>
<td>Acquired dorsal metatarsophalangeal bursa</td>
<td>1</td>
</tr>
<tr>
<td>Bicipital bursa</td>
<td>1</td>
</tr>
<tr>
<td>Coxofemoral joint</td>
<td>1</td>
</tr>
<tr>
<td>Carpal canal</td>
<td>1</td>
</tr>
<tr>
<td>Extensor carpi obliquus tendon sheath</td>
<td>1</td>
</tr>
<tr>
<td>Scapulohumeral joint</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>140</strong></td>
</tr>
</tbody>
</table>

Only antimicrobial drugs, 14 antimicrobial and nonsteroidal anti-inflammatory drugs and 4 anti-inflammatory drugs only. Prereferral antimicrobial medication was given for 2–24 (mean 13) days and nonsteroidal anti-inflammatory medication for 2–31 (mean 8) days. Eight of the animals treated surgically had received through and through lavage and 3 had undergone arthroscopy. One animal had received tendon sheath drainage and intraarticular antimicrobial medication and 5 had undergone wound debridement and closure including in one case placement of an indwelling drain. All of the horses previously treated surgically had received antimicrobial medication.

Clinical evaluation

All cases were evaluated radiographically. No significant abnormalities were present in 86 (71%) cases. Identified lesions included gas shadows in synovial cavities (n = 12), foreign bodies (n = 2) and osseous defects (n = 30). The latter included fractures or fragmentation (n = 16), evidence of infected osteitis or osteomyelitis (n = 12) and reactive perisynovial new bone (n = 2). Ultrasonography was performed in 22 horses. It was supportive of synovial contamination or infection in all cases and identified additional features in 10 involving, evidence of foreign bodies (n = 4), tendon defects (n = 3), peri-articular foci of infection (n = 2) and a distended bicipital bursa.

Endoscopy

The interval between injury (or first observed clinical signs) and surgery varied from 2 h to 10 weeks (Table 3). All horses underwent endoscopy using a 4 mm 25° forward oblique arthroscopes under general anaesthesia. Esmarch bandages and tourniquets were employed with distal limb involvement. Endoscopic observations are recorded in Table 4. Foreign material was observed in 41 animals; 17 with wounds and 24 with self-sealing punctures. This included hair (n = 21), thorns (n = 8), other wood (n = 4), glass (n = 1), road grit (n = 1), paint (n = 1) and unspecified or unrecognisable foreign material (n = 5). Foreign material was free floating (n = 22), attached to synovium or to pannus (n = 11), embedded in cartilage or bone (n = 17), penetrating synovial membrane (n = 4) or embedded within tendons (n = 4).

Pannus was present in 64 cases and classified as marked (n = 44), moderate (n = 6) or minor (n = 14). Osseous lesions were identified in 38 and osteochondral lesions in 51 horses. Intraarticular tendon or ligament defects were present in 32 horses all of which had wounds or punctures.

There were significant associations between presence of pannus and increasing duration of clinical signs (P < 0.001), presence of osteochondral lesions (P = 0.011) and presence of osteomyelitis (P = 0.002). Synovial fluid protein levels >61 g/l (P = 0.010) and presence of >90% neutrophils (P = 0.026) also were significantly associated with presence of pannus. Foreign material was significantly associated with osteochondral lesions (P = 0.025) and both osteochondral lesions (P < 0.001) and osteomyelitis (P < 0.001) were significantly associated with clinical signs of >7 days duration.

<table>
<thead>
<tr>
<th>Synovial structures</th>
<th>Foreign material (%)</th>
<th>Pannus (%)</th>
<th>Osseous or osteochondral fragmentation (%)</th>
<th>Osteochondral defects without fragmentation (%)</th>
<th>Cartilage loss (%)</th>
<th>Osteomyelitis (%)</th>
<th>Tendon/ligament defects (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joints (88)</td>
<td>31 (35)</td>
<td>47 (53)</td>
<td>17 (19)</td>
<td>3 (4)</td>
<td>8 (10)</td>
<td>11 (13)</td>
<td>9 (10)</td>
</tr>
<tr>
<td>Tendon sheaths (37)</td>
<td>8 (22)</td>
<td>12 (55)</td>
<td>0 (0)</td>
<td>1 (3)</td>
<td>0 (0)</td>
<td>2 (5)</td>
<td>14 (38)</td>
</tr>
<tr>
<td>Bursae (15)</td>
<td>6 (40)</td>
<td>11 (73)</td>
<td>0 (0)</td>
<td>1 (7)</td>
<td>5 (33)</td>
<td>4 (27)</td>
<td>9 (60)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>45 (32)</strong></td>
<td><strong>70 (50)</strong></td>
<td><strong>17 (12)</strong></td>
<td><strong>5 (4)</strong></td>
<td><strong>13 (9)</strong></td>
<td><strong>17 (12)</strong></td>
<td><strong>32 (23)</strong></td>
</tr>
</tbody>
</table>
Following initial lavage, to improve visibility, thorough endoscopic evaluation, usually with multiple portals, was performed in order to evaluate completely all aspects of each cavity. Foreign material was removed; large pieces with Ferris-Smith arthroscopic rongeurs and small pieces with a motorised synovial resector. If embedded in bone, arthroscopic curettes were also employed. Osteochondral fragments were removed with rongeurs; fracture beds and osteochondral defects were debrided with curettes. Foci of infected osteitis/osteomyelitis were debrided with rongeurs before debridement with curettes. A motorised synovial resector was used to debride contaminated and infected tendon and ligament lesions. Localised deposits of pannus were retrieved with rongeurs but, when widespread, were removed using a motorised synovial resector. Contaminated or infected synovium immediately adjacent to wounds or punctures was resected using a motorised synovial resector in 22 cases. Partial resection of synovium remote from wounds was performed in a further 9 cases and extensive synovial resection was carried out in 34 animals involving 36 synovial cavities (25 joints, 5 tendon sheaths and 6 bursae).

All structures received thorough, directed, high-pressure lavage with sterile buffered polyionic solution (Isolec) delivered by a peristaltic pump using multiple arthroscope and instrument/egress portals until all areas were visibly clean. In cases with traumatic wounds or punctures these were used as instrument/egress portals. Antimicrobial drugs were added to the final lavage fluid in all cases. In 118 animals this consisted of sodium benzyl penicillin G (Crystapen) at 2.5 x 10^6iu/l and gentamicin sulphate (Genta 100) at 250 mg/l. Sodium ceftiofur (Excenel) at 500 mg/l and gentamicin sulphate at 250 mg/l were used in the remaining 3 animals.

All arthroscope and instrument portals were closed either with simple interrupted sutures of monofilament polyamide (Ethilon) or with stainless steel staples (PMW). Solar puncture wounds were debrided, dressed and allowed to heal by second intention. All other traumatic wounds were debrided or excised and whenever possible these were closed. Skin closure only was performed in 12 animals using either simple interrupted sutures of monofilament polyamide or stainless steel staples. In 9 cases, closure was effected by buried sutures of polyglactin 910 (Vicryl) followed by stainless steel staples in the skin and in 27 animals, buried sutures of polydioxanone (PDS) followed by stainless steel staples in the skin. In 11 animals extensive soft tissue loss precluded closure and these were managed as open wounds.

Medical treatment and post operative care

At the end of surgery, antimicrobial drugs were given by a regional i.v. technique to 20 horses with established distal limb infection. This involved 5 forelimbs and 15 hindlimbs in which 13 joints, 2 tendon sheaths and 10 bursae were affected. Eighteen animals received sodium benzyl penicillin G (2.5 x 10^8iu) and gentamicin sulphate (500 mg) and 2 animals received sodium ceftiofur (500 mg) and gentamicin sulphate (500 mg).

Following surgery 16 limbs with large skin wounds were immobilised in plaster of Paris (Gypsonosa) and fibreglass (Vetcast) combination casts for 7–28 (mean 17) days. In the remaining cases counterpressure was applied to the affected areas by layered cotton wool (modified Robert-Jones) bandages in 48 limbs, elasticated carpal or tarsal bandages (Pressage) in 32 limbs, by stent bandages oversewn with sheathed braided polyamide (Supramid) in 18 limbs and combinations of these techniques in 6 limbs.

All cases received systemic antimicrobial medication which began preoperatively. Seventy-five animals received i.v. sodium benzyl penicillin G (30,000iu/kg bwt i.v. q. 8 h) and gentamicin sulphate (2.2 mg/kg bwt i.v. q. 8 h). Forty-two cases also received metronidazole (Metronex) (20 mg/kg bwt p.o. q. 8 h). Sodium ceftiofur (3–4 mg/kg bwt i.v. q. 8 h) and gentamicin sulphate (2.2 mg/kg bwt i.v. q. 8 h) were given to 3 animals. The duration of antimicrobial administration was 6–54 (mean 13) days and was determined by the clinical features and response of individual animals. Administration continued until there was consistent improvement in lameness, and reduced surface temperature adjacent to the affected cavity, synovial distension, perisynovial swelling and engorgement of visible draining veins.

Phenylbutazone (Equipalazone) (4 mg/kg bwt) was given as part of the anaesthetic protocol to all animals. Post operatively, 8 cases received a further single dose of phenylbutazone and 14 horses received nonsteroidal anti-inflammatory drugs for 3–53 (mean 22) days post operatively. In 5 cases, this began immediately after surgery and, in the others, commenced once clinical signs of infection had subsided.

With the exception of animals in casts, all horses began walking exercise the day after surgery. Animals that had been immobilised in casts began walking after cast removal. Horses were discharged from the clinic shortly after cessation of antimicrobial medication. The duration of hospitalisation ranged from 6–71 (mean 18) days.

Post operative complications

Four animals had anaesthetic related complications. One suffered a fractured femur when attempting to stand and was destroyed immediately, one suffered radial neurapraxia which recovered over 3 days and 2 had post operative colic which responded to medical management. Thirteen animals had signs of persistent or recurrent infection. Synoviocentesis was performed in 10 of these and 7 revealed fluid consistent with an infective process. Further radiographs were taken in 6 cases of which 5 exhibited evidence of infected foci. Six horses underwent further endoscopic surgery. Additional lesions identified endoscopically included foreign material (1), osteomyelitis (1), sequestrum formation (1) and a periarticular focus of infection (1). One animal was destroyed at this time. Seven animals with persistent or recurrent infection changed antimicrobial regimens. Three horses received sodium ceftiofur (4 mg/kg bwt i.v. q. 8 h) and gentamicin sulphate (2.2 mg/kg bwt i.v. q. 8 h), 2 horses received enrofloxacin (Baytril) (5 mg/kg bwt i.v. q. 24 h), 1 received sodium benzyl penicillin G (30,000iu/kg bwt i.v. q. 8 h) and amikacin sulphate (Amikin) (6.6 mg/kg bwt i.v. q. 8 h) and 1 received sodium ceftiofur (4 mg/kg bwt i.v. q. 8 h) alone.

Four animals suffered additional post operative complications. One horse, which had an infected digital flexor tendon sheath including a wound into the deep digital flexor tendon, underwent further tenoscopy 4 months after the first surgery to remove a granuloma and associated adhesions that had formed at the tendon trauma site. Another horse treated for an infected hindlimb digital flexor tendon sheath showed signs of progressive forelimb lameness 12 days post operatively caused by an infected bicipital bursa and this, in turn underwent endoscopic evaluation and treatment. One animal developed...
TABLE 5: Results of the logistic regression of survival vs. nonsurvival

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. cases</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>P value</th>
<th>Max likelihood test P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteochondral pathology</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>70</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>51</td>
<td>6.384</td>
<td>1.313–31.028</td>
<td>0.022</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Osteomyelitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>104</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>17</td>
<td>6.250</td>
<td>1.651–23.654</td>
<td>0.007</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pannus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate/minor/none</td>
<td>77</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marked</td>
<td>44</td>
<td>5.487</td>
<td>1.081–27.854</td>
<td>0.040</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

CI = confidence interval; Max = maximum.

Follow-up

Follow-up information was available for 118 cases of which 106 (90%) horses survived with 96 (81%) animals returning to a preoperative level of performance. Joint involvement had an 89% survival rate with 65 (81%) animals returning to a preoperative level of performance. Tendon sheaths had a 94% survival rate with 30 (86%) animals recording a preoperative level of performance. Thirteen (87%) animals with bursal involvement survived with 9 (60%) returning to a preinjury level of performance. Eleven of 12 (92%) animals with a combination of synovial structures affected survived, and 8 (67%) returned to a previous level of performance. Eight of the 10 animals that received additional or amended treatment following endoscopy survived and 6 returned to their previous level of performance.

Endoscopic evidence of osteochondral lesions, osteitis/osteomyelitis and marked pannus deposits were associated significantly with nonsurvival (Table 5). In the survivors, non-Thoroughbred horses, a combination of synovial structure involvement and regional i.v. administration of antimicrobial drugs had significant associations with reduced post operative performance (Table 6). When nonsurvivors and horses which had reduced post operative performance were grouped together there were significant associations with the presence of marked pannus, regional i.v. administration of antimicrobial drugs, and duration of antimicrobial administration (Table 7). The poor outcome associated with involvement of a combination of synovial cavities (4 of 4) and regional i.v. administration of antimicrobial drugs (7 of 8) were cases involving the navicular bursa and/or distal interphalangeal joint.

Discussion

The subject details in this series reflects the clinic population and differs from other published series of synovial infection in the predominance of Thoroughbreds and a smaller proportion of foals (Schneider et al. 1992b). In the current series, 79% of cases resulted from wounds and punctures which contrast with 24% of infected joints and tendon sheaths reported by Schneider et al. (1992b) but is less than the 88% of infected tendon sheaths reported by Honnas et al. (1991a). Synovial infection of haematogenous origin, involving animals less than age 6 months was the most common aetiology (34%) reported by Schneider et al. (1992b) which also recorded 35% of cases having iatrogenic aetiologies contrasting with the 3% reported here.

Endoscopy revealed osseous defects in 38 horses while radiography predicted accurately these lesions in 21 (55%) animals. In 4 horses, there were additional or more extensive lesions than identified radiographically and, in the remaining 13 horses, there were radiologically silent/undetected defects. Fifty-one horses (42%) had endoscopically identifiable osseous or chondral lesions of which only 25 (49%) were predicted before endoscopy. Foreign material was found at endoscopy in 34% of the total and 43% of the horses with wounds or punctures, but was predicted prior to endoscopy in only 15% of animals. Frees et al. (2002) identified tenosynovial foreign material in 20% of 20 open digital flexor tendon sheaths. An association has been reported in man between the presence of retained foreign material and the development of infected synovitis following penetrating wounds (Reginato et al. 1990). Foreign material acts as a nidus for infection and causes...
intrasynovial irritation and damage both physically and biochemically. Endoscopy permits thorough evaluation of synovial cavities, facilitating removal of foreign material and debridement of infected and devitalised tissue (Baxter 1996; Tudor et al. 1998; Wright et al. 1999). It also permits extensive, visually directed lavage (Gaughan 1994; Frees et al. 2002) aiding removal of small free floating debris, debulking microorganisms and removing destructive radicals and enzymes. In the current series, the authors prioritised physical cleansing of the synovial environment over medical management of pathogens and the reactions that they precipitate.

In managing the reported case series, the authors hypothesised that contaminated and infected wounds can be debrided effectively or excised to a clean/contaminated state and then closed safely. This is based also on the premise that endoscopic surgery can cleanse thoroughly synovial cavities and that a closed wound minimises the risk of further and/or secondary contamination or infection. These principles contrast with others in the literature (Gibson et al. 1989; Schneider et al. 1992a) who advocate open management of infected synovial cavities. The authors also consider that the application of counterpressure is a positive contributor to the control of infection and to promotion of first intention wound healing. It appears also to reduce pain associated with synovial infection (Nixon 1990). Movement is necessary for restoration of normal synovial physiology and an early return of exercise has been advocated also by Nixon (1990) and Frees et al. (2002).

Horses that develop synovial infection following penetrating wounds are likely to have multiple bacteria involvement (Schneider et al. 1992b). The bacteriological studies published by Snyder et al. (1987), Jackman et al. (1989), Honnas et al. (1991a), Moore et al. (1992) and Schneider et al. (1992b) and susceptibility patterns determined by Snyder et al. (1987), Moore et al. (1992), and Schneider et al. (1992b) suggest that the synergistic combination of penicillin and gentamicin is, in most circumstances, an appropriate systemic antimicrobial regimen. If involvement of an anaerobic species is suspected then metronidazole is a logical addition (Moore et al. 1992). In this series the adoption, in the first instance, of restricted systemic antimicrobial regimens independent of positive identification of organisms appears justified and is considered to have been facilitated by the efficient mechanical cleansing and debulking of microorganisms by the endoscopic techniques. For similar reasons, the period of systemic antimicrobial medication was shorter than other reports and recommendations in the literature (Bertone et al. 1987; Gibson et al. 1989; Nixon 1990; Honnas et al. 1991a; Bertone 1999). A reduced period of systemic antimicrobial administration following arthroscopic treatment of infected joints has also been reported in man (Smith 1986).

Nonsteroidal anti-inflammatory drugs have been advocated in the treatment of synovial infection to provide analgesia and to inhibit (in part) the inflammatory cascade in order, it is theorised, to limit the deleterious effects of inflammatory mediators on the synovial environment (Bertone and McIlwraith 1987; Cook and Bertone 1998; Schneider 1999). However, their administration limits the use of clinical signs to monitor response to treatment and to determine an appropriate time for cessation of antimicrobial medication.

Schneider et al. (1992b) reported an average period of hospitalisation of 21 days for 192 cases of infected arthritis and tenosynovitis and an unspecified number continued to receive antimicrobial drugs after this time. By contrast in the current series all antimicrobial administration had ceased prior to discharge from the clinic. Reduced hospitalisation of animals with contaminated and infected navicular bursae has been reported previously (Wright et al. 1999) and the results of the current series indicate that, with endoscopic treatment, this also is possible with other synovial structures.

Most previous reports of treatment of contaminated and infected synovial cavities involved multiple treatment protocols (Gibson et al. 1989; Ross et al. 1991; LaPointe et al. 1992; Schneider et al. 1992a,b; Steel et al. 1999; Frees et al. 2002). Nonetheless, the results reported with the current series compare favourably with all other regimens both for survival and return to preinjury levels of performance. The presence of osteochondral lesions and of infected bone are related, in the current series, to non survival but not to poor post operative performance which suggests that these features may not be of causative significance. By contrast, the presence of marked pannus which was related both to non survival and reduced post operative performance appears likely to be of prognostic significance. The association between use of regional i.v. administration of antimicrobial drugs with non survival and reduced post operative performance may reflect the surgeon’s selection criteria. This technique was used most frequently with established infection of navicular bursae, distal interphalangeal joints and/or digital flexor tendon sheaths. The association between nonsurvival and reduced post operative performance with duration of systemic antimicrobial administration was predictable with the criteria used to determine their period of use. The lack of relationship between outcome and duration of clinical signs prior to treatment is a surprising observation. It appears logical that this feature should be of prognostic significance (McIlwraith 1983; Wright and Scott 1989; Gaughan 1994) and has been reported as such by Gibson et al. (1989) and Baxter (1987). However, other authors also found no correlation between the duration of clinical signs prior to treatment and case outcome (Honnas et al. 1991a; LaPointe et al. 1992; Frees et al. 2002).

It is concluded that endoscopic treatment of contaminated and infected synovial cavities permits thorough evaluation with appropriate debridement, effective lavage and minimal tissue trauma. Multiple synovial cavities may be treated simultaneously, there is early pain relief, few complications and minimal post operative care. A standard systemic antimicrobial regimen has given consistent good results, animals have been able to make an early return to exercise and the prognosis appears to be better than with other reported protocols.

Manufacturers’ addresses

1 Karl Storz GmbH & Co., Tuttingen, Germany.
2 Sontec, Englewood, Colorado, USA.
3 Dynolite/Smith & Nephew Endoscopy Inc., Andover, Massachusetts, USA.
4 Iven Pharmaceuticals, Larne, UK.
5 Cole Palmer, London, UK.
6 Pitman-Moore Ltd, Crewe, Cheshire, UK.
7 CP-pharma, Burgdorf, Germany.
8 Upjohn Animal Health, Crawley, Sussex, UK.
9 Ethicon, Edinburgh, UK.
10 Smith & Nephew, Le Mans, France.
11 JMI, St Paul, Minnesota, USA.
12 Jupier Products, Cardiff, UK.
13 Smith, Hunningen, Belgium.
15 Arnolds Veterinary Products, Shrewsbury, Shropshire, UK.
16 Bavier plc, Bury St Edmunds, Suffolk, UK.
17 Bristol-Myers Squibb, Hounslow, Middlesex, UK.


Noninfected tenosynovitis of the digital flexor tendon sheath: a retrospective analysis of 76 cases

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Keywords: horse; tenosynovitis; tenoscopy; digital flexor tendons

Summary

Reasons for performing study: Until recently, the pathogenesis of noninfected tenosynovitis of the digital flexor tendon sheath (DFTS) has been considered obscure. With the advent of tenoscopy some inciting causes have been described, but to date few cases with a precise diagnosis have been reported.

Hypothesis: Tenoscopic surgery enables accurate diagnosis and facilitates treatment of noninfected tenosynovitis of the DFTS.

Methods: The case records of all horses with noninfected tenosynovitis of the DFTS admitted to a referral clinic over a 7-year period were evaluated retrospectively. Follow-up information was obtained by telephone questionnaire.

Results: A total of 76 horses were referred; all were evaluated tenoscopically and 11 of these subsequently were explored by open surgical techniques. The most common diagnoses were marginal tears of the deep digital flexor tendon (DDFT) (n = 44) and tears of the manica flexoria (MF) (n = 23). Greater than 6 months’ follow-up information was available for 61 animals, of which 68% were sound and 54% returned to preoperative levels of performance. Sheath distension was eliminated in 33% and improved in 69% of horses. Marginal tears of the DDFT, marked preoperative distension and open surgical repair of deep digital flexor lesions were associated with reduced levels of post operative performance: Marginal tears were associated with post operative lameness and long tears with a reduced performance level compared with short tears. Marginal tears of the deep digital flexor tendon, marked preoperative distension and increasing duration of clinical signs also were associated with lack of improvement in distension following surgery.

Conclusion: Tenosynovitis of the DFTS may result from tears in the deep or superficial digital flexor tendons, manica flexoria or other structures with synovial communication. To date, these can be identified confidently only by tenoscopy, which also permits appropriate lesion management.

Potential relevance: The diagnostic information obtained from and therapeutic options offered by tenoscopy justify its early use in cases of noninfected tenosynovitis of the DFTS.

Introduction

The digital flexor tendon sheath (DFTS) is a complex synovial cavity housing the superficial digital flexor tendon (SDFT), deep digital flexor tendon (DDFT) and their associated plicae, mesotenons, manicae and vinculae. It extends from the distal metacarpus/metatarsus to the middle of the middle phalanx. Palmar/plantar to the metacarpal/tarsalphalangeal joint, the DFTS passes through an inelastic canal created by the palmar/plantar annular ligament (PAL), the palmar/plantar surface of the proximal sesamoid bones and the intervening intersesamoidean ligament (Sisson 1975; Redding 1991; Wright and McMahon 1999).

Lameness associated with noninfected tenosynovitis of the DFTS has been ascribed to tendonitis of the SDFT (McIlwraith 2002) and DDFT (Barr et al. 1995), desmitis of the PAL (Dik et al. 1991), DFTS rupture (Dyson and Denoix 1995), longitudinal tears of the DDFT (Wright and McMahon 1999; Wilderjans et al. 2003), tears of the manica flexoria (MF) (Wright and McMahon 1999), complex tenosynovitis (Fortier et al. 1999; Nixon 2003), desmitis of the proximal digital annular ligament (Schramme and Smith 2003) and proliferative synovitis of unknown cause (Gerring and Webbon 1984). This report describes the clinical features, diagnoses, treatment and outcomes of a series of horses with noninfected tenosynovitis of the DFTS.

Materials and methods

The records of 76 cases of noninfected tenosynovitis of the DFTS presented to a referral clinic between 1st January 1996 and 31st December 2003 were identified, 2 of which were reported previously by Wright and McMahon (1999). For inclusion in the study, the affected DFTS had to be examined tenoscopically by the second author. Case records were reviewed retrospectively by the first author. Data collected included: age, breed, gender and use of the horse; historical information, including duration of clinical signs and treatments administered prior to admission; clinical findings, including affected limb, palpable abnormalities, degree of lameness graded out of 10 (0 = sound, 10 = nonweightbearing; Ross 2004); radiographic and ultrasonographic abnormalities; tenoscopic findings; surgical treatment; and post operative treatments.

Ultrasonographic images were reviewed blindly and subsequently compared with tenoscopic findings. For marginal
tears of the DDFT and tears of the MF, the sensitivity, specificity, positive predictive value and negative predictive value of ultrasound examination were calculated.

Follow-up information was obtained by telephone questionnaire from owners and referring veterinary surgeons, detailing complications, current degree of lameness, current activity, period of convalescence and appearance of the affected limb. Owners or referring veterinarians were asked to classify the horse as sound or lame, performing at a level equal to or better than before onset of lameness or having a reduced level of performance. Additionally, animals were grouped as having no identifiable distension, reduced distension or distension of the DFTS equal to or worse than before surgery.

For analysis, each individual clinical variable (detailed above) was divided into statistically and biologically plausible groups. In addition, marginal tears of the DDFT were classified as long (extending from beneath the MF to terminate within or distal to the sesamoidean canal) or short. Logistic regression was used to identify significant associations between the 3 dependent variables (lameness; performance; distension) and the clinical variables. A significance level of P<0.05 was assumed for all tests.

Results

Clinical details

The age, breed and gender of cases are displayed in Table 1. The animals were used for a variety of purposes; 10 were showjumpers, 8 racehorses, 8 polo ponies, 10 event horses, 37 used for general purpose riding and the use of 3 horses was not recorded.

Duration of clinical signs prior to referral was 1–104 (mean 16) weeks. Twenty-one cases (36%) presented within 4 weeks of onset of clinical signs, 19 (32%) between 5 and 15 weeks and 19 (32%) at greater than 15 weeks. For 17 horses this information was not recorded. During this period, 53 horses had received no recorded treatment other than rest, controlled exercise and bandaging. Twenty-two animals had received medical treatment and one had also been treated surgically. Of those receiving medical treatment 9 animals received systemic nonsteroidal anti-inflammatory drugs (NSAIDs) alone, one NSAIDs and intrathecal medication and 12 intrathecal medication alone. Intrathecal medication in 11 cases consisted of corticosteroids, in one case corticosteroids and hyaluronan and in one animal hyaluronan alone. The case treated surgically had undergone 2 previous tenoscopic procedures.

Digital flexor tendon sheaths were affected in 41 forelimbs (including 1 animal with bilateral affliction) and 36 hindlimbs. All animals were lame at presentation with severities varying from grade 1–5/10. All DFTSs were distended; distension was classified as mild (n = 15), moderate (n = 26) or marked (n = 36). In addition, there was palpable synovial thickening in 49 horses and nodular masses in 17 horses. An increase in overlying skin temperature and a pain response on palpation of the flexor tendons were frequent but nonquantified clinical findings. Tendon thickening was palpable in 17 DFTSs (10 DDFT, 3 SDFT and 4 medial branches of insertion of SDFT). The PAL was palpably thickened in 4 cases and in 2 horses a defect of the DFTS wall was palpated.

Radiography

Thirty-six horses were radiographed before surgery. In 2 cases there was irregular new bone on the abaxial margin of both proximal sesamoid bones and in one case foci of dystrophic mineralisation were evident in the region of the PAL. No significant abnormalities were detected in the remaining 33 animals.

Ultrasonography

Ultrasonographic images were available for retrospective evaluation in 71 cases. These confirmed the palpable findings of fluid distension (71/71) and synovial thickening (32/32), and demonstrated additional nonspecific changes including thickened proximal plicae of the DDFT (n = 10), intrasynovial echogenic debris (n = 41) and..
adhesion formation (n = 2). In 36 DFTSs (35 cases), ultrasonographic findings (truncated tendon margin, echogenic mass extending from and contiguous with the tendon or hypoechoic areas extending to the tendon margin) were consistent with marginal tears of the DDFT (n = 32) (Fig 1a) and SDFT (n = 4). Twelve DFTSs had ultrasonographic evidence of a torn MF (incompletely identifiable MF, strands of echogenic material adjacent to the normal position of the MF, and irregular margin of the SDFT adjacent to the MF) (Fig 1b). Further ultrasonographic abnormalities were noted in 13 cases; heterogeneous and enlarged medial branch of the SDFT (n = 3), thickened PAL/subcutaneous tissue palmar to PAL (n = 6), DFTS rupture and synoviocoele (n = 2), and marked thickening of the lateral plica of the DDFT (n = 2). Nonspecific ultrasonographic changes only were present in the remaining 21 cases.

Tenoscopy

All horses were evaluated tenoscopically under general anaesthesia; 73 animals were positioned in lateral recumbency and 3 (including the bilaterally affected horse) in dorsal recumbency. In lateral recumbency the affected limb was placed uppermost in 70 cases; in 3 animals, in which ultrasonography suggested a medial lesion, the affected limb was positioned lowermost. The limbs were exsanguinated with an Esmarch bandage and tourniquets were applied to the distal antebrachium or crus.

In all cases, tenoscopy was performed using a 4 mm 25° forward oblique arthroscope introduced between the PAL and proximal digital annular ligament (Nixon 1990). Eleven horses (including the bilaterally affected individual) subsequently underwent open surgery for repair or removal of lesions identified at tenoscopy. In 2 animals in which the PAL was thickened, this was sectioned by an endoscopically assisted ‘freehand’ technique using a curved meniscectomy knife.

The tenoscopic findings are summarised in Table 2. Forty-six marginal tears of the DDFT were identified in 45 DFTSs (44 horses) involving 32 fore- and 13 hindlimbs. Tears of the DDFT occurred in combination with tears of the MF (n = 5) and SDFT (n = 1). Two further cases had thickening of the PAL, and in 4 cases adhesions were present between the torn surface of the DDFT and the DFTS wall. In all except one case, torn tendon fibrils protruded into the synovial cavity and were identified suspended in the irrigating fluid (Figs 2a–c). Tears were located proximal to (n = 33), within (n = 3) and distal to (n = 10) the sesamoidean canal. The length of the tear was described in 35 cases (37 tears in 36 DFTSs). Long tears (n = 16) invariably involved the proximal portion of the DFTS, were orientated longitudinally and generally extended from beneath the MF to terminate within or distal to the sesamoidean canal (Fig 3). Shorter tears were found proximal to (n = 8), within (n = 3) or distal to (n = 10) the sesamoidean canal. Tears varied in location around the tendon, but proximal tears were predominantly lateral (n = 20) with the remaining tears being medial (n = 5), palmar (n = 2), dorsal (n = 1) or unrecorded (n = 5). Tears further distad affected the lateral (n = 3), medial (n = 3),

### Table 2: Tenoscopic diagnoses in 77 digital flexor tendon sheaths

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marginal tear DDFT</td>
<td>45</td>
</tr>
<tr>
<td>Marginal tear DDFT only</td>
<td>37</td>
</tr>
<tr>
<td>Marginal tear + other*</td>
<td>8</td>
</tr>
<tr>
<td>Torn MF</td>
<td>23</td>
</tr>
<tr>
<td>Torn MF only</td>
<td>14</td>
</tr>
<tr>
<td>Torn MF + other*</td>
<td>9</td>
</tr>
<tr>
<td>SDFT tear</td>
<td>11</td>
</tr>
<tr>
<td>Marginal tear SDFT only</td>
<td>5</td>
</tr>
<tr>
<td>Marginal tear + other*</td>
<td>2</td>
</tr>
<tr>
<td>Torn medial branch only</td>
<td>3</td>
</tr>
<tr>
<td>Torn medial branch + other*</td>
<td>1</td>
</tr>
<tr>
<td>Sheath tear</td>
<td>5</td>
</tr>
<tr>
<td>Sheath tear only</td>
<td>1</td>
</tr>
<tr>
<td>Sheath tear + other*</td>
<td>4</td>
</tr>
<tr>
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</tr>
<tr>
<td>Torn lateral plica DDFT</td>
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</tbody>
</table>

*See text for details.

Fig 2: Variations in the tenoscopic appearance of marginal tears of the deep digital flexor tendon (DDFT). a) A bundle of torn fibrils (T) from the medial margin of the DDFT emerging from beneath the manica flexoria (MF). b) A mass of disrupted fibrils (T) emerging from the lateral margin of the DDFT within the sesamoidean canal. c) Multiple strands of disrupted tendon fibrils (T) at the distal margin of a defect in the lateral border of the DDFT. PS = lateral proximal sesamoid bone.
Fig 3: Tenoscopic appearance of a long marginal tear of the lateral margin of the deep digital flexor tendon (DDFT). a) Torn tendon fibrils are seen protruding into the sheath lumen. b) The arthroscope is rotated onto the lateral margin of the DDFT to view into the depth of the cleft. MF = manica flexoria; SW = dorsal sheath wall.

Fig 4: a) and b) Tenoscopic appearance of granulomata (G) associated with the distal limit of marginal tears of the deep digital flexor tendon (DDFT). S = proliferative synovium.

Fig 5: Tenoscopic appearance of a complete tear of the manica flexoria (MF) from the medial margin of the superficial digital flexor tendon. a) The MF is grossly thickened with areas of haemorrhagic staining. b) The torn free margin has recoiled proximally and adhered (A) to the sheath wall (SW). c) Appearance of the proximal sheath reflection (P) dorsal to the deep digital flexor tendon (DDFT) following removal of the MF. d) Gross appearance of the excised MF (cm).
Noninfected tenosynovitis of the digital flexor tendon sheath

dorsal (n = 2) and palmar/plantar (n = 5) aspects of the tendon. The depth of tears varied from surface fibrillation to large clefts extending into the centre of the tendon. In one case, an intact epitenon was identified overlying the tear. Masses of torn tendon fibrils capped with granulation tissue, with varying degrees of organisation (granulomata) (Fig 4), were identified at the proximal and/or distal margins of 12 tears (11 DFTSs) and in 3 cases covered focal tears entirely. The granulomata varied in size and appearance, but frequently contained areas of haemosiderin staining and bundles of torn tendon fibrils.

Tears of the MF were identified in 23 DFTSs; 17 in hind- and 6 in forelimbs. Tears of the MF occurred alone and in combination with marginal tears of the DDFT (n = 5) and SDFT (n = 2), DFTS wall tears (n = 3) and in one case contusion of the DDFT. In all cases, tears occurred at or adjacent to the SDFT with torn strands of collagenous tissue visible extruding along the torn edge. In 18 cases there was complete separation of the MF from the tendon on one side (Fig 5), and in 5 cases tears were partial (Fig 6). Partial tears most commonly affected the distal free border of the MF, but in one case a tear was identified at the proximal margin adjacent to its attachment with the proximal DFTS. Torn MF were usually thickened with areas of haemorrhage or haemosiderin staining. In 8 cases the torn MF had adhered to the DFTS wall (Fig 5b) and in 6 of these this had occurred following reflection of a completely torn MF to the opposite side of the DFTS. Tears affected both lateral (n = 6) and medial (n = 12) attachments; the side was not recorded in 5 cases.

Eleven cases had marginal tears of the SDFT. Seven of these were proximal to the bifurcation of the tendon; 4 involved the medial and 3 the lateral margin of the tendon. Five tears were proximal to, one within and one distal to the sesamoidean canal. Three tears were long and 4 short; 2 of the latter had associated granulomata. The 4 cases with tears distal to the bifurcation all affected the medial branch of insertion and 3 of these had associated granuloma formation. Tears occurred alone or in combination with other lesions (n = 3), as described above. In one case a tear of a branch of the SDFT was seen in combination with tearing of the DFTS wall.

Ultrasoundography predicted accurately the lesions identified at tenoscopy in 35 of 72 (49%) horses. Marginal tears of the DDFT were predicted with a sensitivity of 71%, specificity 71%, positive predictive value (PPV) 71% and negative predictive value (NPV) 55%. Tears of the MF were predicted with a sensitivity of 38%, specificity 92%, PPV 67% and NPV 78%. In 4 cases ultrasonographic abnormalities were recorded on the contralateral side to the tear in the MF. In these cases, tenoscopy revealed that the torn MF had displaced from the side of the tear, apparently hinging on its intact side.

### Treatment

In DFTSs with torn tissue, treatment aimed to reduce exposure of disrupted tissue to the synovial environment. In 65 cases treatment was performed using tenoscopic techniques only, with instrument portals sited according to lesion location. Large masses of torn tendon tissue were dissected free with arthroscopic scissors or meniscectomy knives before removal with Ferris-Smith arthroscopic rongeurs. Tendonous defects were also debrided with a motorised synovial resector (Fig 7) in an oscillating mode with suction applied. When necessary, arthroscopic and instrument portals were interchanged to optimise accessibility of lesions for assessment and treatment. Access to tears of the DDFT which commenced beneath the MF were debrided and the MF was removed in its entirety when one margin was

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**TABLE 3: Results of the logistic regression model comparing horses which returned to preinjury performance with those with reduced performance**

<table>
<thead>
<tr>
<th>Outcome variable</th>
<th>Predictor variable</th>
<th>No. cases</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>P value</th>
<th>Maximum likelihood test P value</th>
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<td>Return to preinjury levels of performance</td>
<td>Marginal tear DDFT</td>
<td></td>
<td></td>
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<td></td>
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<td>Present</td>
<td>44</td>
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<tr>
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<td>Long</td>
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<td>1.162–39.200</td>
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<tr>
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<td></td>
<td>Performed</td>
<td>7</td>
<td>9.000</td>
<td>1.006–80.522</td>
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<tr>
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<td>&gt;15 weeks</td>
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<td>5.657</td>
<td>1.416–22.602</td>
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</table>

CI = Confidence interval.
disrupted completely. The opposite, intact margin was divided from the SDFT using arthroscopic scissors and/or meniscectomy knives. Occasionally, a second instrument was utilised concurrently to grasp and stabilise the torn MF in order to aid division. All tenoscopy skin portals were closed with simple interrupted sutures of monofilament polyamide (Ethilon).

Following tenoscopic evaluation, 11 cases (including the bilaterally afflicted individual) were approached using open surgical techniques; in 8 cases (9 DFTSs) tears of the DDFT (n = 7), SDFT (n = 1) and MF and DDFT (n = 1) were repaired. To facilitate repair, the DFTS was opened through a linear incision from the proximal DFTS reflection to a point just distal to the PAL (Wright and McMahon 1999). The MF also was divided in 6 cases to provide access to DDFT tears. Early in the series, 2 tears (1 DDFT, 1 SDFT) were considered incompletely accessible tenoscopically and open surgery was elected to allow complete debridement. The tear of the SDFT was approached as described above; the DDFT lesion was a short palmar tear which was approached through the proximal linear incision distal to the PAL. In the remaining case, a MF completely torn from its medial attachment was removed. Repair of torn DDFT, SDFT and MF was performed with simple continuous sutures of 2 or 3 metric polyglactin 910 (Vicryl). Wound closure involved repair of sectioned MF, PALs and sheath walls with simple continuous sutures of 3 metric polygactin 910. This was followed by a subcuticular layer of the same material and by stainless steel staples in the skin (Precise).

At the end of surgery, counter-pressure was applied to all operated limbs by multilayered cottonwool bandages. Horses which underwent tenoscopy only received perioperative sodium benzyl penicillin (30000 iu/kg bwt i.v.) (Crystapen); those that underwent subsequent open surgery received sodium benzyl penicillin (30000 iu/kg bwt i.v. q. 8 h) and gentamicin sulphate (2.2 mg/kg bwt i.v. q. 8 h) (Genta 100) for between 2 and 5 days post operatively. All horses received phenylbutazone (4 mg/kg bwt i.v.) (Equipalazone) immediately before surgery and, in 8 animals, administration was continued for 1–10 days post operatively. Skin sutures or staples were removed between 10 and 14 days post operatively and bandages were maintained for 2–4 weeks after surgery. Following suture or staple removal all horses began a gradually ascending controlled exercise regime. This varied according to the severity of the lesion identified, surgery performed and the clinical response of individual horses. However, all animals received a minimum of 6 weeks hand or horsewalker exercise and a similar period of ridden walking and trotting exercise. Horses which were sound returned to normal working regimes 3–18 (mean 7.4) months post operatively.

Follow-up

Follow-up information of 26 (range 6–64; mean 21) months post surgery was available for 61 horses, detailing various outcome measures (60 soundness; 57 performance; 52 distension). Forty-one horses (68%) were sound and 31 (54%) had returned to a level of work equal to or better than before the onset of lameness. A further 4 horses (7%) were sound on return to work, but subsequently suffered recurrence of clinical signs and were retired. One horse which initially suffered a tear of the SDFT, MF and DFTS wall returned to work successfully for 6 months, but reinjured the DFTS 12 months after surgery. Repeated tenoscopy revealed a long deep tear of the lateral margin of the DDFT which was repaired by an open surgical technique, but the horse remained lame. Distension of the DFTS was reduced in 36 DFTSs (69%), including 17 (33%) in which it was resolved.

Results of statistical analysis are displayed in Table 3. Marginal tears of the DDFT and open surgical repair of DDFT lesions were associated with reduced post operative performance. In addition, long tears were associated with reduced post operative performance when compared with short tears (25% and 69% respectively returned to work at their previous levels). Marginal tears of the DDFT were also associated with post operative lameness and absence of improvement in DFTS distension. Only 14 of 33 horses (42%) with marginal tears of the DDFT returned to their previous level of performance. This contrasts to those with tears of the MF, of which 10 of 15 (67%) horses returned to preinjury levels of performance. Of these, 14 horses were treated by removal of the MF; 12 became sound and 10 returned to preinjury levels of performance. Marked preoperative distension was associated with both reduced levels of post operative performance and no improvement in post operative distension. Presence of clinical signs for >15 weeks prior to surgery was also associated with persistent post operative distension. No association with outcome measures was found between age, breed, gender, use, historical information, affected limb, degree of lameness, radiographical and ultrasonographical abnormalities, complications or period of convalescence.

Discussion

No breed, gender, age or use predispositions were identified in this series but, subjectively, young animals and racing Thoroughbreds were under-represented compared with the general clinic caseload. All affected horses had been in regular work and none had a history of trauma, which suggests that the lesions identified may have an exercise-related aetiology.

As reported by Wright and McMahon (1999) and Wilderjans et al. (2003), marginal tears of the lateral aspect of the DDFT were the most frequently identified lesions. However, in the present series, a range of other lesions were also identified. Wilderjans et al. (2003) observed longitudinal tears of the DDFT to be the cause of noninfected tenosynovitis of the DFTS in 17 of 25 (68%) horses. In the current series, 55 of 76 (72%) cases were attributable to tendon tears, with 45 (59%) involving the DDFT. Tenosynovial masses as described by Fortier et al. (1999) and Nixon (2003) were not identified in the current series; although granulomata were present at the proximal and distal limits of 27% of the marginal tears described. Compared with other reports (Fortier et al. 1999; Wilderjans et al. 2003) true intrasynovial adhesions were uncommon in the current series; adherence of torn tendonous tissue to the DFTS wall was more frequently identified.

The morphology of the lesions identified varied widely. The reasons for this and for differences in the location of the lesions are not known. The proximodistal position of tears was described with respect to the semiflexed limb position at tenoscopy and is relevant to this position only.

The ultrasonographic accuracy of lesion prediction was poor. Ultrasonography was most useful in predicting marginal tears of the DDFT. A similar observation was made by Wilderjans et al. (2003), who reported supporting evidence of longitudinal tears of
Noninfected tenosynovitis of the digital flexor tendon sheath

the DDFT in 11 of 17 (65%) cases but specific changes in only 6 (35%) animals. Tears of the MF were frequently not identified with ultrasonography, but the PPV remained similar to marginal tears of the DDFT.

There was no overall difference in the prevalence of lesions in fore- or hindlimbs. However, marginal tears of the DDFT were identified in a greater number of fore- than hindlimbs and tears of the MF were more frequent in hind- than forelimbs. In a series of 17 longitudinal tears of the DDFT, Wilderjans et al. (2003) also noted a greater prevalence in fore- than hindlimbs. The reasons for these differences are not understood, but may suggest differing pathogenesis of the 2 lesions.

Exposed torn collagenous tissue appears to be a source of irritation to the synovial membrane (Wright and McMahon 1999). In the absence of effective intrinsic mechanisms for repair or removal, surgical intervention is considered logical. In early cases some severe lesions were repaired based on the observation that intrinsic tendon repair mechanisms, derived primarily from the epitenon, promoted tendon healing following surgical repair in experimental conditions (Gelberman et al. 1983). However, as reported by Wright and McMahon (1999), the results obtained with tendon repair in this study were inferior to those of tenoscopic debridement. Therefore, all lesions were excised and/or debrided tenoscopically with a view to optimising conditions for second intention healing.

Nixon (2003) hypothesised that the pathogenesis of complex tenosynovitis involves inflammation and subsequent fibrous thickening of the DFTS and PAL. Desmotomy of the PAL has therefore been advocated in the treatment of complex tenosynovitis of the DFTS and has been used with varying success for the treatment of annular ligament syndrome regardless of and without specific treatment of the primary cause (Gerring and Webbon 1984; Barr et al. 1995). In addition, Wilderjans et al. (2003) reported that transection of the PAL also improved tenoscopic visualisation of the DFTS, although these authors no longer consider transection of the PAL to contribute to case management (H. Wilderjans, personal communication). In the current series, it was theorised that intrasynovial tendon lesions caused the tenosynovitis and that management of these would result in resolution of clinical signs. The PAL was only considered contributory when thickened. Therefore, desmotomy of the PAL was performed in only 2 cases in which the PAL subjectively appeared thickened with a loss of compliance when probed at tenoscopy.

The overall prognosis for horses following tenoscopy is reasonable, although the outlook for animals with marginal tears of the DDFT appears to be worse than for other lesions. This may be useful preoperative information when there is ultrasonographic evidence of such lesions. However, the predictability of ultrasonography in this series indicates that caution should be exercised when assessing ultrasonograms for the presence of marginal tears of the DDFT, SDFT and MF. In addition, longer tears appear to carry a worse outlook than shorter tears. The relationship between duration of clinical signs and outcome is predictable and supports early intervention. Marked preoperative distension may reflect the severity of synovial insult and explain its association with outcome. Persistence of post operative distension with marginal tears of the DDFT may indicate incomplete healing, and in turn contribute to their worse outcome.
The results of this study indicate that tenosynovitis of the DFTS may result from tears in the DDFT, SDFT, MF or other structures in the synovial environment. These lesions are not predicted reliably by ultrasonography, and tenoscopy is necessary to identify accurately their presence and morphology. Treatment by tenoscopic debridement is appropriate and can offer a reasonable prognosis for the majority of animals with noninfected tenosynovitis of the DFTS.

Acknowledgements

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Manufacturers’ addresses

1 Karl Storz GmbH & Co., Tuttlingen, Germany.
2 Scanlan, St Paul, Minnesota, USA.
3 Dyonics/Smith & Nephew Endoscopy Inc., Andover, Massachusetts, USA.
4 Ethicon, Edinburgh, Midlothian, UK.
5 J&M Health Care, St Paul, Minnesota, USA.
6 Pitman-Moore Ltd, Crewe, Cheshire, UK.
7 CP-Pharma, Burgdorf, Germany.
8 Arnolds Veterinary Products, Shrewsbury, Shropshire, UK.

References


General Articles

Endoscopic assessment and treatment of lesions of the deep digital flexor tendon in the navicular bursae of 20 lame horses

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Keywords: horse; navicular bursa; bursoscopy; deep digital flexor tendon

Summary

Reasons for performing study: Clinical lesions of the deep digital flexor tendon and navicular bone are being reported with increasing frequency. However, the role of direct visualisation by navicular bursoscopy in the diagnosis and management of such injuries has not been explored.

Hypothesis: Navicular bursoscopy: 1) corroborates information obtained from other, noninvasive imaging modalities; 2) allows direct visualisation of lesions unidentified by other diagnostic modalities; 3) provides further information on morphology of lesions; and 4) permits minimally invasive surgical access to lesions.

Methods: The case records of all horses that underwent diagnostic navicular bursoscopy for the investigation of lameness admitted to 2 referral clinics (the Royal Veterinary College and Reynolds House Referrals) were evaluated retrospectively. Follow-up information was obtained by telephone questionnaire.

Results: Twenty-three bursae were examined endoscopically in 20 horses. Tears of the deep digital flexor tendon were seen in all horses (22 bursae). In 8 bursae, cartilage lesions were also present and in one bursa this was the only abnormal finding. Computed tomography and low field magnetic resonance imaging predicted tendon lesions in most cases, but failed to identify cartilage damage. Greater than 6 month follow-up information was available for 15 animals of which 11 were sound and 9 had returned to preoperative levels of performance.

Conclusion: Lameness localised to the foot may result from tears of the deep digital flexor tendon and/or navicular fibrocartilage loss. Navicular bursoscopy allows comprehensive evaluation of these changes and also permits appropriate lesion management.

Potential relevance: The diagnostic information obtained from and therapeutic options offered by bursoscopy justify its use in horses with clinical findings localising lameness to the navicular bursa.

Introduction

The navicular bursa is a closed synovial sac interposed between the deep digital flexor tendon (DDFT) and navicular bone (Sisson 1975). Its dorsal margins are, from distal to proximal the distal sesamoidean impar ligament, palmar/plantar surface of the navicular bone, collateral sesamoidean ligaments and intervening T-ligament. The latter is thin and consists of little more than the fibrous capsules of the distal interphalangeal joint, navicular bursa and digital flexor tendon sheath. The dorsal surface of the DDFT forms the palmar/plantar margin of the bursa.

Wright et al. (1998) examined post mortem the navicular region of 38 clinical cases of navicular disease. Intrabursal lesions were present in all limbs and included, in decreasing frequency of occurrence, DDFT surface fibrillation, partial thickness loss of the fibrocartilage of the navicular bone, distal border fragmentation of the navicular bone, torn DDFT fibres adhered to the navicular bone, full thickness loss of the fibrocartilage of the navicular bone, palmar cortex erosions and DDFT core lesions. The limitations of radiography for the assessment of navicular disease were highlighted, with the 2 most common lesions being undetectable radiographically. In addition, in one limb a DDFT core lesion was seen in the absence of changes affecting the navicular bone or other adjacent structures, adding further support to the concept that navicular disease may be present in the absence of radiological abnormalities. These findings prompted investigation into alternative in vivo imaging modalities. Whitton et al. (1998) identified DDFT tendonitis within the digit using both magnetic resonance imaging (MRI) and computed tomography (CT), verified using gross inspection and histopathology. Subsequently there have been clinical reports of lesions in the navicular apparatus identified by CT and MRI (Teitje et al., 2001; Dyson et al. 2003a,b, 2005) and, recently, correlative studies to histopathological findings in horses with foot pain have been performed demonstrating good agreement for lesions of the DDFT and moderate to good agreement for abnormalities of the palmar surface of the navicular bone (Murray et al., 2006).

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Without accurate characterisation of the pathology in clinical cases of navicular disease, reports of outcome have been limited to groups of horses with a variety of lesions. More recently, high field MRI has enabled follow-up of horses with primary DDFT injuries within the digit, and for those with combined injuries of the DDFT and navicular bone. Treated conservatively, only 28% and 5% of cases, respectively, returned to full work (Dyson et al. 2005). Endoscopy of the navicular bursa was first reported by Wright et al. (1999) for treatment of contamined and infected bursae. The surgical technique for navicular bursal endoscopy and a transthecal approach have been described by McIlwraith et al. (2005). However, to date there has been little information reported on the potential benefits to management of nonpenetrating lesions.

The objectives here were to describe the clinical features and endoscopic findings within the navicular bursae of 20 lame horses. The authors hypothesised that navicular bursography: 1) subjectively corroborates the information obtained from other noninvasive imaging modalities; 2) allows direct visualisation of lesions unidentified by other diagnostic modalities; 3) provides further information on the morphology of lesions previously identified by other imaging techniques; and 4) permits minimally invasive surgical access to and treatment of lesions.

Materials and methods

Database

Retrospective analysis of case records of 20 lame horses that underwent diagnostic navicular bursal endoscopy at the Royal Veterinary College (n = 11) and Reynolds House Referrals (n = 9) was performed. Horses were selected for endoscopy by localisation of pain to the navicular region by diagnostic analgesia, in combination with either supporting CT findings, low field MRI, ultrasonography or a consistent pain response with digital compression over the distal portion of the DDFT. Data were collected for each horse to include age, breed, gender, use, duration of clinical signs and treatments administered prior to admission.

Diagnostic procedures

All horses were subjected to a lameness examination including palpation of the limbs and gait evaluation at a walk and trot. Lameness was graded out of 10 (0 = not lame to 10 = nonweightbearing; Ross 2004). All horses underwent diagnostic analgesia with mepivacaine hydrochloride (Intra-Epicaïne) consisting of various combinations of perineural analgesia of the palmar digital nerves, perineural analgesia of the palmar nerves at the level of the proximal sesamoid bones, intra-articular analgesia of the distal interphalangeal (DIP) joint (5 ml) and intrabursal analgesia of the navicular bursa (3 ml with 2 ml of radiopaque contrast material [Omnipaque] to confirm intrabursal injection).

All horses underwent radiographic examination of both affected and contralateral feet including lateromedial, 2 dorsoproximal-palmarodistal obliques (one positioned and exposed for evaluation of the navicular bone and one for evaluation of the distal phalanx), and palmaroproximal-palmarodistal oblique images (Butler et al. 2000). Ultrasonography of the DDFT was carried out only from the palmar aspect of the pastern (Sage and Turner 2002) in 13 horses using a 10 MHz linear array probe (GE Healthcare). Nuclear scintigraphic examination of both front feet using dorsal and lateral pool phase, and dorsal, lateral and solar bone phase images was performed in one horse using Hermes imaging software (Dyson 2002). Computed tomography examination of both front feet was performed in 6 horses under general anaesthesia using a 4th generation helical scanner; an axial scan was performed using parameters of 120 kV, 150 MA and a 2 sec scan time. Slice thickness was set at 4 mm and a pitch of 2 mm. The data set was then reconstructed using a reconstruction index of 4 mm to give transverse slices. Magnetic resonance imaging of both front feet was performed in 4 standing horses as described by Mair et al. (2005) using a low field 0.27 Tesla magnet scanner. All radiographs, ultrasonograms and CT images were interpreted retrospectively by M.R.W.S. All MRI images were interpreted by a clinician (T.S.M.) experienced with the low field MRI unit.

Endoscopic examination

Endoscopy of the navicular bursa was performed in all horses under general anaesthesia in dorsal recumbency. In 4 animals, this was performed bilaterally. In 8 cases the limb was positioned close to vertical with the distal limb in a semi-flexed position and arthroscopic manipulations performed with the surgeon standing behind the limb. In all other cases, the limb was positioned with the carpus flexed at approximately 90° and the distal limb in a semi-flexed position, and arthroscopic manipulations performed with the surgeon standing in front of the limb. In all cases endoscopy was performed using a 4 mm 25° forward oblique arthroscope. In 14 cases endoscopic examination was performed as described by Wright et al. (1999); and, in 6 cases a transthecal portal was made at the dorsal margin of the DDFT through the distal reflection of the digital flexor tendon sheath/palmar surface of the T ligament (McIlwraith et al. 2005). In all cases a contralateral instrument portal was used to access lesions. Lesions were palpated with an arthroscopic probe. Large bundles of torn tendon tissue were sectioned with arthroscopic scissors before removal with Ferris Smith arthroscopic Rongeurs. The defects remaining and smaller tendonous lesions were debrided with a motorised synovial resector in an oscillating mode with suction applied. Fibrillated fibrocartilage was also debrided using a motorised synovial resector. Full thickness lesions in the navicular fibrocartilage were debrided with arthroscopic curettes to the level of firm subchondral bone. When necessary, arthroscope and instrument portals were interchanged to optimise accessibility of lesions for assessment and treatment. At the end of surgery, bursae were lavaged and skin incisions were closed with simple interrupted sutures of 3 Metric monofilament polyamide. The limbs were then protected with sterile dressings.

All surgery at the Royal Veterinary College was performed by M.R.W.S. and R.K.W.S., and all surgery at Reynolds House Referrals by I.M.W. Images taken during surgery for all cases were also reviewed by M.R.W.S. to ensure interobserver agreement in lesion interpretation.

Additional surgical procedures

Navicular suspensory desmotomy was performed as described by Wright (1993) in 2 cases (13 and 16). Distal interphalangeal joint arthroscopy (McIlwraith et al. 2005) was performed in 2 cases (5 and 12), and tenoscopic examination of the digital flexor tendon sheath (McIlwraith et al. 2005) in one horse (Case 3).
Post operative care

Perioperative antimicrobial prophylaxis consisted of 20,000–30,000 iu/kg bwt penicillin i.v. (Crystapen)\textsuperscript{13} or i.m. (Norocillin)\textsuperscript{14}, and analgesia with a single pre-operative dose of phenylbutazone (EquiPalazone)\textsuperscript{1} (4 mg/kg bwt) or flunixin meglumine (Finadyne)\textsuperscript{14} (1.1 mg/kg bwt). Post operatively, dressings were maintained until 14 days after surgery, at which point the sutures were also removed. All horses were confined to their stables while receiving increasing amounts of hand-walking or horse-walker exercise b.i.d. for a minimum of 2 months. Horses typically then received a further month of ridden walking exercise. At the end of this period, if the horse was sound at a trot, 2 months of gradually increasing amounts of trotting exercise was recommended. If the horse was still sound at the end of this period then a gradual re-introduction to work was attempted.

Follow-up information was obtained by re-examination (n = 4) and/or telephone questionnaire from owners (n = 20). This detailed complications, current degree of lameness, current activity and when appropriate time to return to work.

Results

Horses were 6–17 (mean 11) years and included Thoroughbreds (n = 1), ponies (n = 3) and sports horses (n = 15) with one unknown. Genders were represented equally between mares (n = 10) and geldings (n = 10). Horses were used for a variety of purposes; eventing (n = 5), dressage (n = 2), show jumping (n = 2), hunting (n = 2), endurance (n = 1) and general purpose (n = 7) with one unknown.

Case histories

The horses had been lame for 1–9 months (mean 4 months) prior to examination. In 16 horses the onset of lameness was acute and of moderate to severe intensity. In one horse, lameness was insidious in onset but acutely exacerbated by work, but only in 2 horses was lameness intermittent and insidious in onset (1 unknown). Prior to admission, horses had received a variety of treatments consisting of combinations of rest (n = 20), remedial farriery (n = 9), oral phenylbutazone (n = 4), isoxsuprine hydrochloride (n = 2) and intramuscular polysulphated glycosaminoglycan mediation (n = 1). Distal interphalangeal joints had been medicated on one (n = 8) or multiple (n = 2) occasions with triamcinolone acetonide (n = 5), hyaluronan (n = 1), triamcinolone acetonide and hyaluronan (n = 4), and polysulphated glycosaminoglycan (n = 2). The navicular bursa of one horse had been medicated once with triamcinolone acetonide.

Case diagnoses

Forelimbs were affected in 19 horses (5 bilaterally) and in one a hindlimb was involved. On presentation all animals were lame when trotted in a straight line on a hard surface (range 1–9 out of 10; median 3). Lameness was exacerbated when the horse was lunged on a hard surface with the more severely affected limb on the inside of the circle (n = 14/14). Other common clinical findings included distension of the DIP joint (n = 9), thickening (n = 6) and pain (n = 6) on palpation of the distal DDFT in the palmar pastern, pointing of the affected limb at rest (n = 8) and a reduced caudal phase to the stride (n = 5).

Palmar digital analgesia of the more severely affected limb produced an improvement in lameness in 18/18 cases; improvement was slight (n = 1), partial (n = 9) or complete (n = 8). Residual lameness was abolished by analgesia of the palmar nerves at the level of the proximal sesamoid bones in all except one horse, which had concurrent tenosynovitis of the digital flexor tendon sheath. Intra-articular analgesia of the DIP joint was

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Radiography</th>
<th>Ultrasonography</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Irregular palmar cortex navicular bone</td>
<td>NAD</td>
</tr>
<tr>
<td>2</td>
<td>LF: irregular palmar cortex and radiolucent area within sagittal ridge navicular bone</td>
<td>LF: increased fluid within navicular bursa RF: NAD</td>
</tr>
<tr>
<td>3</td>
<td>NAD</td>
<td>RF: NAD</td>
</tr>
<tr>
<td>4</td>
<td>NAD</td>
<td>Enlarged medial lobe DDFT with irregular margin, increased fluid within navicular bursa</td>
</tr>
<tr>
<td>5</td>
<td>Extensor process fragmentation P3; irregular palmar cortex and large radiolucent zone with surrounding ‘sclerosis’ in medulla of navicular bone</td>
<td>NP</td>
</tr>
<tr>
<td>6</td>
<td>NAD</td>
<td>NP</td>
</tr>
<tr>
<td>7</td>
<td>NAD</td>
<td>Enlarged lateral lobe DDFT</td>
</tr>
<tr>
<td>8</td>
<td>NAD</td>
<td>NAD</td>
</tr>
<tr>
<td>9</td>
<td>Poor corticomedullary demarcation and (re)modelling wings navicular bone</td>
<td>Irregular palmar margin DDFT</td>
</tr>
<tr>
<td>10</td>
<td>Fragment distal lateral border navicular bone</td>
<td>Enlarged medial lobe DDFT and irregular medial abaxial margin, peritendinous thickening</td>
</tr>
<tr>
<td>11</td>
<td>NAD</td>
<td>Enlarged medial lobe DDFT</td>
</tr>
<tr>
<td>12</td>
<td>NAD</td>
<td>NP</td>
</tr>
<tr>
<td>13</td>
<td>Large radiolucent zone with surrounding ‘sclerosis’ medulla navicular bone, radiolucent defect in adjacent palmar cortex and sagittal ridge, irregular palmar surface</td>
<td>NAD</td>
</tr>
<tr>
<td>14</td>
<td>LF: (re)modelling wings navicular bone</td>
<td>NP</td>
</tr>
<tr>
<td>15</td>
<td>NAD</td>
<td>NAD</td>
</tr>
<tr>
<td>16</td>
<td>New bone proximal border navicular bone</td>
<td>NAD</td>
</tr>
<tr>
<td>17</td>
<td>Thickened palmar cortex navicular bone, new bone at impar ligament insertion</td>
<td>Thicken DDT, increased fluid within navicular bursa</td>
</tr>
<tr>
<td>18</td>
<td>NAD</td>
<td>NAD</td>
</tr>
<tr>
<td>19</td>
<td>NAD</td>
<td>NAD</td>
</tr>
<tr>
<td>20</td>
<td>NAD</td>
<td>NAD</td>
</tr>
</tbody>
</table>

NAD = no abnormality detected; NP = not performed; LF = left fore; RF = right fore; P3 = distal phalanx; DDFT = deep digital flexor tendon.
performed in all horses producing an improvement in lameness in 17 (slight n = 3; partial n = 7; complete n = 7). Intrathecal analgesia of the navicular bursa (n = 15) produced a partial (n = 10) or complete (n = 5) improvement in lameness. Comparing palmar digital, navicular bursal and distal interphalangeal joint analgesia (n = 15), the best response was seen with bursal analgesia in 6 cases, palmar digital analgesia in 6 cases and in 3 cases the horse was sound following both bursal and palmar digital analgesia. In all but one case (where DIP analgesia also rendered one of the latter horses sound) DIP joint analgesia was less positive than both bursal and palmar digital analgesia.

**Diagnostic imaging and bursoscopy:** Results of diagnostic imaging and bursoscopic findings are displayed in Tables 1 and 2. Twenty-three bursae were examined endoscopically in 20 horses. DDFT lesions were seen in all horses (22 bursae). In 20 bursae, tears of the DDFT were seen as torn collagenous tissue, displaying birefringency, extruded into the bursal lumen (Fig 1a,b). Morphologically, tears most frequently were longitudinally orientated, with a torn flap of tendon tissue hinged proximally in the bursa. In 4 bursae with torn DDFT and in one additional bursa (as the sole finding), a focal granulomatous lesion was identified on the surface of the DDFT that, when removed, was found to consist of torn tendon fibrils with a cap of granulation tissue.

In one horse (one bursa) the dorsal surface of the DDFT was intact, but this was soft and readily penetrated with an arthroscopic probe to reveal underlying torn tendinous tissue. In the remaining bursa a small partial thickness fibrocartilage lesion was the only abnormal finding. Of the bursae with DDFT lesions, fibrocartilage lesions were identified in 8 bursae, of which 4 were full thickness (Fig 2) and 4 partial thickness (Fig 3). Torn and disrupted tissue within bursae frequently limited visibility such that the full extent of lesions could not be assessed confidently until removal was complete. In addition, in 5 cases (3, 4, 8, 10 and 14) the most distal margins of lesions could not be visualised with sufficient confidence to ensure that all disrupted tissue had been removed. The authors considered that movement of arthroscope and instruments within affected bursae was often more restricted than in normal bursae and those that suffered acute penetrating injuries. When attempting to debride DDFT tears in the distal aspect of the bursa, iatrogenic partial thickness excoriation of the fibrocartilage of the navicular bone was produced in only 3 cases.

**Arthroscopy:** Of the 2 cases that underwent distal interphalangeal joint arthroscopy, one had fragmentation of the extensor process of the distal phalanx that was removed, and one had minor partial thickness cartilage lesions that were not debrided. The case that underwent a digital flexor tendon sheath tenoscopy had a tear of the lateral margin of the DDFT in the digital flexor tendon sheath and this was debrided.

**Radiography and ultrasonography:** These techniques revealed abnormalities of the navicular apparatus in 9/20 and 7/15 horses respectively (Table 1). However, neither modality was able to predict subsequent endoscopic findings. The case examined scintigraphically had an increase in uptake of radiopharmaceutical in the region of the DDFT at the level of the navicular bone during the pool and bone phase, and an increased uptake in the region of the DDFT insertion in the bone phase. Endoscopically identified DDFT lesions were identified by CT in 6/7 limbs (Fig 4), although in one limb a smaller area of disruption of the bursal surface of the

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**Fig 1:** Endoscopic view of a bundle of torn fibrils (T) extruding from the DDFT a) case 19 b) case 9. N = flexor surface of the navicular bone; P = arthroscopic probe.

**Fig 2:** Endoscopic appearance of fibrillation (F) of the navicular fibrocartilage seen in case 15. The opposing DDFT has an area of haemosiderin staining.

**Fig 3:** Endoscopic view of a full thickness erosion of fibrocartilage (E) on the palmar surface of the navicular bone in case 2. Torn fibres (T) can be seen on the opposing DDFT.
### TABLE 2: Results of computer tomographic (CT), magnetic resonance imaging (MRI) and navicular bursal endoscopy

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Computed tomography/magnetic resonance imaging</th>
<th>Bursal endoscopy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>NP</td>
<td>Focal tear medial lobe DDFT, small area full thickness fibrocartilage loss medial to sagittal ridge.</td>
</tr>
<tr>
<td>2</td>
<td>MRI</td>
<td>LF: torn lateral lobe DDFT, full thickness fibrocartilage loss. RF: fibrillated surface DDFT, discoloured epitenon, small area partial thickness fibrocartilage loss.</td>
</tr>
<tr>
<td>3</td>
<td>NP</td>
<td>Fibrillated DDFT, full thickness fibrocartilage loss.</td>
</tr>
<tr>
<td>4</td>
<td>CT</td>
<td>LF: core lesion in medial and lateral lobes DDFT from level of bifurcation to mid navicular bone, tissue protruding from both lobes into navicular bursa at distal end of lesion, larger lesion medially. Moderate distension navicular bursa. RF: core lesion medial lobe DDFT from level of PIP joint to proximal navicular bone, lesion extends to dorsal border of DDFT distally. Moderate distension navicular bursa.</td>
</tr>
<tr>
<td>5</td>
<td>NP</td>
<td>Torn DDFT, discoloured synovium.</td>
</tr>
<tr>
<td>6</td>
<td>CT</td>
<td>LF: large area of torn DDFT medial lobe, extends distally out of sight. RF: NP</td>
</tr>
<tr>
<td>7</td>
<td>MRI</td>
<td>Fibrillated DDFT over navicular bone, haemosiderin stained synovium distally adjacent to impar ligament medial to midline and shallow defect in adjacent DDFT with haemosiderin staining.</td>
</tr>
<tr>
<td>8</td>
<td>NP</td>
<td>Torn DDFT with granulomatous end attached by stalk, hyperaemic synovium throughout, small partial thickness fibrocartilage defect proximal navicular bone.</td>
</tr>
<tr>
<td>9</td>
<td>NP</td>
<td>DDFT surface discolouration with tearing medially, extending out of sight distally.</td>
</tr>
<tr>
<td>10</td>
<td>MRI</td>
<td>Haemoglobin staining and torn medial lobe DDFT at site of reflection of bursal wall and T-ligament.</td>
</tr>
<tr>
<td>11</td>
<td>NP</td>
<td>Granuloma capped defect lateral lobe DDFT.</td>
</tr>
<tr>
<td>12</td>
<td>NP</td>
<td>Torn DDFT.</td>
</tr>
<tr>
<td>13</td>
<td>MRI</td>
<td>LF: short tear lateral lobe DDFT at level proximal navicular bone. RF: torn lateral lobe DDFT from proximal navicular bursa to distal navicular bone.</td>
</tr>
<tr>
<td>14</td>
<td>MRI</td>
<td>LF: mild patchy areas decreased signal medulla navicular bone, increased signal dorsal surface lateral lobe DDFT proximal P2 to insertion. RF: mild patchy areas decreased signal medulla navicular bone, increased signal core DDFT from proximal P2 to proximal navicular bone extending to split from dorsal to palmar surfaces with irregular dorsal surface from level proximal navicular bursa to distal navicular bone, dorsalpalmar split in opposing DSIL.</td>
</tr>
<tr>
<td>15</td>
<td>CT</td>
<td>LF: haemosiderin stained synovium medial recess, short tear medial lobe DDFT at level of mid navicular bone, fibrillated fibrocartilage. RF: mild cartilage fibrillation.</td>
</tr>
<tr>
<td>16</td>
<td>CT</td>
<td>LF: NP</td>
</tr>
<tr>
<td>17</td>
<td>NP</td>
<td>RF: Partial thickness fibrocartilage loss lateral to sagittal ridge, fibrillated medial lobe DDFT.</td>
</tr>
<tr>
<td>18</td>
<td>CT</td>
<td>Torn medial lobe DDFT with granulomatous end proximally. Synovial proliferation proximal navicular bursa, haemosiderin staining over medial lobe DDFT, torn medial lobe DDFT from T-ligament to proximal navicular bone with haemoglobin staining distally.</td>
</tr>
</tbody>
</table>

NP = not performed; DDFT = deep digital flexor tendon; SDFT = superficial digital flexor tendon; LF = left fore; RF = right fore; P2 = middle phalanx; PIP = proximal interphalangeal joint; DIP = distal interphalangeal joint.
DDFT was identified endoscopically than predicted by CT. The DDFT lesion that was not identified by CT consisted of fibrillation of the dorsal border of the tendon. This technique failed to predict fibrocartilage lesions identified endoscopically in 4 bursae, although in one there was irregularity of the palmar cortex of the navicular bone. Low field MRI identified endoscopically visualised DDFT lesions in 3/4 bursae (Fig 5), although in one limb disruption of the bursal surface of the DDFT could not be established. Fibrocartilage defects were not detected with low field MRI in either of the 2 bursae with such lesions visualised endoscopically, but in both there were abnormalities of the palmar cortex of the navicular bone. The DDFT lesion that was not identified by low field MRI was fibrillation of the dorsal border of the tendon.

**Follow-up**

There were no post operative complications and at the time of discharge, all horses were walking comfortably. Follow-up information was obtained for all cases at 3 to 84 (mean 17) months after surgery. Five horses were <6 months since surgery and still convalescing. Of the remaining 15 horses, 2 horses (Cases 2 and 6) were sound in work but at a lower level than before surgery, and 9 horses were sound and in full work. Four cases (10, 11, 14 and 16) remained lame; 2 improved compared to before surgery and returned to light work and 2 showed no improvement.

**Discussion**

Endoscopy of the navicular bursa identified lesions in all cases, subjectively corroborating CT and low field MRI predicted DDFT lesions. DDFT lesion morphology within the navicular bursa was better characterised endoscopically and, in addition, fibrocartilage lesions not predicted by other imaging modalities, were visualised. The principal lesion identified endoscopically in all except one case was a defect in the dorsal surface of the DDFT. Radiography is unable to image soft tissues adequately to identify such lesions, although frequently abnormalities of the associated navicular bone are identified. Ultrasonography via the palmar aspect of the pastern is able to image the DDFT distally to the level of the proximal recess of the navicular bursa (Sage and Turner 2002) and, in some cases, lesions extend sufficiently proximad to be recognised through this ultrasonographic window. This technique enabled identification of abnormalities in the navicular apparatus in almost 50% of cases in which it was used, therefore supporting its usefulness in evaluating horses with lameness localised to this area. However, a negative ultrasound examination did not rule out a more distal DDFT lesion. Examination through a transcuneal approach has been described but the window of evaluation of the DDFT is limited to the midline (Sage and Turner 2002). Lesions of the DDFT and navicular bone detected ultrasonographically, through a transcuneal approach, have been reported (Denoix and Audigie 2004), but the lesions identified in this series affected either lobe of the DDFT and not the central portion; and the authors suspected that it would have been difficult to detect these lesions with this technique.

Both CT and low field MRI were accurate in identifying pathology involving the DDFT, although with low field MRI it was difficult to predict disruption of its bursal surface confidently. These modalities, together with ultrasonography, also identified further extra-bursal abnormalities not visible endoscopically, including in 6 limbs (4 cases) proximal extension of intrabursal DDFT tears as core lesions (Table 2). Low field MRI predicted a greater range of abnormalities than CT in these cases, but it was not possible to confirm or refute their presence by endoscopic visualisation. Fibrocartilage lesions were detected endoscopically in 4 cases imaged by CT and 2 cases by low field MRI. Both modalities failed to detect the fibrocartilage lesions, but low field MRI identified changes in the palmar cortex of the navicular bone in both cases and CT in one. Murray et al. (2006) recently correlated irregularities of the chondro-osseous margin of the palmar navicular bone, identified with high field MRI, with fibrocartilage defects seen histologically. In addition, they were able to detect signal changes and defects in the fibrocartilage layer, which also correlated to histology of fibrocartilage defects. It is not known whether the fibrocartilage lesions in this series would have been better defined with high field MRI.

In the present study, arthroscopic and instrument manipulations at the site of lesions were difficult in some cases, although this was made easier by the surgeon operating from in front of the limb. However, the restricted movement of the arthroscope still limited accessibility of lesions distally within the navicular bursa. Utilisation of the transthecal approach (McIlwraith 2005) increased accessibility, although it was still not possible to access the entirety of all lesions. It is possible that chronic inflammation contributes to these problems and supports the case for early surgical intervention.

Localisation of lameness confidently to the navicular bursa can be clinically difficult. However, whenever possible, palmar digital, DIP joint and navicular bursal analgesia were all performed, enabling the most accurate localisation of pain within the foot. The
possibility of peribursal pathology cannot be dismissed, although all cases had intrabursal lesions identified endoscopically and those that underwent CT or low field MRI also had lesions identified that were mostly restricted to the navicular bursa, supporting the clinical localisation of lameness to this site. The 20 horses in the present study represent all cases that had undergone diagnostic endoscopy of the navicular bursa at the 2 referral hospitals and all had lesions identified. Five horses had surgical procedures performed in addition to navicular bursal endoscopy. In these cases the lesions identified endoscopically were considered a significant cause of lameness; and additional surgical procedures were performed as adjunctive therapy. In one of these cases, the successful outcome cannot reasonably be attributed to the endoscopic procedures performed within the navicular bursa, because a distal phalanx extensor process fragment was removed from the DIP joint and a palmar digital neurectomy also performed. However, in the remaining 4 cases the adjunctive procedures were performed to treat pathology exterior to the navicular bursa, and would have had no influence on the resolution of lameness referable to the bursa. Follow-up of 65 horses demonstrated by high field MRI to have either primary DDF tendonitis in the navicular region or combined navicular bone and DDFT lesions, revealed 28% and 5% of cases, respectively, returning to full work when treated conservatively (Dyson et al. 2005). Many of the cases shared similar clinical and diagnostic imaging findings to those in the current series (parasagittal split-like tendon lesions, altered navicular bone MR signal intensity, reduced caudal stride phase, exacerbation of lameness on a hard circle, response to navicular bursal analgesia etc.). The present authors postulated that intrabursal lesions are limiting factors in healing, due to the lack of a functional intrinsic mechanism for removal and repair of torn collagen (McIlwraith 1992; Phillips and Wright 1994; Wright 1995; Wright and McMahon 1999). Endoscopic removal of disrupted tendon tissue was therefore considered logical. Following debridement, 60% of the current series of horses returned to full work, supporting the use of endoscopic surgery. In conclusion, navicular bursal endoscopy is a useful diagnostic and potentially therapeutic option for noninfected bursitis of the navicular bursa; and should be considered for animals with clinical findings localising lameness to the navicular bursa or without supporting ultrasonographic, CT or MRI findings.

Acknowledgements

The authors thank Ehud Eliashar, Justin Perkins and Mike Archer for contributing to the work-up and treatment of 4 cases in this series, and Tim Mair for assistance in MRI image interpretation.

Manufacturers’ addresses

1Arnolds, Shrewsbury, Shropshire, UK.
2Amersham Health, Oslo, Norway.
3GE Healthcare, Chalfont St Giles, Buckinghamshire, UK.
4Nuclear Diagnostics Ltd., Gravesend, Kent, UK.
5Phillips Medical Systems Inc., Ohio, USA.
6Hallman Veterinary Imaging, Guildford, Surrey, UK.
7Karl Storz GmbH & Co., Tuttingen, Germany.
8Wolf Instruments, Hanover, Germany.
9Sontec, Englewood, Colorado, USA.
10Scilcan, St Paul, Minnesota, USA.
11Dyonics/Smith & Nephew Endoscopy Inc., Andover, Massachusetts, USA.
12Ethicon, Edinburgh, UK.
13Pirmann-Moore Ltd, Crewe, Cheshire, UK.
14Norbrook Laboratories Ltd., Newry, County Down, UK.

References


Author contributions All authors contributed to the initiation, planning, conception, pathology, execution and writing of this study. Statistics were by M.R.W.S.
Arthroscopic treatment of fractures of the lateral malleolus of the tibia: 26 cases

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Newmarket Equine Hospital, UK.

Keywords: horse; fracture; lateral malleolus; tibia; arthroscopy

Summary

Reasons for performing the study: A minimally invasive arthroscopic technique for removal of fractures of the lateral malleolus of the tibia is considered to be beneficial but data to this effect are required.

Hypothesis: Arthroscopic removal of fractures of the lateral malleolus of the tibia is technically feasible, provides a comprehensive evaluation of the tarsocrural joint and enables removal of remote comminuted fragments and disrupted short collateral ligaments. The technique is associated with low patient morbidity, requires only short periods of hospitalisation and affords a good prognosis to affected horses.

Methods: The case records of all horses that underwent arthroscopic removal of a fractured lateral malleolus of the tibia, admitted to a referral hospital, were evaluated retrospectively. Follow-up information was obtained from race records and by telephone questionnaire.

Results: Fractures were successfully removed arthroscopically in all cases following dissection from the short lateral collateral ligaments. Significant post operative complications occurred in only one horse. All other horses recovered well from surgery and of 22 horses with long-term follow-up, 18 returned to their previous use.

Conclusion: Arthroscopic removal of fractures of the lateral malleolus of the tibia is technically demanding, but can be performed with minimal complications and with low patient morbidity and short periods of hospitalisation. The majority of horses are able to successfully return to work following the procedure.

Potential relevance: The advantages of arthroscopic removal compared to removal via arthrotomy make this the technique of choice for treatment of fractures of the lateral malleolus of the tibia.

Introduction

The 2 tibial malleoli represent the bones distal extremity medially and laterally. Developmentally, the lateral malleolus forms from a separate centre of ossification (the distal end of the fibula), fusing to the tibia during the first year of life (Getty 1975). The lateral malleolus of the tibia has extensive attachments from both the long and short lateral collateral ligaments, and their anatomy has been previously described in detail (Updike 1984). Superficial to the long lateral collateral ligament and sitting in a groove in its surface, a fascial tunnel houses the lateral digital extensor tendon and its synovial sheath. The long lateral collateral ligament has distal attachments to the talus, calcaneus, fourth tarsal bone and third and fourth metatarsal bones. There are 2 short lateral collateral ligaments, which lie axial to the long lateral collateral ligament and are named according to their respective insertions (the pars tibiotalaris and pars tibiocalcaneae). The pars tibiotalaris can be further divided into superficial and deep components (Updike 1984). Along with the medial collateral ligaments, these confer axial stability to the hock throughout its range of flexion and extension.

The short lateral collateral ligaments lie subsynovially within the tarsocrural joint and can be visualised arthroscopically covering almost the entire articular surface of the lateral malleolus of the tibia (McIlwraith et al. 2005).

Fractures of the lateral malleolus of the tibia have been previously described (Jakovljevic et al. 1982; Wright 1992). As a result of the extensive collateral ligament attachments, distracting forces on the fracture fragment(s) usually result in displacement. When managed conservatively, nonunion with proliferative fibrosis around the fracture and disrupted short collateral ligaments are inevitable. Fragment removal is now accepted as the treatment of choice, and good outcomes have been achieved by surgical treatment via arthrotomy (Wright 1992).

The authors hypothesise that a minimally invasive (arthroscopic) approach to removal should afford a similarly good prognosis, with the advantages of comprehensive evaluation of the tarsocrural joint, ability to remove remote comminuted fragments and disrupted short collateral ligaments, minimal morbidity and short periods of hospitalisation and convalescence.

Materials and methods

Study

Retrospective analysis of case records of all horses admitted to Newmarket Equine Hospital from June 2003 to August 2009 that underwent arthroscopic removal of a fractured lateral malleolus of the tibia, was performed. Fractures were defined by involvement of the full dorsoplantar width of the lateral malleolus of the tibia.

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Data were collected for each horse to include clinical history, age, breed, gender, use and duration of clinical signs.

**Diagnostic procedures**

All horses were subjected to a lameness examination including palpation of the limbs and gait evaluation at a walk and trot. Lameness was graded out of 10 (0 = sound to 10 = nonweightbearing) (Ross 2004).

All horses underwent radiographic examination of the affected tarsus including dorsoplantar, lateromedial, dorsolateral-plantaromedial oblique and dorsomedial-plantarolateral oblique images (Butler et al. 2008). In selected cases, dorsal (10°) lateral-plantaromedial oblique and dorsal (10°) medial-plantarolateral oblique images were obtained to further define the location and extent of the fracture (Wright 1992). Ultrasonography of the tarsocural joint and collateral ligaments was carried out in 9 horses using a 10 MHz linear array probe.

**Surgical technique**

Horses were operated under general anaesthesia in dorsal recumbency, with the affected limb suspended and retracted caudally, with a tarsal angle of approximately 130°. An Esmarch bandage and tourniquet were applied to the proximal crus. Arthroscopic manipulations were performed with the surgeon standing on the medial aspect of the limb and facing across the leg. In all cases, arthroscopy was performed using a 4 mm 25° forward oblique arthroscopic probe. The tarsocural joint was inflated with sterile polyionic fluids before an arthroscopic portal was made at the proximodistal midpoint of the dorsolateral outpouching of the tarsocural joint, just abaxial to the extensor tendon bundle. The positions of instrument portals were determined by percutaneous needle placement abaxial to this site such that instrument trajectories were perpendicular to the long axis of the tibia.

In the acute cases, joints contained sanguinous fluid and blood clots, and these were evacuated. In such cases the fracture usually was covered with haemorrhage. In longer standing cases fractures were covered by granulation tissue with varying degrees of organisation or by disorganised fibrous tissue (Figs 1b, 4c). Frequently, this obscured immediate recognition of the fracture. In such cases elevation of the fracture fragment was required before further dissection. Fractures disrupted varying amounts of the origins of the short lateral collateral ligaments. Fragment removal required always dissection of the insertions of the same from the talus. This was effected with straight and curved fixed blade meniscectomy knives and scissors. During this procedure divided material was removed intermittently from the dissection plane using a motorised synovial resector in an oscillating mode with suction applied. Complete division of the origin of these ligaments is necessary for removal and this required frequently that the fragment or most plantar fragment was pushed into the plantar pouch in order to visualise the ligament attachment. Dissection at this point was continued by passage of the arthroscope between the long collateral ligament and the talus. Fragments were then retrieved, usually through the same dissection plane but on occasions this was most readily performed by creation of an ipsilateral plantar instrument portal. Large, full thickness fragments were retrieved utilising 6 × 10 mm arthroscopic rongeurs. Smaller fragments were removed utilising 2, 3 or 4 × 10 mm rongeurs. Frayed short collateral ligaments (Fig 1d) were debrided with a motorised synovial resector. Large fragments frequently exposed the long lateral collateral ligament and, on occasions, the tendon of insertion of the lateral digital extensor and its synovial sheath (n = 2) (Fig 2d). The fracture bed was debrided utilising 2, 3 or 4 mm arthroscopic curettes. Loose osteochondral fragments that had displaced in either the dorsal or plantar compartments of the joint were removed utilising appropriately placed instrument portals. In one case a large comminuted, displaced fracture of the medial malleolus of the tibia was also removed arthroscopically. This required dorsomedial arthroscopic and instrument portals, to enable dissection of capsular and collateral ligament (short and long) attachments, prior to removal.

At the end of surgery, joints were lavaged and evacuated before skin portals closed with simple interrupted sutures of 3 metric monofilament polyamide. The limbs were then protected with sterile elasticated dressings (Pressage) for 14 days or until there was concurrent damage to the long collateral ligament, support was given by application of a short hock cast (n = 2). This extended from the junction of the middle and distal thirds of the tibia to the junction of the proximal and middle thirds of the metatarsus. The tarsus was immobilised in a passively extended position. The casts consisted of a single role of conforming synthetic orthopaedic padding (Soffban) followed by 6 roles of 10 cm plaster of Paris (Gypsona), and 2 roles of 7.5 cm and 2 rolls of 12.5 cm fibreglass tape (Vetcast). In 24 cases surgery was performed by I.M.W., and in 2 cases surgery was performed by M.R.W.S.

**Post operative care**

Perioperative antimicrobials consisted of penicillin (30,000 iu/kg bwt i.v. q. 8 h) (Crystapen) commencing presurgery and administered for 24–96 (mean 47) h. All horses received a single preoperative dose of phenylbutazone (Equipalazone) (4 mg/kg bwt i.v.); in 4 cases this continued at 2 mg/kg bwt i.v. for 2, 4 and 21 (n = 2) days. Post operatively dressings were maintained until 14 days after surgery, at which point the sutures were also removed. All horses were confined to their stables until sutures were removed and then received increasing amounts of hand walking or horse-walker exercise twice daily for a minimum of 2 months. Horses typically then received a further month of ridden walking exercise. At the end of this period, if the horse was sound at a trot, 2 months of gradually increasing amounts of trotting exercise was recommended. If the horse was still sound at the end of this period, a gradual re-introduction to work was attempted.

Follow-up information was obtained from race records (n = 12) and/or telephone questionnaire from owners (n = 12). The latter detailed complications, residual lameness, current activity and, when appropriate, time to return to work.

**Results**

Case details of cases are displayed in Table 1. Horses were aged 4 months to 13 years (mean 4 years) and included 25 Thoroughbreds and one sports horse. There were 18 geldings, 5 mares and 3 colts. Horses were used for a variety of purposes: racing (n = 17), intended for racing (n = 4), intended for eventing (n = 1), intended for dressage (n = 1), general purpose (n = 2) and breeding (n = 1).

The animals had been lame for 5–161 (mean 33) days prior to referral. In 13 horses there was a known history of trauma (6 following falls, 5 became cast in a stable, 2 following kicks). In
13 the cause of injury was unknown. In all horses lameness was acute in onset and reported to be initially of moderate to severe intensity. All animals were lame at presentation with a range of severity from 1–8 out of 10 (median 3). The affected tarsocrural joint was distended in all horses (mild $n = 1$, moderate $n = 10$, marked $n = 12$, not recorded $n = 3$). Other commonly recorded clinical findings included thickening of the tarsocrural joint capsule ($n = 15$), a plaque of pitting soft tissue swelling on the lateral aspect of the tarsus ($n = 8$) and loss of definition of the lateral malleolus ($n = 8$). Crepitus was palpable in only 5 cases.

Radiographs demonstrated comminution (Figs 1a, 2a,b, 3a, 4a) in all except one case. One case (25) exhibited biaxial fractures of the tibial malleolus; there were no bilateral cases. In 4 cases,
Fig 2: Radiographic and corresponding arthroscopic images (viewed from a dorsolateral portal) from Case 21. a) Dorsoplantar radiographic projection demonstrating a comminuted, displaced and rotated fracture of the lateral malleolus of the tibia. b) A dorsal 5° lateral-plantaromedial oblique radiographic projection skylines the fractured lateral malleolus. c) At surgery the fracture fragment has rotated 90°. d) Following removal of the fracture, in this case the lateral digital extensor tendon is visible. LM = lateral malleolus; LTR = lateral trochlear ridge talus; LDE = lateral digital extensor tendon.
<table>
<thead>
<tr>
<th>Case</th>
<th>Age (years)</th>
<th>Gender</th>
<th>Limb</th>
<th>Use</th>
<th>Duration of fracture</th>
<th>Fracture configuration</th>
<th>SLCL disruption</th>
<th>LLCL disruption</th>
<th>Other osteochondral pathology</th>
<th>Capsular/plica tearing</th>
<th>Portals</th>
<th>Time of follow-up (months)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>Gelding</td>
<td>Right</td>
<td>RH (flat and hurdle)</td>
<td>Unknown</td>
<td>Comminuted, hinged on SLCL and rotated</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal</td>
<td>40</td>
<td>Sound, returned to racing at 10 months</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>Gelding</td>
<td>Right</td>
<td>RH (flat and hurdle)</td>
<td>14</td>
<td>Comminuted, hinged on SLCL, loose fragments plantar pouch</td>
<td>Complete</td>
<td>Partial</td>
<td>None</td>
<td>None</td>
<td>Dorsal</td>
<td>35</td>
<td>Sound, returned to racing at 12 months</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>Filly</td>
<td>Right</td>
<td>RH (flat and hurdle)</td>
<td>28</td>
<td>Comminuted, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>76</td>
<td>Lame, retired to study</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>Gelding</td>
<td>Left</td>
<td>RH (NH flat and hurdles)</td>
<td>161</td>
<td>Comminuted, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>47</td>
<td>Sound, returned to racing at 11 months</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>Gelding</td>
<td>Left</td>
<td>RH (NH flat and Chase)</td>
<td>28</td>
<td>Comminuted, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>22</td>
<td>Sound, returned to racing at 11 months</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
<td>Gelding</td>
<td>Left</td>
<td>FRH</td>
<td>Acute</td>
<td>Major fragment hinged on SLCL and rotated</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>20</td>
<td>Sound, returned to racing at 10 months</td>
</tr>
<tr>
<td>7</td>
<td>1</td>
<td>Gelding</td>
<td>Left</td>
<td>Intended for RH</td>
<td>Unknown</td>
<td>Major fragment hinged on SLCL, small comminuted fragments dorsal and plantar pouches</td>
<td>Complete</td>
<td>Partial</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>5</td>
<td>Sound, sold at yearling sales 5 months post operatively, lost to further follow-up</td>
</tr>
<tr>
<td>8</td>
<td>9</td>
<td>Gelding</td>
<td>Left</td>
<td>FRH</td>
<td>Acute</td>
<td>Comminuted, hinged on SLCL, small comminuted fragments in plantar pouch</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>29</td>
<td>Sound, returned to racing at 11 months</td>
</tr>
<tr>
<td>9</td>
<td>5</td>
<td>Filly</td>
<td>Right</td>
<td>FRH</td>
<td>Unknown</td>
<td>Comminuted, hinged on SLCL, small scattered comminuted fragments dorsal and plantar pouches</td>
<td>Complete</td>
<td>None</td>
<td>Capsular tearing dorsal pouch</td>
<td>Dorsal-plantar</td>
<td>5</td>
<td>Lame, retired to study</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>4</td>
<td>Gelding</td>
<td>Right</td>
<td>FRH</td>
<td>49</td>
<td>Comminuted, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>8</td>
<td>Sound, returned to racing at 7 months</td>
</tr>
<tr>
<td>11</td>
<td>1</td>
<td>Colt</td>
<td>Left</td>
<td>for FRH</td>
<td>Acute</td>
<td>Comminuted, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>Avulsion fragment dorsal central tarsal bone</td>
<td>None</td>
<td>Capsular-plantar</td>
<td>n/a</td>
<td>Lost to follow-up</td>
</tr>
<tr>
<td>12</td>
<td>5</td>
<td>Gelding</td>
<td>Right</td>
<td>FRH</td>
<td>Acute</td>
<td>Comminuted, hinged on SLCL, small comminuted fragments dorsal pouch</td>
<td>Complete</td>
<td>None</td>
<td>Avulsion fragment dorsal central tarsal bone</td>
<td>Capsular and plical tearing dorsal pouch</td>
<td>Capsular-plantar</td>
<td>21</td>
<td>Sound, returned to racing at 11 months</td>
</tr>
<tr>
<td>13</td>
<td>4</td>
<td>Mare</td>
<td>Left</td>
<td>Intended for eventing</td>
<td>Unknown</td>
<td>Major fragment hinged on SLCL, small comminuted fragments dorsal and plantar pouches</td>
<td>Complete</td>
<td>Partial</td>
<td>Avulsion fragment dorsal central tarsal bone</td>
<td>Capsular and plical tearing dorsal pouch</td>
<td>Dorsal-plantar</td>
<td>23</td>
<td>Returned to lower level of work, remained low grade lame so retired to stud</td>
</tr>
<tr>
<td>14</td>
<td>6</td>
<td>Gelding</td>
<td>Right</td>
<td>RH (hurdles)</td>
<td>Acute</td>
<td>Comminuted, hinged on SLCL, small scattered comminuted fragments dorsal and plantar pouches</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>24</td>
<td>Sound, returned to training at 12 months, unacclimated since injury</td>
</tr>
<tr>
<td>15</td>
<td>6</td>
<td>Gelding</td>
<td>Left</td>
<td>RH (hurdles and chase)</td>
<td>10</td>
<td>Comminuted fracture hinged on SLCL, small scattered comminuted fragments dorsal and plantar pouches</td>
<td>Complete</td>
<td>None</td>
<td>Small DIRT OCD</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>21</td>
<td>Sound, returned to racing at 6 months</td>
</tr>
<tr>
<td>16</td>
<td>5</td>
<td>Gelding</td>
<td>Left</td>
<td>RH (hurdles and chase)</td>
<td>Unknown</td>
<td>Comminuted, hinged on SLCL</td>
<td>Partial</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>33</td>
<td>Sound, returned to racing at 12 months</td>
</tr>
<tr>
<td>17</td>
<td>0.8</td>
<td>Colt</td>
<td>Left</td>
<td>Intended for dressage</td>
<td>24</td>
<td>Comminuted, hinged on SLCL, small scattered fragments dorsal and plantar pouches</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>7</td>
<td>Subjected to euthanasia because of persistent lameness 7 months post operatively</td>
</tr>
<tr>
<td>18</td>
<td>4</td>
<td>Mare</td>
<td>2</td>
<td>Brood mare</td>
<td>48</td>
<td>Comminuted, hinged on SLCL, small comminuted fragments dorsal and plantar pouches</td>
<td>Complete</td>
<td>Partial</td>
<td>None</td>
<td>Large tear in capsule dorsolaterally</td>
<td>Dorsal</td>
<td>12</td>
<td>Sound, returned to stud as brood mare</td>
</tr>
<tr>
<td>19</td>
<td>3</td>
<td>Gelding</td>
<td>Left</td>
<td>FRH</td>
<td>26</td>
<td>Comminuted, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>Avulsion fragment dorsal central tarsal bone</td>
<td>Dorsal-plantar</td>
<td>5</td>
<td>Sound, sold at horses in training 5 months post operatively, lost to further follow-up</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>0.33</td>
<td>Filly</td>
<td>Right</td>
<td>Intended for RH</td>
<td>Unknown</td>
<td>Comminuted, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
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<td>Lost to follow-up</td>
</tr>
<tr>
<td>21</td>
<td>4</td>
<td>Gelding</td>
<td>Left</td>
<td>FRH and hurdles</td>
<td>10</td>
<td>Comminuted, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>10</td>
<td>Sound, returned to racing at 4 months</td>
</tr>
<tr>
<td>22</td>
<td>7</td>
<td>Gelding</td>
<td>Right</td>
<td>RH (hurdles and chase)</td>
<td>28</td>
<td>Comminuted, hinged on SLCL, small comminuted fragments dorsal and plantar pouches</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>9</td>
<td>Sound, returned to racing at 9 months</td>
</tr>
<tr>
<td>23</td>
<td>2</td>
<td>Colt</td>
<td>Right</td>
<td>Intended for RH</td>
<td>5</td>
<td>Comminuted 2 piece fracture, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>5</td>
<td>Sound and in full training, not yet raced</td>
</tr>
<tr>
<td>24</td>
<td>13</td>
<td>Gelding</td>
<td>Left</td>
<td>GP</td>
<td>28</td>
<td>One piece fracture hinged on SLCL and rotated</td>
<td>Partial</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>n/a</td>
<td>Convealing</td>
</tr>
<tr>
<td>25</td>
<td>3</td>
<td>Gelding</td>
<td>Right</td>
<td>GP</td>
<td>28</td>
<td>Comminuted, hinged on SLCL</td>
<td>Partial</td>
<td>None</td>
<td>Large comminuted fracture medial malleolus</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>n/a</td>
<td>Convealing</td>
</tr>
<tr>
<td>26</td>
<td>2</td>
<td>Gelding</td>
<td>Right</td>
<td>Intended for RH</td>
<td>9</td>
<td>Comminuted, hinged on SLCL</td>
<td>Complete</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Dorsal-plantar</td>
<td>7</td>
<td>Sound, due to start training</td>
</tr>
</tbody>
</table>

RH = racehorse; FRH = flat racehorse; SLCL = short lateral collateral ligament; LLCL = long lateral collateral ligament; OCD = osteochondrosis dissecans; DIRT = distal intermediate ridge tibia.
comminuted fragments were identified displaced throughout the joint (dorsal, Cases 9 and 18; dorsal and plantar, Cases 14 and 19) (Fig 4b). In 2 cases there was further fragmentation from the dorsal aspect of the central tarsal bone (Cases 12 and 13). Two additional cases had capsular new bone on the distal tibia (Cases 10 and 19), and one case fragmentation of the proximal medial eminence of the talus (Case 13). In all cases where it was performed, ultrasonographic examination (n = 9) identified the fracture fragment(s) attached to disrupted short lateral collateral ligaments. In 2 horses this also revealed disruption of the long lateral collateral ligament (Fig 3b).

Arthroscopic findings are documented in Table 1. In 3 cases, the dorsal border of the short lateral collateral ligament was intact, and the fracture could not be visualised initially. Dissection of the short lateral collateral ligaments in these cases was required to allow identification of the fracture fragment(s) from the lateral malleolus. Fragments were invariably hinged on the short lateral collateral ligaments (Fig 1c), and frequently were rotated approximately 90° (Fig 2c). Only 4 cases had damage to the long lateral collateral ligament. Four cases had further concurrent osteochondral pathology related to the injury and 5 had concurrent capsular and/or plical tearing. Displaced comminuted fragments within the dorsal and/or plantar pouch of the joint were identified in 11 cases, which had been radiographically identified prior to surgery in only 5 animals.

All horses recovered uneventfully from general anaesthesia, including the 2 cases with hock casts. There were no post operative complications during the period of hospitalisation, and at the time of discharge (3–23 days post operatively, mean 7) all horses were walking comfortably. All incisions healed by first intention. One horse developed infection of the affected tarsocrural joint following suture removal; this required a further arthroscopic procedure and antimicrobial treatment. Long-term follow-up information was available for 24 of 26 animals at 5–76 (mean 19) months after surgery. Two horses were <4 months since surgery and still convalescing. Eighteen horses returned to previous function at 4–12 (mean 9.5) months after surgery. This included 16 racing Thoroughbreds which were sound and had returned to racing (n = 12) or entered training (n = 4), one brood mare (that had suffered post operative tarsocrural joint infection), which was sound and had returned to breeding duties, and one yearling Thoroughbred, which was sound and sold at yearling sales. Four animals remained lame; 3 were retired to stud and one was subjected to euthanasia 7 months after surgery; there was no identifiable commonality in the available data between these individuals.

Discussion

This paper describes a safe and repeatable arthroscopic technique for assessment and treatment of fractures of the lateral malleolus of the tibia and documents a favourable success rate in treating affected horses. The technique is efficient but technically demanding, requiring careful dissection of the fragment from the soft tissue attachments, utilising arthroscope and instrument portals relatively closely positioned to one another. The latter increases the difficulty of triangulation. However, with experience, surgical time can be reduced compared to arthrotomy and it enjoys all of the other benefits of minimally invasive techniques.

An additional advantage of the arthroscopic technique is allowing comprehensive evaluation of the tarsocrural joint. This

Fig 3: Radiographic and corresponding ultrasonographic image of Case 18. a) Dorsal 5° lateral-plantaromedial oblique radiographic projection demonstrating a comminuted, displaced fracture of the lateral malleolus of the tibia. b) Longitudinal ultrasonogram (proximal to the left) of the long lateral collateral ligament. There is partial disruption to the ligament adjacent to the fracture (arrows).
facilitated identification and removal of scattered pieces of osteochondral debris, which were identified in over twice as many cases as predicted by preoperative radiography. In addition, arthroscopic examination enabled identification and appropriate management of additional capsular and plical pathology. When comparing this to removal by arthrotomy, displaced comminuted fragments were identified radiographically in 5 of 16 cases prior to surgery. In 5 cases there was greater comminution adjacent to the

Fig 4: Radiographic and corresponding arthroscopic images (viewed from a dorsolateral portal) from Case 19. a) Dorsoplantar radiographic projection demonstrating a displaced fracture of the lateral malleolus of the tibia. b) Lateromedial radiographic projection demonstrating a small comminuted fragment displaced into the proximal intertarsal joint. c) The fracture line (arrows) is covered by fibrous tissue. LM = lateral malleolus of the tibia; DIRT = distal intermediate ridge tibia; LTR = lateral trochlear ridge talus.
lateral malleolus than preoperative radiographs had predicted (Wright 1992), although there was no mention of further scattered osteochondral debris. In addition, no further soft tissue (capsular or plical) pathology was identified in this earlier series.

The period of perioperative antibiosis in the current series was dictated by the degree of periarticular swelling post operatively, and was less than that reported by Wright (1992) following removal via arthrotomy (5 days). Requirements for analgesia were also minimal post operatively, with only 4 horses receiving more than a single preoperative dose. This reflects the less invasive surgical approach used, and although not documented by Wright (1992), probably enabled a shorter period of hospitalisation.

The outcome of horses previously reported following removal via arthrotomy was good, with 13 of 16 horses returning to work at a level similar to preinjury (Wright 1992). The case population in the current series is similar to that reported by Wright (1992), with the majority also used for racing. Successful outcomes were achieved in a similar proportion (20 of 24 horses). Considering this, and the aforementioned advantages afforded by arthroscopy, the current technique should replace removal via arthrotomy as the treatment of choice for managing fractures of the lateral malleolus of the tibia.

Consistent with previous reports (Wright 1992), in the majority of cases no tarsal instability was demonstrable at the end of surgery, and therefore horses were recovered from general anaesthesia in bandages only. In 2 cases additional support was provided with short hock casts. In both, surgeons had concerns regarding minor instability on manipulation of the tarsus at the end of surgery. One case had marked disruption to the long lateral collateral ligament and the other a concurrent fracture of the medial malleolus, which also was removed. Horses recovered well in the casts and there were no untoward sequelae.

Interestingly, in 3 cases in this series the dorsal border of the short lateral collateral ligaments had remained intact and arthroscopic examination of the joint therefore initially failed to visualise the fracture. There was, however, haemorrhage within the dorsal pouch of the joint, and synovial fluid was discoloured. This presentation may lead to confusion the first time encountered. The short hock casts and there were no untoward sequelae.

In all cases in which it was performed, ultrasonography identified disruption to the short lateral collateral ligaments. In both cases where ultrasonographic examination identified disruption of the long lateral collateral ligament, this was confirmed arthroscopically. Logically, cases with concurrent long lateral collateral ligament disruption represent an increased risk for anaesthetic recovery. Preoperative prediction enables this to be communicated to relevant parties prior to surgery and allows the surgeon to prepare for cast support if this is deemed advantageous. On the basis of the information obtained in this series, we recommend ultrasonographic evaluation on a routine basis prior to surgery.

In conclusion, arthroscopic removal of fractures of the lateral malleolus of the tibia is technically demanding, but can be performed with minimal complications, with low patient morbidity and short periods of hospitalisation. The majority of horses are able to successfully return to work following the procedure and the advantages of arthroscopic removal compared to removal via arthrotomy should make this the technique of choice in clinical practice for treatment of fractures of the lateral malleolus of the tibia.

Acknowledgement

We acknowledge Gaynor Minshall MRCVS for ultrasonographic evaluation of 4 cases in this series.

Manufacturers’ addresses

1GE Healthcare, Amersham, Buckinghamshire, UK.
2Karl Storz, Tuttingen, Germany.
3Sontec, Centennial, Colorado, USA.
4Dyonics/Smith & Nephew Endoscopy, Godmanchester, Cambridgeshire, UK.
5Scanlan, St Paul, Minnesota, USA.
6Ethicon, Edinburgh, UK.
7Jupiter Products, Cardiff, UK.
8Smith & Nephew, London, UK.
93M, St Paul, Minnesota, USA.
10Pitman-Moore Ltd, Crewe, Cheshire, UK.
11Arnolds, Shrewsbury, Shropshire, UK.

References


The use of small (2.7 mm) screws for arthroscopically guided repair of carpal chip fractures

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Newmarket Equine Hospital, UK.

Keywords: horse; carpus; chip fracture; arthroscopy; repair

Summary

Reasons for performing the study: Removal of large chip fractures of the carpal bones and the osteochondral deficits that result, have been associated with a worse prognosis than removal of small fragments in similar locations.

Hypothesis: Reducing the articular defects by repair of large osteochondral fragments may have advantages over removal.

Methods: Horses with osteochondral chip fractures that were of sufficient size and infrastructure to be repaired with small (2.7 mm diameter) AO/ASIF cortex screws were identified and repair effected by arthroscopically guided internal fixation.

Results: Thirty-three horses underwent surgery to repair 35 fractures of the dorsodistal radial carpal bone (n = 25), the dorsal margin of the radial facet of the third carpal bone (n = 9) and the intermediate facet of the distal radius (n = 1). There were no surgical complications and fractures healed satisfactorily in 26 of 28 horses and 23 horses returned to racing performance.

Conclusion: Arthroscopically guided repair of carpal chip fractures with small diameter cortex screws is technically feasible and experiences with 33 cases suggest that this may have advantages over fragment removal in managing such cases.

Potential relevance: Surgeons treating horses with large chip fractures of the carpal bones should consider arthroscopically guided internal fixation as an alternative to removal.

Introduction

Articular fractures of the cuboidal carpal bones and distal radius are a common cause of lameness in racehorses. Fractures that involve the subchondral bone and articular cartilage of one articular surface, and that exit the bone (usually dorsally) either within the synovial cavity or within the joint capsule or associated intercarpal carpal ligaments are classified as chip fractures (McIlwraith et al. 1987). Fractures that extend from one articular surface of the cuboidal bone within the middle carpal joint, through the bone proximally to its antebrachiocarpal articular surface, or distally to its carpometacarpal articular surface, are classified as ‘slab’ fractures (Richardson 1986).

Lag screw repair of slab fractures is generally considered to be the treatment of choice (Richardson 1986). These most commonly involve the third carpal bone in a frontal plane, and a technique for arthroscopically guided internal fixation has been described (Richardson 1986, 2002). Minor variations in this technique have also been described (McIlwraith et al. 2005) but all involve delineation of the margins within the middle carpal joint by arthroscopically guided percutaneous needles. The mediolateral mid-point of the fracture is found by the insertion of a percutaneous needle midway between the first 2 needles. A 19 gauge 89 mm needle is employed and this is inserted under arthroscopic visualisation parallel and close to the proximal articular surface of the third carpal bone with a trajectory that is appropriate for a transfixation screw. The distal margin of the bone is identified and marked by insertion of a percutaneous needle into the carpometacarpal joint, directly distal to the needle marking the mid-point of the fracture in the middle carpal joint. A 4.5 or 3.5 mm AO/ASIF cortex screw is then inserted using a lag technique through a stab incision made midway between the needles at the proximodistal mid-point of the fractured bone (McIlwraith et al. 2005). In this location the screw is placed through the dorsal intercarpal ligaments. The dorsal surface of the third carpal bone is irregularly convex at this point and an adequate concavity must be created for the screw head in order to avoid point contact with the bone, consequential compromise of compression, predisposition to screw fracture and to minimise protrusion of the screw head into the dorsal intercarpal ligaments.

Parasagittal fracture of the third carpal bone and slab fracture involving other and multiple carpal bones may be repaired using similar principles but varying the sites of needle and implant placement as appropriate (McIlwraith et al. 2005).

Arthroscopic removal of chip fractures of the carpal bones and the osteochondral deficits that result, have been associated with a worse prognosis than removal of small fragments in similar locations.

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carpal chip fractures deteriorates as the size of the resultant articular defect increases (McIlwraith et al. 1987, 2005).

This paper describes a technique for arthroscopically guided repair of carpal chip fractures, the authors’ experience with the technique and the results of treatment in 33 horses.

**Materials and methods**

**Study**

Retrospective analysis of case records of all horses that underwent carpal arthroscopy and repair of a cuboidal carpal chip fracture at Newmarket Equine Hospital from 2004–2008 was performed. Data were collected for each horse to include age, breed, gender and use. Radiologically, chip fractures were defined as linear defects extending through the subchondral bone with or without a discernable exit point dorsally, which do not extend to the opposite articular surface.

**Diagnostic procedures**

All horses were subjected to a lameness examination including palpation of the limbs and gait evaluation at a walk and trot. Lameness was graded out of 10 (0 = sound to 10 = nonweightbearing) (Ross 2003).

All horses underwent radiographic examination of the affected and contralateral carpus including dorsopalmar, lateromedial, dorsolateral-palmaromedial oblique, dorsomedial-palmarolateral oblique, flexed lateromedial and dorsal 30° proximal-dorsodistal oblique images. In selected cases, further dorsoproximal-dorsodistal oblique images angled at either 55° or 80° to skyline the proximal row of carpal bones or distal radius (respectively) were also obtained (Butler et al. 2008).

Cases were selected as potentially suitable on the basis of fragment size and with radiologically identifiable infrastructure in the fragment and parent bone that might permit repair. None or minimal displacement was also considered desirable; fragments that were loose bodies and displaced markedly in the joint were excluded.

The convex dorsal distal articular surface of the radial carpal bone determines the maximal length of subchondral screws to avoid emergence and impingement on the third carpal bone. This distance was predetermined in flexed lateromedial radiographic projections prior to surgery. The flat proximal articular surface of the third carpal bone does not create such limitations. The dorsal distal articular surfaces of the radius are concave but this is shallow compared to the depth of most fragments so that implant length and trajectory are less critical.

Fractures were classified as incomplete if there was linear discontinuity in the subchondral bone and underlying spongiosa that did not extend to the dorsal surface of the bone. When this was identifiable, fractures were classified as complete. Fractures were classified as displaced only if complete and with incongruity at the articular surface.

**Surgical technique**

All horses underwent arthroscopic surgery using a 4 mm 25° forward oblique arthroscope (Karl Storz) under general anaesthesia and in dorsal recumbency. For middle carpal joints, the carpus was positioned at an approximately 70° flexion angle and for the antebrachio-carpal joint at approximately 120°. In all cases the arthroscope was inserted through a dorsolateral portal and assessment of the dorsal compartment of joints (middle carpal n = 32; antebrachio-carpal n = 1) was performed as previously described (McIlwraith et al. 2005).

Following identification of fractures (Figs 1a, 2a), the medial and lateral margins were defined by placement of percutaneous marker needles (23 gauge 25 mm) (Fig 1b). An 18 gauge 89 mm spinal needle was then placed midway between the marker needles close and parallel to the articular surface of the fractured bone to act as a guide for drill trajectory (Fig 2b). A further needle (19 gauge 1.5 inch (1.1 × 38 mm) was then used to mark the proposed site of screw placement just proximal (radial carpal bone and radius) or
distal (third carpal bone) to the subchondral bone plate. This was consistently an intra-articular location that also permitted direct visualisation of the repair process. A stab incision was made at this point and a 2.7 mm glide hole created through the fracture fragment to a radiographically predetermined depth. Following confirmation of fragment depth, a 2 mm hole was then drilled into the parent bone to the predetermined distance to permit a cortical screw to be inserted using the lag principle. All holes were countersunk and then tapped to cut a thread, before an appropriately selected length 2.7 mm AO/ASIF cortex screw was inserted and tightened (Fig 2c). A lateromedial radiograph was then obtained to confirm appropriate implant position.

Following fracture repair, small loose osteochondral fragments and cartilage flaps were removed but there was little or no additional debridement. Any further pathology within the joint (Table 1) was managed accordingly before lavage and evacuation of the joint. Arthroscopy portals and the incision for implant placement were closed with simple interrupted skin sutures of 3 Metric monofilament polyamide (Ethilon)2. Limbs were dressed with elasticated bandages (Pressage)3 and the horses allowed to recover unassisted from general anaesthesia.

Post operative care

Perioperative antimicrobials consisted of sodium benzyl penicillin (Crystapen)4 (30,000 iu/kg bwt i.v. q. 8 h) given preoperatively and administered for 24–72 (mean 43) h following surgery. All horses received a single preoperative dose of phenylbutazone (Equipalazone)5 (4 mg/kg bwt i.v.). Post operatively, dressings were maintained for 4–17 (mean 8) days and sutures were removed 10–14 (mean 11) days after surgery. Horses were confined to their stables for 1–4 (mean 2.3) weeks, before undertaking increasing amounts of hand-walking or horse-walker exercise twice daily for a minimum of 4 (range 4–12, mean 8.4) weeks. At the end of this period, if the horse was sound at a trot, gradually increasing amounts of trotting exercise was recommended (range 4–12, mean 6 weeks). Alternatively, horses were allowed pasture exercise in gradually increasing areas until ridden work was resumed. If the horse was still sound at the end of this period then a gradual re-introduction to training was attempted.

Case follow-up information was obtained from details recorded at re-examinations either at the hospital or by referring veterinarians during the convalescence period, and finally by tracing race records (number or races run, number of places) at http://www.racingpost.com (n = 23). If horses were unraced (n = 10), owners and/or trainers were contacted to enquire why the horse had not raced and about progress following surgery, including residual lameness, current activity and, when appropriate, time to return to work.

Results

From 2004–2008, 329 horses underwent carpal arthroscopy, of which 33 horses were identified with 35 chip fractures of the cuboidal carpal bones that on radiological and arthroscopic evaluation were considered to be of sufficient size and integrity to permit internal fixation.

Case details of horses are displayed in Table 1. The horses were aged 1–4 years old (median 2). There were 12 fillies, 10 geldings and 6 colts (5 unknown). All were in flat race training. More fractures involved left (n = 24) than right (n = 11) carpi (including 2 cases that had fractures repaired bilaterally). Fractures involved
<table>
<thead>
<tr>
<th>Case</th>
<th>Age (years)</th>
<th>Gender</th>
<th>Limb</th>
<th>Radiological fracture description</th>
<th>Additional arthroscopic findings</th>
<th>Time of follow-up (months)</th>
<th>Outcome</th>
<th>Racing record</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>Gelding</td>
<td>Right</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>None</td>
<td>25</td>
<td>Returned to racing at 9 months.</td>
<td>Ran 9, won 2, placed 2</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>Unknown</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Small nonmarginal area of fragmentation dorsodistal Cr; partial thickness cartilage defects dorsoproximal C3.</td>
<td>9</td>
<td>Returned to racing at 9 months.</td>
<td>Ran 2</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td>Colt</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Small area of fragmentation dorsodistal Cr.</td>
<td>11</td>
<td>Returned to racing at 10 months.</td>
<td>Ran 3, placed 2</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>Colt</td>
<td>Right</td>
<td>Partially healed, incomplete nondisplaced frontal plane fracture radial facet dorsoproximal C3. Loss of infrastructure within fragment. Loss of infrastructure and increased density adjacent parent bone. Remodelled dorsodistal Cr.</td>
<td>Detached cartilage flaps within fracture plane.</td>
<td>12</td>
<td>Returned to racing at 7 months.</td>
<td>Ran 4, won 1, placed 1</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>5</td>
<td>2</td>
<td>Colt</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Small osteochondral defect at lateral aspect of fracture, small full thickness cartilage defect C4. Commination within fracture line, cartilage thinning and impingement lesions.</td>
<td>10</td>
<td>Returned to racing at 9 months.</td>
<td>Ran 2, placed 1</td>
<td>Reduced</td>
</tr>
<tr>
<td>6</td>
<td>2</td>
<td>Unknown</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Cartilage defects dorsoproximal C3 and dorsodistal Cr.</td>
<td>6</td>
<td>Returned to training at 6 months. Lost to further follow-up.</td>
<td>Equal/improved</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>1</td>
<td>Unknown</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Partial thickness cartilage lesions palmar to fracture Cr.</td>
<td>19</td>
<td>Returned to racing at 13 months.</td>
<td>Ran 13</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
<td>Filly</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Partial thickness cartilage defect dorsodistal Cr.</td>
<td>24</td>
<td>Returned to racing at 22 months.</td>
<td>Ran 4</td>
<td>Reduced</td>
</tr>
<tr>
<td>9</td>
<td>3</td>
<td>Gelding</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Fragmentation dorsoproximal radial facet C3 and adjacent to MPICL. Multiple partial thickness cartilage defects throughout joint.</td>
<td>22</td>
<td>Returned to racing at 22 months.</td>
<td>Ran 2</td>
<td>Reduced</td>
</tr>
<tr>
<td>10</td>
<td>4</td>
<td>Gelding</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Small partial thickness cartilage defects dorsodistal Cr.</td>
<td>11</td>
<td>Returned to racing at 11 months.</td>
<td>Ran 3</td>
<td>Reduced</td>
</tr>
<tr>
<td>11</td>
<td>3</td>
<td>Gelding</td>
<td>Right</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Fissure fracture Cr.</td>
<td>14</td>
<td>Returned to racing at 11 months.</td>
<td>Ran 8</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>12</td>
<td>3</td>
<td>Unknown</td>
<td>Right</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr.</td>
<td>Comminuted fracture dorsodistal Cr; partial thickness cartilage defects dorsoproximal C3.</td>
<td>11</td>
<td>Returned to racing at 11 months.</td>
<td>Ran 8</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>Case</td>
<td>Age (years)</td>
<td>Gender</td>
<td>Limb</td>
<td>Radiological fracture description</td>
<td>Additional arthroscopic findings</td>
<td>Time of follow-up (months)</td>
<td>Outcome</td>
<td>Racing record</td>
<td>Class</td>
</tr>
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<tr>
<td>13</td>
<td>2</td>
<td>Colt</td>
<td>Right</td>
<td>Incomplete nondisplaced frontal plane fracture dorsoproximal radial facet C3. Loss of infrastructure and increased density within fragment and adjacent parent bone.</td>
<td>None</td>
<td>54</td>
<td>Returned to racing at 10 months.</td>
<td>Ran 54, won 1, placed 10</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>14</td>
<td>2</td>
<td>Filly</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment. Remodelled dorsodistal Cr. Enthesious new bone dorsal Cr.</td>
<td>None</td>
<td>16</td>
<td>Returned to racing at 16 months.</td>
<td>Ran 1</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>15</td>
<td>1</td>
<td>Filly</td>
<td>Right</td>
<td>Incomplete nondisplaced frontal plane fracture dorsoproximal radial facet C3. Loss of infrastructure within fracture fragment and increased density within parent bone.</td>
<td>None</td>
<td>45</td>
<td>Returned to racing at 8 months.</td>
<td>Ran 8, won 1, placed 1</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>16</td>
<td>4</td>
<td>Gelding</td>
<td>Right</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment.</td>
<td>None</td>
<td>20</td>
<td>Returned to racing at 10 months.</td>
<td>Ran 6, placed 4</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>17</td>
<td>3</td>
<td>Unknown</td>
<td>Right</td>
<td>Complete displaced frontal plane fracture dorsoproximal radial facet C3. Loss of infrastructure within fragment.</td>
<td>None</td>
<td>11</td>
<td>Returned to racing at 8 months.</td>
<td>Ran 5, placed 1</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>18</td>
<td>2</td>
<td>Gelding</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture intermediate facet distal radius. Loss of density and infrastructure within fragment.</td>
<td>None</td>
<td>8</td>
<td>Returned to racing at 8 months.</td>
<td>Ran 2</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>19</td>
<td>?</td>
<td>Filly</td>
<td>Left</td>
<td>Complete displaced frontal plane fracture dorsoproximal radial facet C3. Loss of infrastructure within fragment.</td>
<td>Trough shaped osteochondral defect within fracture line.</td>
<td>NAD</td>
<td>Lost</td>
<td>n/a</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>2</td>
<td>Filly</td>
<td>Bilateral</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment.</td>
<td>Trough of comminution within fracture line.</td>
<td>11</td>
<td>Returned to racing at 3 months.</td>
<td>Ran 4, placed 1</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>21</td>
<td>3</td>
<td>Filly</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment.</td>
<td>Trough of comminution within fracture line, fragmentation dorsoproximal radial facet C3.</td>
<td>Avulsion fragment within LPICL.</td>
<td>Exported after surgery and lost to further follow-up.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>3</td>
<td>Filly</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment.</td>
<td>None</td>
<td>4</td>
<td>Returned to training at 4 months, subsequently lost to follow up.</td>
<td>Ran 3</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>23</td>
<td>2</td>
<td>Colt</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsoproximal radial facet C3. Loss of density and infrastructure within fragment.</td>
<td>None</td>
<td>9</td>
<td>Returned to racing at 7 months.</td>
<td>Ran 3</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>24</td>
<td>4</td>
<td>Gelding</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment.</td>
<td>Full thickness cartilage defect C4.</td>
<td>25</td>
<td>Returned to racing at 7 months.</td>
<td>Ran 10, won 1</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>25</td>
<td>2</td>
<td>Gelding</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment.</td>
<td>Trough of fragmentation within fracture line.</td>
<td>None</td>
<td>Exported after surgery and lost to further follow-up.</td>
<td>Ran 10, won 1</td>
<td>Equal/improved</td>
</tr>
</tbody>
</table>
### Table 1: Cont.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (years)</th>
<th>Gender</th>
<th>Limb</th>
<th>Radiologic fracture description</th>
<th>Additional arthroscopic findings</th>
<th>Time of follow-up (months)</th>
<th>Outcome</th>
<th>Racing record</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>26</td>
<td>2</td>
<td>Filly</td>
<td>Bilateral</td>
<td>Left: incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment. Loss of infrastructure and increased density within adjacent parent bone. Mild enthesis capsular new bone dorsal Cr.</td>
<td>None</td>
<td>6</td>
<td>Returned to racing at 6 months.</td>
<td>Ran 1</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>27</td>
<td>3</td>
<td>Filly</td>
<td>Right</td>
<td>Complete displaced frontal plane fracture dorsoproximal radial facet C3. Loss of infrastructure within fragment. Loss of infrastructure within adjacent parent bone.</td>
<td>None</td>
<td>11</td>
<td>Returned to racing at 11 months.</td>
<td>Ran 1</td>
<td>Reduced</td>
</tr>
<tr>
<td>28</td>
<td>2</td>
<td>Colt</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment. Loss of infrastructure and increased density within adjacent parent bone. Remodelled dorsodistal Cr. Enthesious capsular new bone dorsal Cr.</td>
<td>None</td>
<td>7</td>
<td>Failed to train successfully due to poor fracture healing.</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>29</td>
<td>2</td>
<td>Gelding</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment. Loss of infrastructure and increased density within adjacent parent bone. Remodelled dorsodistal Cr. Irregular enthesis capsular new bone dorsal Cr.</td>
<td>None</td>
<td>14</td>
<td>Returned to racing at 8 months. Subsequent recurrence of injury.</td>
<td>Ran 2</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>30</td>
<td>1</td>
<td>Filly</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsoproximal radial facet C3.</td>
<td>None</td>
<td>15</td>
<td>Returned to racing at 8 months.</td>
<td>Ran 7, won 2</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>31</td>
<td>4</td>
<td>Gelding</td>
<td>Right</td>
<td>Incomplete nondisplaced frontal plane fracture dorsoproximal radial facet C3. Loss of density and increased density within fragment and adjacent parent bone.</td>
<td>Complete tear lateral branch MPICL</td>
<td>19</td>
<td>Returned to racing at 19 months.</td>
<td>Ran 1</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>32</td>
<td>2</td>
<td>Filly</td>
<td>Left</td>
<td>Incomplete nondisplaced frontal plane fracture dorsoproximal radial facet C3. Loss of infrastructure and increased density within fragment and adjacent parent bone.</td>
<td>Fragmentation at medial and lateral margins of fracture. Fragmentation dorsodistal Cr.</td>
<td>15</td>
<td>Returned to racing at 8 months.</td>
<td>Ran 2</td>
<td>Equal/improved</td>
</tr>
<tr>
<td>33</td>
<td>3</td>
<td>Filly</td>
<td>Left</td>
<td>Incomplete, comminuted, nondisplaced frontal plane fracture dorsodistal Cr. Loss of density and infrastructure within fragment. Loss of infrastructure and increased density within adjacent parent bone. Mild remodeling dorsodistal Cr. Enthesious capsular new bone dorsal Cr.</td>
<td>Unstable chip fracture dorsodistal Cr. cartilage defect dorsoproximal radial facet C3.</td>
<td>3</td>
<td>Retired to be a brood mare without attempt to train again.</td>
<td>n/a</td>
<td>n/a</td>
</tr>
</tbody>
</table>

Cr = radial carpal bone; C3 = third carpal bone; MPICL = medial palmar intercarpal ligament; LPICL = lateral palmar intercarpal ligament; C4 = fourth carpal bone; Ci = intermediate carpal bone. NAD = no abnormalities detected.
the dorsodistal radial carpal bone \( n = 25 \), dorsal margin of the radial facet of the third carpal bone \( n = 9 \) and intermediate facet of the distal radius \( n = 1 \). Proportionally more radial carpal bone fractures involved left carpi (20/25) than third carpal bone fractures \( (3/6) \). The radiological details and configuration of fractures are recorded in Table 1. All horses were lame; 14 unilaterally with lameness graded 1–7 (median 2) out of 10 and 13 were bilaterally lame in the forelimbs. All affected joints were distended (mild \( n = 9 \); moderate \( n = 18 \); marked \( n = 3 \); not recorded \( n = 5 \)) and in 9 horses there was thickening of the affected capsule.

Fig 3: Radiographs demonstrating 2 cases with incomplete nondisplaced frontal plane chip fractures of the dorsodistal aspect of the radial carpal bone. a) Flexed lateromedial projection (Case 33). b) dorsolateral-palmaromedial oblique projection (Case 29).

Fig 4: Radiographic examples of frontal plane chip fractures of the dorsoproximal aspect of the radial facet of the third carpal bone. a) Lateromedial projection (Case 19). b) Dorsolateral-palmaromedial oblique projection (Case 30). c) Flexed dorsal 30° proximal-dorsodistal oblique projection (Case 30).

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The radiological features of the fractures are displayed in Table 1. Fractures of the dorsodistal radial carpal bone were most readily identified and assessed in flexed lateromedial (Fig 3a) and dorsolateral-palmaromedial oblique (Fig 3b) projections. Fractures of the third carpal bone (n = 10) were sometimes recognised on lateromedial (Fig 4a) and dorsolateral-palmaromedial oblique (Fig 4b) projections but flexed dorsal 30° proximal-dorsodistal oblique projections (Fig 4c) were most useful for identification and assessment. Thirty-one fractures were radiologically classified as incomplete and nondisplaced, and 4 as complete.

In all cases fractures were readily identifiable arthroscopically. Additional arthroscopic findings, including the contralateral joint where applicable, are documented in Table 1. There were no post operative complications, and at the time of discharge (3–9 days post operatively, mean 5) all horses were walking comfortably.

Radiological follow-up information was available in 19 cases 3–9 (mean 5) months after surgery. Fractures exhibited satisfactory radiological healing in 18 of 19 cases (Figs 5, 6). Healing was typically characterised by infilling of the fracture line, and concurrent improvements in infrastructure and density of both the fracture fragment and adjacent parent bone. In 7 cases, fracture healing was incomplete, but at the time of examination 6 of these cases were ≤4 months since surgery. In 7 of 9 cases radiographed at ≥5 months after surgery fractures had healed completely. Clinical follow-up information was available for 28 of 33 animals at 3–54 (mean 15) months after surgery (Table 1). Twenty-three horses (82%) returned to racing 3–22 (mean 10) months after surgery. Nineteen horses (68%) raced at a level equal or better than before injury, and 4 at a reduced level. Two additional horses returned to training without reported problems, but were subsequently lost to follow-up. Two horses were retired as brood mares following surgery without attempting to train. Two horses suffered recurrence of injury (one following 2 races post operatively and one after resuming training). Of those with

Fig 5: Dorsolateral-palmaromedial oblique radiographic projections demonstrating an incomplete frontal plane chip fracture of the dorsodistal aspect of the radial carpal bone in Case 26. Over the 16 post operative weeks there is progressive fracture healing and improvement in osseous infrastructure within the fracture fragment a) preoperative. b) 8 weeks post operative. c) 12 weeks post operative. d) 16 weeks post operative.

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follow-up information, according to fragment location, 15 of 19 (79%) of horses with dorsodistal radial carpal bone fragments, and 7 of 8 (88%) with dorsoproximal third carpal bone fragments raced after surgery. Twelve of 19 (63%) with radial carpal bone fractures and 6 of 8 (75%) with third carpal bone fractures raced at a level equal or better than before injury.

Discussion

The primary indication for surgical treatment of osteochondral fragments in the carpus is to minimise articular insult and prevent the development of osteoarthritis (McIlwraith and Bramlage 1996). Removal has been considered the treatment of choice, and arthroscopic surgery affords the advantages of superior visualisation, minimal soft tissue trauma, effective lavage and early return to exercise (McIlwraith et al. 1987, 2005). However, removal of large fragments can lead to a degree of microinstability and progression to osteoarthritis (McIlwraith et al. 2005). Compression of articular fractures has also been shown to enhance cartilage healing (Mitchell and Shepard 1980). In the repair of slab fracture of the carpal bones, arthroscopic evaluation of the entire joint and fracture reduction are superior to open surgical techniques (Richardson 2002). Arthroscopy also permits adherence to the AO goal of atraumatic operative technique, accurate anatomic reduction, rigid internal fixation and early post operative ambulation.

In grading carpal articular deficits and assessing their influence on outcome, McIlwraith et al. (1987) reported that 20 of 37 horses (54%) with grade 4 lesions returned to racing at a level equal or better than before injury (grade 4 lesion = significant loss of subchondral bone), grade 3 (loss of ≥50% of the articular cartilage from the affected carpal bone) and grade 4 lesions had a significantly worse prognosis than grade 1 (minimal fibrillation or fragmentation at the edge of the defect left by the fragment, extending ≤5 mm from the fracture line) and grade 2 (articular cartilage degeneration extending >5 mm back from the defect and including up to 30% of the articular surface of that bone) lesions, with 53.2% of the former group compared to 71 and 75% of the latter groups, respectively, racing at an equal or improved level after surgery. Overall results of arthroscopic fragment removal according to location, for horses returning to racing at a level equal or better that pre-injury, are 55% for fragments from the dorsodistal radial carpal bone and 59% from the dorsoproximal third carpal bone (McIlwraith et al. 1987). The horses in the current series all
had grade 4 fragmentation and overall 68% returned to racing at a level equal or better than before injury. According to fragment location, 63% of horses with dorsodistal radial carpal bone fragments, and 75% with dorsoproximal third carpal bone fragments returned to racing at a level equal or better than before injury. These results compare favourably with those reported for removal of similarly sized fragments.

Carpal chip fractures commonly occur at joint margins previously altered by subchondral bone degeneration (Pool and Meagher 1990). It is postulated that exercise induced microdamage, which may manifest as subchondral disorganisation and densification, leads to osteochondral failure (Kawcak et al. 2000, 2001). This is considered important when determining the post operative management. In addition to the requirements for fracture healing, a suitable period of time and appropriate stimuli are required to enable re-establishment of osseous infrastructure in both the repaired fragment and parent bone. It should also be recognised that returning the horse to the previous training regime will reproduce the environment that originally resulted in subchondral failure and fracture. With the regime described above, there was a relatively low incidence (6%) of recurring clinical problems in the repaired bone, compared to 11% reported by McIlwraith et al. (1987) following fragment removal.

The reported results suggest a positive effect of the technique on horses’ recovery which is presumed to be the result of conservation of the articular surface and subchondral bone. However, continued mechanical support may also be offered by the screw location, although subchondral microfracture resulting from the insertion process or a combination of these processes may contribute to the reduced incidence of recurrent osseous failure noted in this series.

The convalescent period (i.e. time to first race) is frequently determined by the seasonal nature of Thoroughbred racing and may have resulted in longer periods of convalescence than would be required according to fracture healing and rehabilitation.

The difference in left to right distribution of fragments is interesting but cannot readily be explained. In the UK, horses race and train in both clockwise and anticlockwise directions with similar frequency. Greater numbers of cases are required to confirm the significance of this association.

Manufacturers’ addresses
1Karl Storz, Tuttlingen, Germany.
2Ethicon, Edinburgh, UK.
3Pressage, Jupiter Products, Cardiff, UK.
4Pitman-Moore Ltd, Crewe, Cheshire, UK.
5Arnolds, Shrewsbury, Shropshire, UK.

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CONCLUSIONS

The body of work presented documents some novel applications and developments of arthroscopic techniques in equine surgery. Previously unreported and/or unsubstantiated diagnoses are described, and information on incidence within a referral hospital provided. Therapeutic strategies are suggested, results following surgery reported and prognostic information obtained. From this work it is possible to draw a number of conclusions.

Advantages of novel endoscopic surgical applications over traditional techniques:

Work in the dissertation highlights the previously described advantages of arthroscopic surgery (see introduction) in novel situations: These include, but are not limited to, identification and removal of remote comminuted fragments in horses with fractures of the lateral malleolus of the tibia; assessment and management of other concurrent injuries when repairing carpal chip fractures, or when removing fractures of the lateral malleolus of the tibia; identification of foreign material or osteochondral pathology which had not been predicted pre-operatively in cases of synovial contamination and infection (Wright et al. 2003; Smith and Wright 2010; Wright and Smith 2010). With experience, surgical times are also shorter, as illustrated by comparison of navicular bursoscopy with the Streetnail procedure (Richardson et al. 1986; Wright et al. 1999). In turn, these advantages result in reduced patient morbidity (and less requirements for analgesic treatments), shorter periods of hospitalization, lower complication rates, reduced post-operative fibrosis, early mobilization, and improved functional outcome (Jackson 1974; O'Connor 1974). With particular reference to
tenoscopy and bursoscopy, early mobilization dramatically reduces adhesion formation following tendon surgery (Gelberman et al. 1983), and is greatly facilitated by endoscopic techniques.

Before publication of the first paper in this dissertation, synovial contamination and infection was managed infrequently with arthroscopy, but instead by combinations of needle lavage, arthrotomy, open drainage, and closed suction drainage (McIlwraith 1983; Schneider et al. 1992). Fractures of the lateral malleolus of the tibia were routinely removed by arthrotomy (Wright 1992). Development of endoscopic surgical techniques for these applications has afforded all of the above-described benefits (Wright et al. 2003; Smith and Wright 2010). In addition, in cases of synovial contamination and infection, endoscopic surgery has resulted in greatly improved rates of survival and return to athletic function (Wright et al. 2003).

There are minimal disadvantages of endoscopic surgery over open techniques. Surgeons are required to develop additional skill sets, including relearning of synovial anatomy and training in visuomotor techniques. In addition, significant levels of experience are necessary to be able to perform competently some of the complex endoscopic procedures described.

*Diagnosis of intra-thecal tendon injuries:* Currently, the accuracy of non-invasive imaging modalities, including ultrasonography and MRI, for identification of marginal injuries of the deep digital flexor tendon within both the digital flexor tendon sheath and navicular bursa is limited (Smith and Wright 2006; Smith et al. 2007; Smith and Wright 2011b). Progress has been made, but endoscopy still provides the most accurate appraisal.
of lesion morphology, assessment of severity, and thus, it is argued, the best correlation with prognosis. This situation is similar in man for injuries to intra-synovial tendons and ligaments. Examples include the biceps tendon, and the teres ligament within the hip joint (Byrd and Jones 2001; Kuo et al. 2004). The extra information obtained from endoscopy justifies early use in clinical cases.

Pathobiology and treatment of intra-thecal tendon injuries: Deep digital flexor tendon injuries within the navicular bursa and digital flexor tendon sheath are now considered to be frequent causes of bursitis and tenosynovitis respectively (Fortier et al. 1999; Wright and McMahon 1999; Wilderjans et al. 2003; Smith and Wright 2006; Smith et al. 2007; Smith and Wright 2011a). Experience with large numbers of cases has enabled classification of injuries according to lesion morphology, and treatment strategies have been developed. Marginal intra-synovial tendon injuries have been recorded elsewhere, including the tarsal sheath of the lateral (deep) digital flexor tendon (Cauvin et al. 1999), and the extensor carpi radialis tendon sheath (Platt and Wright 1997). In man, although marginal intra-synovial tendon injuries are relatively uncommon, some similarities are seen with the biceps tendon (Kuo et al. 2004; Ahrens and Boileau 2007).

Endoscopic debridement currently is the main stay of treatment for marginal intra-synovial tendon and ligament injuries, regardless of lesion location (McIlwraith 1992; Nixon 1994; Phillips and Wright 1994; Platt and Wright 1997; Whitton et al. 1997; Cauvin et al. 1999; Minshall and Wright 2006; Smith and Wright 2006; Smith et al. 2007). The rationales for surgical debridement of lesions are i) disrupted collagen within a synovial environment causes persistent irritation, and associated inflammation and
lameness (Evans et al. 1984) ii) the surface area of exposed disrupted collagen is proportional to the severity of synovitis (McIlwraith et al. 2005b) iii) it has been observed clinically that tendon does not heal well within a synovial environment (McIlwraith 1992; Phillips and Wright 1994; Wright 1995; Wright and McMahon 1999) iv) an intrinsic mechanism for removal of disrupted collagen from an intra-thecal synovial location has not been identified or reported. It is hypothesised that debridement facilitates the development of an inert scar over the affected area, and this is supported by observations at second-look arthroscopic procedures (Firth et al. 1991; Kannegieter and Colgan 1993; Smith and Wright 2011c). Reported results following surgery also justify debridement as a treatment (McIlwraith 1992; Platt and Wright 1997; Cauvin et al. 1999; Minshall and Wright 2006; Smith and Wright 2006; Smith et al. 2007). In man, complete and partial rupture of the ligamentum teres is a relatively common injury recognized arthroscopically, and debridement has produced similarly good results (Byrd and Jones 2001; Byrd and Jones 2002). It should however be acknowledged that there are no studies with controls i.e. arthroscopically diagnosed tendon / ligament tearing which was not debrided.

Within both the navicular bursa and digital flexor tendon sheath, the prognosis for marginal intra-synovial tendon injuries appears related to the extent of deep digital flexor tendon disruption (Smith and Wright 2006, 2011a). There also appears to be a relationship between lesion characteristics and outcome; those with features characteristic of degenerative tendinopathy (Khan et al. 1999; Rees et al. 2006; Blunden et al. 2009) seem to have a worse prognosis (Smith and Wright 2011d). The results obtained after surgery which are presented here for both extensive traumatic lesions and proposed
degenerative lesions are modest only (Smith and Wright 2006; Smith et al. 2007; Smith and Wright 2011a). Therefore, it would seem logical to look towards development of regenerative therapies. Stem cell treatment appears well suited in theory, but evidence of efficacy is lacking, and in vivo survival of mesenchymal cells appears limited (Guest et al. 2010). It has subsequently been suggested that mesenchymal stem cells may have the ability to orchestrate regenerative processes before their demise, but evidence is limited (Ohtaki et al. 2008; Shi and Li 2008). Evidently further refinement of the technique is required. Embryonic stem cells may offer a more efficacious means of inducing regeneration, as superior tropism for injured tissue and more prolonged cell survival have been reported (Guest et al. 2010). Further research into this area is justified and required.

An alternative treatment, which has been performed in a limited number of cases, is repair using open surgical techniques (Wright and McMahon 1999). This was discontinued, as results appeared inferior (Smith and Wright 2006). However, information from a greater number of cases shows that the outcomes seen actually compare reasonably with endoscopic debridement alone for extensive tears of the deep digital flexor tendon (Wright and Smith 2011). The proposed advantages of repair were based on the observation that intrinsic tendon repair mechanisms, derived primarily from the epitenon, promoted tendon healing following surgical repair in experimental conditions (Gelberman et al. 1983). The current modest success seen with debridement of extensive lesions justifies revisiting the repair option. Development of an endoscopic technique, although technically challenging, might offer further advantages such as reduced morbidity and development of restrictive fibrosis, early mobilization, and lower complication rates (Starke et al. 2009; Anderson et al. 2010).
Repair of intra-articular fractures under endoscopic guidance: At their outset in 1958, the AO formulated four treatment principles to improve the results of fracture repair, and internal fixation in particular. They were i) anatomical reduction ii) stable internal fixation iii) preservation of blood supply iv) early active pain-free mobilization (Allgower et al. 1990). These dovetail well with arthroscopic surgical techniques, which have been described for repair of fractures of the third carpal bone, third metacarpal and metatarsal condyles, and mid-body fractures of the proximal sesamoid bones (Richardson 1986, 2002; Busschers et al. 2008). Although the latter has only recently been described, the techniques for the former two are accepted as the standard of surgical care. Advantages reported include superior evaluation of the joint, more accurate reduction / reconstruction of articular surfaces, reduced soft tissue trauma, less scar tissue, and the ability to fully debride all regions of the affected joint (Richardson 2002). Arthroscopically guided repair is also the standard of treatment in man for fractures in the knee, shoulder, hip, wrist, elbow and ankle, with the same advantages ascribed to the technique (Berkowitz and Bottoni 2004).

Slab fractures of the third carpal bone can be removed, but it is generally accepted that whenever articular surfaces can be accurately and safely reconstructed, this should be the primary treatment goal (Mellwraith et al. 2005a). Although not previously considered, it has been possible to successfully transfer the principles used for repair of slab fractures of the third carpal bone, to large chip fractures of the third and radial carpal bones, and also the distal radius (Wright and Smith 2010). The improved results obtained are likely to be as a result of conservation of the articular surfaces, prevention of micro-instability and
thus progression to osteoarthritis (McIlwraith et al. 1987; McIlwraith et al. 2005a). In light of this work, application to other locations warrants investigation.

In conclusion, arthroscopic surgery has revolutionized surgical treatment of disorders of joints, tendon sheaths and bursae. As experience, and confidence has grown, novel applications such as those presented in this dissertation have received further attention. With each step forward further ideas often are formulated, and undoubtedly development and refinement of endoscopic surgical techniques will continue to benefit many patients in the future. Accurate observations on clinical disease processes and continual review of understanding of healing processes are central to progress. This dissertation outlines the authors’ attempts to follow this philosophy.
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Manuscript (Appendix 3).


of lesions of the deep digital flexor tendon in the navicular bursae of 20 lame


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Appendix 1: Digital flexor tendon sheath tenoscopy; 271 cases

The authors of the original paper have now collected data from 271 tenoscopic examinations of the digital flexor tendon sheath, performed between 1996-2009.

Table 1: Tenoscopic diagnoses in 271 digital flexor tendon sheaths

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Torn DDFT</td>
<td>84 (31%)</td>
</tr>
<tr>
<td>Torn Manica Flexoria</td>
<td>64 (24%)</td>
</tr>
<tr>
<td>Disrupted sheath wall</td>
<td>37 (14%)</td>
</tr>
<tr>
<td>Torn SDFT</td>
<td>21 (8%)</td>
</tr>
<tr>
<td>Torn DDFT plicae</td>
<td>7 (3%)</td>
</tr>
<tr>
<td>Degenerative DDFT tendinopathy</td>
<td>5 (2%)</td>
</tr>
<tr>
<td>Intrathecal disruption of the SDSL</td>
<td>4 (2%)</td>
</tr>
<tr>
<td>Torn digital manica</td>
<td>3 (1%)</td>
</tr>
<tr>
<td>Contamination and infection</td>
<td>28 (10%)</td>
</tr>
</tbody>
</table>

DDFT = deep digital flexor tendon; SDFT = superficial digital flexor tendon; SDSL = straight distal sesamoidean ligament

Surgical technique and treatment principles have remained the same as described in the preceeding manuscript.

Deep digital flexor tendon tears: Approximately 66% of tears of the deep digital flexor tendon occurred on its lateral margin and proximal to the sesamoidean canal. The majority of these commenced beneath the manica flexoria and extended varying distances distally. The depths of the tears varied between surface disruption and deep clefts which
extended into the body of the tendon. In approximately 30% of cases focal granulomata were present at the proximal and/or distal margins of the tear. There was approximately 2:1 ratio of forelimbs to hindlimbs.

_Tearing of the manica flexoria:_ 85% of tears occurred in hindlimbs. Disruption was complete in 71% of cases and partial in the remainder; 50% of tears were medial, 30% lateral and 20% proximal and/or biaxial.

_Sheath wall tearing:_ This was most common at the proximal reflection and was frequently associated with asymmetric proximal outpouching which became exaggerated when the sheath was maximally distended. Palmar disruption proximal to the palmar or plantar annular ligament was the second commonest site, again frequently predicted by the presence of free fluid palmar to the sheath wall reflection from the superficial digital flexor tendon (outwith the normal sheath confines).

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Sound and working</th>
<th>Returned to previous levels of performance</th>
</tr>
</thead>
<tbody>
<tr>
<td>DDFT tear</td>
<td>47%</td>
<td>38%</td>
</tr>
<tr>
<td>Short tear</td>
<td>74%</td>
<td>63%</td>
</tr>
<tr>
<td>Long tear</td>
<td>28%</td>
<td>18%</td>
</tr>
<tr>
<td>Torn manica flexoria</td>
<td>84%</td>
<td>78%</td>
</tr>
</tbody>
</table>

DDFT = deep digital flexor tendon
Endoscopic evaluation of the navicular bursa; observations, treatment and outcome in 92 cases

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Key Words: endoscopy; navicular bursa; deep digital flexor tendon; surgery

WORD COUNT: 4121

Summary

Reasons for performing study: Endoscopic examination of the navicular bursa for the investigation of lameness localising to this region has to date been described in a limited number of cases only. Review of a greater number of cases is needed to define the potential contribution to case management, and to provide prognostic values.

Objectives: To report i) the clinical and endoscopic findings in a series of clinical cases ii) the surgical techniques performed and outcome after surgery, and iii) prognostic values for the injuries identified. The authors hypothesise that i) lameness localising to the navicular bursa is commonly associated with lesions of the dorsal border of the DDFT ii) endoscopy allows the extent of injuries
to be assessed and treated iii) case outcome is related to the severity of DDFT injury iv) the technique is safe and there is little associated morbidity.

Materials and Methods: All horses which underwent endoscopy of a forelimb navicular bursa for the investigation of lameness (excluding contamination and infection), were identified. Case files were reviewed and those with injuries within the navicular bursa identified at surgery were selected for further analysis.

Results: 114 horses which underwent diagnostic navicular bursoscopy were identified of which 92 had injuries identified within the navicular bursa. DDFT injuries were identified in 98% of abnormal bursae. Of those cases examined with MRI (28) 56% had combination injuries involving the DDFT and navicular bone. 61% of horses returned to work and were sound, with 44% returning to a level of performance equal or greater than achieved before surgery. Horses with extensive tearing of the DDFT, and those with combination injuries of the DDFT and navicular bone identified with MRI, had worse outcome measures.

Conclusions: Lameness localising to the navicular bursa is commonly associated with injuries to the dorsal border of the DDFT. Endoscopy permits identification and characterisation of injuries within the navicular bursa, and enables appropriate lesion management. Outcome following surgical debridement is related to the severity of injury, but overall is reasonable.

Potential Relevance: Horses with lameness localising to the navicular bursa may have tears of the DDFT. Bursoscopy is able to contribute diagnostic and prognostic information to case management, and debridement of lesions improve outcome compared with non-surgical treatment regimes.

Introduction
Lameness resulting from injuries involving components of the forelimb navicular apparatus is common (Ostblom et al. 1984; Wright 1993a; Dyson et al. 2005; Dyson and Murray 2007). Gross pathological and histomorphometric examinations have documented a variety of injuries affecting the dorsal, intra-synovial surface of the deep digital flexor tendon (DDFT) and the opposing palmar (flexor) fibrocartilage of the navicular bone (Wright et al. 1998). Some of these have been subsequently diagnosed ante mortem with computed tomography (CT) (Widmer et al. 2000; Eliashar et al. 2006; Puchalski et al. 2007; Puchalski et al. 2009) and magnetic resonance imaging (MRI) (Dyson et al. 2003a; Busoni et al. 2005; Mair et al. 2005; Dyson and Murray 2007; Sherlock et al. 2007; Sampson et al. 2009).

Latterly, a surgical technique suitable for the investigation of lameness localising to this region has been described (McIlWraith et al. 2005c), and results in a limited number of clinical cases have been reported (Smith et al. 2007). The aims of this study are to report with more substantial numbers i) the clinical and endoscopic findings in a series of clinical cases ii) to describe the surgical techniques performed and outcome after surgery, and iii) to define prognostic values for injuries identified. The authors hypothesise that i) lameness localising to the navicular bursa is commonly associated with lesions of the dorsal border of the DDFT ii) endoscopy allows the extent of injuries to be assessed and treated iii) case outcome is related to the severity of DDFT injury iv) the technique is safe and there is little associated morbidity.

**Materials and Methods**

**Inclusion criteria:** All horses which underwent endoscopy of forelimb navicular bursae (n = 114), for the investigation of lameness (excluding contamination and infection), by the first (n = 67) or second (n = 47) author between 1999 and 2010 were identified. Case files were reviewed and
those with injuries within the navicular bursa identified at surgery were selected for further analysis (n = 92 horses, n = 105 bursae). 19 of these cases have been reported previously (Smith et al. 2007).

Case selection: Surgery was recommended in horses which met all of the following criteria; i) unilateral or bilateral forelimb lameness ii) lameness localised by regional anaesthesia to the foot iii) absence of radiological features of navicular disease (Wright 1993b), with in addition either a positive response to analgesia of the navicular bursa, or MRI features which were considered to be consistent with an intrathecal bursal lesion.

Diagnostic Procedures: All horses were subjected to a lameness examination, diagnostic analgesia and radiography of both front feet as described previously (Smith et al. 2007). Ultrasonography of the DDFT was carried out from the palmar aspect of the pastern (Bolen et al. 2007) in 36 horses using a 10MHz linear array probe (GE Healthcare¹). Nuclear scintigraphic examination of both front feet using dorsal and lateral pool phase, and dorsal, lateral and solar bone phase images was performed in 4 horse using Hermes² imaging software (Dyson 2002). CT examination of both front feet was performed in 8 horses under general anaesthesia using a 4th generation helical scanner (Phillips³); an axial scan was performed using parameters of 120 kV, 150 MA and a 2 second scan time. Slice thickness was set at 4 mm and a pitch of 2 mm. The data set was then reconstructed using a reconstruction index of 4 mm to give transverse slices. Standing low field MRI was used to examine both front feet of 39 horses with a 0.27 Telsa open C-shaped magnet and dedicated hoof radiofrequency coil (Hallmarq⁴) as described previously (Mair et al. 2005). High field MRI was used to examine both front feet of 4 horses under general anaesthesia with a 1.5 Telsa magnet (GE Healthcare¹) (Dyson et al. 2003b).
Surgical technique: Endoscopy of the navicular bursa was performed in all horses under general anaesthesia using a transthecal approach (McIlWraith et al. 2005c; Smith et al. 2007) (Figure 1a and 1b).

Synovial fluid was frequently xanthochromic. Synovial changes (hyperaemia, proliferation, haemorrhage or haemosiderin staining) were common but varied between cases. Any lesions identified were further defined by palpation with an arthroscopic probe (Karl Stortz). Injuries of the DDFT varied in appearance, and were subjectively classified as linear splits, dorsal border fibrillation, and small (shallow tears involving only a small part of the surface area of the affected lobe of the DDFT) (Figure 1b) or extensive dorsal border tears (Figure 2a). Large pieces of disrupted tendon protruding into the bursa were sectioned with arthroscopic scissors (Sontec) or fixed blade meniscectomy knives before removal with arthroscopic rongeurs (Scanlan). The margins of tears, splits and smaller lesions were debrided (Figure 2b) with a motorised synovial resector (Dyonics) with suction applied. Lesions of the fibrocartilage were defined as partial or full thickness. Where necessary, arthroscopic and instrument portals were interchanged to optimize lesion access. At the end of surgery, skin portals were closed with simple interrupted sutures of 3 Metric monofilament polyamide (Ethicon) or polypropylene (SurgiPro) and limbs dressed with light sterile bandages enclosing the foot. All horses recovered unassisted from general anaesthesia.

Additional surgical procedures

Navicular suspensory ligament desmotomy was performed in 2 cases (Wright 1993c), distal interphalangeal joint arthroscopy (McIlWraith et al. 2005b) in 14 cases, and tenoscopic examination of the digital flexor tendon sheath (McIlWraith et al. 2005d) in 1 horse.

Postoperative care

Antimicrobials in the form of penicillin (20000-30000 iu/kg bwt i.v. q. 8h) (Crystapen) alone or in combination with gentamicin (6.6 mg/kg bwt i.v. q. 8h) (Genta Equine) depending on clinic...
protocol, commenced pre-surgery and continued for 24-72 h. All horses received a single pre-operative dose of phenylbutazone (Equipalazone\textsuperscript{13}) (4mg/kg bwt i.v.). Post-operative analgesia was decided on an individual case basis. Remaining post-operative care and exercise were performed as described previously (Smith et al. 2007).

Data analysis

Follow-up information was obtained by telephone questionnaire with owners (n = 89), detailing complications, residual lameness, current activity and, when appropriate, time to return to work. Case outcomes were classified as either, failure (including those that were persistently lame, sound but receiving NSAID treatment, neurectomized or failed to return to work for any other reason), or sound and working. Horses in the latter category were further subdivided into working at previous levels of performance, or at lower levels. Results were converted into binary data and tabulated, before analysis using a Chi squared test or Fisher’s exact test when individual exposure outcomes were small. Significance was assumed when P<0.05.

Results

Horses were 5-17 (mean 10.5) years old and included Warmbloods (n = 22), Thoroughbred crossbreeds (n = 18), Ponies (n = 6), Irish Sports Horses (n = 5), Thoroughbreds (n = 3), Arabians (n = 1), Welsh Cobs (n = 3), Irish Drafts (n = 3), Quarter Horses (n = 3), Trakheners (n = 1), Draft crossbreeds (n = 1), Trotters (n = 1) and Arabian crossbreeds (n = 1). In 23 cases breed was not recorded. There were 32 mares, 52 geldings and one entire male; 7 genders were not recorded. Horses were used for a variety of purposes; general purpose (n = 33), eventing (n = 14), dressage (n = 7), showjumping (n = 6), hunting (n = 3), western riding (n = 3) and endurance (n = 1) (unknown n = 24).
Animals had been lame for 1 to 24 (mean 6.1) months prior to examination. In 62 horses the onset of lameness was reported as acute and of moderate or severe intensity. In 13 horses lameness was intermittent and insidious in onset and in 17 it was unknown. Prior to admission horses had received a variety of treatments; the majority had undergone periods of rest and received various farriery attention.

Clinical details

Of the 92 cases, 79 were affected unilaterally and 13 bilaterally. All animals were lame when trotted in a straight line on a firm surface (range 1 to 9, median 3, out of 10).

Palmar digital analgesia of the lame or lamest limb produced an improvement in lameness in 71/73 cases in which it was performed; improvement was slight (n = 5), partial (n = 22) or complete (n = 44). Residual lameness was abolished by analgesia of the palmar nerves at the level of the proximal sesamoid bones in all except 4 horses. Overall analgesia of the palmar nerves at the level of the proximal sesamoid bones was performed in 33 horses and resulted in partial improvement in 5 horses and complete improvement in 27. The 5 horses in which lameness was not alleviated by blockade at this level all had additional causes of lameness further proximal in the affected limb.

Intra-articular analgesia of the DIP joint was performed in 69 horses and produced an improvement in lameness in 61 (slight n = 9; partial n = 31; complete n = 21). Intra-thecal analgesia of the navicular bursa (n = 63) produced a slight (n = 3) partial (n = 29) or complete (n = 31) improvement in lameness.

Diagnostic imaging

Radiography identified no osseous abnormalities involving the navicular apparatus in 71/105 feet. Those recorded were: remodelling of the wings of the navicular bone (n = 13); irregularity (n = 6), erosion (n = 3) or thickening (n = 1) of the palmar cortex; medullary densification (n = 6); enlarged or increased number of synovial fossae (n = 5); enthesopathy at the proximal (n = 3) or distal
borders (n = 3); large medullary lucency (n = 2); distal border fragment (n = 1); irregular DDFT (n = 1) or distal sesamoidean impar ligament (n = 1) insertions. Ultrasonographic examination was performed in 41 limbs. The following abnormalities were identified; enlargement of one lobe of the DDFT (n = 7), distension of the navicular bursa (n = 4), DDFT core lesion (n = 3), peritendinous thickening (n = 2), irregular dorsal (n = 1) or palmar (n = 1) margin of the DDFT. No abnormalities were identified in 28 legs. Scintigraphic examination was unremarkable in 1 affected foot. In 3 there was increased radiopharmaceutical uptake associated with the navicular bone, and in 2 there was also increased uptake at the insertion of the DDFT.

CT

CT identified dorsal border tearing of 1 (n = 5) or both lobes (n = 2) of the DDFT within the navicular bursa. In 2 limbs lesions in the DDFT were identified distal to the navicular bone, and in 1 there was irregular new bone at the insertion of the DDFT.

MRI

High field MRI identified lesions of the dorsal border of the DDFT within the bursa in all 4 feet, navicular bone lesions in 3 feet, DIP collateral desmitis in 3 feet, CSL desmitis in 1 foot and DIP peri-articular remodelling in 1 foot. Details of low field MRI findings are described elsewhere (Smith and Wright 2011a). In summary, these included DDFT tendonitis in 44 feet, and abnormalities of the navicular bone in 32 feet. In 7 feet there was of desmitis of the DSIL, and in 10 desmitis of the CSL. Adhesions within the bursa were seen in 11 feet. In 28 limbs there were concurrent DDFT and navicular bone lesions.

Endoscopic findings

DDFT lesions were seen in 103/105 (98%) bursae. Sagittal splits in the DDFT were found in 7 bursae. These commonly were associated with minimal synovial changes and were located adjacent
and proximal to the navicular bone. With sagittal splits there was little or no tissue extruded into the bursa, and thus minimal surgical interference was performed. Fibrillation of the dorsal border was seen in 11 bursae again with minimal synovial changes. Fibrillated areas were usually opposite and proximal to the navicular bone. The most common lesions were sagittal tears of the DDFT, and these were seen in 87 bursae; 24 were classified as small and 63 as extensive. Sagittal tears all involved the tendon proximal to the bone and extended variable distances distally. Torn collagenous tissue was extruded into the bursa and tended to recoil proximally frequently presenting as clumps adjacent to the T ligament (Figure 1a and 2a). Tears were usually wider proximally, and narrowed as they extended distad. Less commonly, extruded torn tendon fibrils were evident along the margins of the tear. In 6 cases the dorsal border of the DDFT was discoloured, friable, and ruptured with gentle probing to expose underlying torn tendon. In some cases the proximally recoiled fibres had granulomatous caps (n = 16) (Figure 3). Large strands of torn tendon occasionally were found adhered to the T-ligament, CSLs or to the contralateral lobe of the DDFT (Figure 4). Fibrinous synovio-synovio adhesions were occasionally seen associated with DDFT tears and were removed. Cartilaginous lesions were seen in 15 bursae; 10 were partial thickness and 2 were full thickness. In 1 bursa a cartilaginous lesion was the sole abnormality and in another the only abnormal finding was tearing of the T-ligament.

Arthroscopy and tenoscopy

Of the 16 distal interphalangeal joints examined arthroscopically, abnormalities were identified in 7; cartilage softening, thinning or fibrillation (n = 6), fragmentation of the extensor process (n = 3). No abnormalities were detected in the other 9 joints, or in the 1 digital flexor tendon sheath.

Complications

Minor iatrogenic damage to the fibrocartilage of the navicular bone was recorded in 5 bursae. Injury to the palmar digital vessels during portal creation was reported in 1 horse. This was not identified
until after surgery due to use of esmarch bandages and tourniquets, and was controlled by application of further counter pressure bandaging. Skin portals became infected in 4 cases and all resolved following treatment with enrofloxacin (5mg/kg i.v. SID) (Baytril\textsuperscript{14}). In 3 horses there were greater than expected degrees of lameness following surgery, requiring additional phenylbutazone. In 1 case each, the following complications were seen after surgery; transient ataxia, laminitis, air embolism, radial neuropathy, colic. All resolved uneventfully with supportive medical treatment.

Follow-up

Follow-up information was obtained for 89 horses. 13 had not had sufficient time to return to work and were still convalescing. Seventy-six cases were therefore classified according to outcome. Of these, 1 horse was euthanized immediately after surgery due to a poor prognosis, and a further 4 horses were considered unsuccessful by the owners within 4 months of surgery, prompting no further opportunity for convalescence. Follow-up information for the remaining horses ranged from 5-46 (mean 17) months after surgery. Sixty-eight cases had greater than 12-month follow-up information.

Thirty cases were classified as failures. Forty-six horses (61\%) were sound and working, with 33 (44\%) performing at a level equal or greater than achieved before surgery. Eight (11\%) were performing at lower levels at the owners’ choice and 3 (7\%) had been unable to reach previous levels of performance. Of the failures, 9 were sound and working with additional treatment (7 were receiving daily NSAID treatment and 2 had subsequently undergone palmar digital neurectomy).

Evaluation of case variables on outcome measures identified that horses with extensive DDFT tears were significantly less likely to be classified as sound and working (50\%) compared to those with small tears (75\%) (P = 0.044). Horses with a combination of DDFT and navicular bone injuries were significantly less likely to be classified as returned to work at previous levels of performance (25\%) compared to those with DDFT injuries alone (74\%) (P = 0.011).
Discussion

As knowledge of causative lesions of foot lameness has evolved, lesions close to the insertion of the DDFT, within and adjacent to the navicular bursa, have been reported in the recent literature with increased frequency (Wright et al. 1998; Dyson et al. 2003a; Busoni et al. 2005; Mair et al. 2005; Blunden et al. 2006; Murray et al. 2006; Dyson and Murray 2007; Bell et al. 2009; Blunden et al. 2009; Boswell 2009; Sampson et al. 2009; Gutierrez-Nibeyro et al. 2010).

The criteria used for selection of cases for surgery in this study appear justified by the high positive identification rate of lesions in operated bursae, and the potential therapeutic options offered by endoscopic surgery. There are similarities between cases in the current series, and other published reports (Dyson et al. 2005; Bell et al. 2009; Boswell 2009; Gutierrez-Nibeyro et al. 2010). However, in contrast to those reports, cases in the current series were not all selected by results of MRI. Comparisons must therefore be made with a degree of caution.

Endoscopy of the navicular bursa was first described in 1999, and was applied with beneficial effects for management of contamination and infection of the navicular bursa following solar wounds (Wright et al. 1999). In 2001, the diagnostic potential of navicular bursa endoscopy was evaluated in cadaver limbs, and it was concluded to be a potentially useful technique for investigation of clinical cases with early podotrochleosis (Cruz et al. 2001). In 2003, an endoscopic approach through the digital flexor tendon sheath (performed blindly) was reported (Rossignol and Perrin 2003), along with endoscopic findings in 6 clinical cases with navicular disease. A transthecal approach in which a window is created in the T-ligament under endoscopic guidance was described by McIlwraith et al. (2005c). This technique was used in a series of 20 cases reported which underwent endoscopy of the navicular bursa for assessment and treatment of lesions of the DDFT (Smith et al. 2007), and also in all of the cases reported in the current series. A further report of a single case was recently published using a similar approach (Scandella et al. 2010). In a cadaver study, improved visualisation of the navicular bursa, and induction of less iatrogenic damage was demonstrated when using a transthecal approach compared to a direct approach (Haupt...
The benefits of a transthecal approach are i) improved visualisation of the proximal recess of the navicular bursa, and ii) less interference of arthroscope and instruments when operating lesions in the proximal bursa. A large window needs to be created in the T-ligament to afford these advantages, allowing the arthroscope to be positioned in the distal DFTS viewing into the bursa.

Endoscopy of the navicular bursa is technically demanding, but once mastered can be performed efficiently and with minimal complications. Iatrogenic damage to the fibrocartilage of the navicular bone occurred as an infrequent surgical complication. Restricted space for arthroscope and instrument manipulations has previously been reported (Smith et al. 2007), and was experienced in further cases from the current series. The cause of this restriction is uncertain, but the authors suspect it is related to enlargement of the collateral sesamoidean ligaments, DDFT, T-ligament and synovial and fibrous lining of the bursa. This restriction precluded in some cases complete examination of the fibrocartilage of the navicular bone and the distal aspect of the bursa. However, due to the propensity for torn collagenous material to recoil into the proximal part of the bursa, this was not considered to be a major limiting factor. Iatrogenic damage to the palmar digital vessels occurred in only 1 case in the current series. Damage to the palmar digital nerves was not identified in any case. The authors recommend careful palpation of the palmar digital neurovascular bundles before creating arthroscopic and instrument portals to minimize the risk of traumatizing these structures.

A variety of lesions, of differing severities, were seen endoscopically. The majority were linear tears of the DDFT, most commonly with torn tendon recoiled into the proximal bursa. It appears that this is not recognised with MRI. Proximal recoil of torn DDFT fibrils has long been recognised in cases of navicular disease (Oxspring 1935). In the current study tears were seen in the absence of identifiable fibrocartilage or navicular bone lesions. The authors hypothesise that fibres recoil as a consequence of the mechanical affect of the DDFT moving distad with respect to the opposing surface of the navicular bone, during the caudal weight-bearing phase of the stride. Sagittal tears,
linear splits and dorsal border fibrillation were encountered less commonly. These lesions are consistent with observations made by others with MRI and/or in pathological studies (Wright et al. 1998; Dyson et al. 2003a; Busoni et al. 2005; Blunden et al. 2006; Murray et al. 2006; Dyson and Murray 2007; Blunden et al. 2009; Sampson et al. 2009).

The rationale for surgical debridement of lesions are that i) disrupted collagen within a synovial environment causes persistent irritation, inflammation and lameness (Evans et al. 1984) ii) the surface area of exposed disrupted collagen is proportional to the severity of synovitis (McIlwraith et al. 2005a) iii) it has been observed clinically that tendon does not heal well within a synovial environment (McIlwraith 1992; Phillips and Wright 1994; Wright 1995; Wright and McMahon 1999) iv) there has been no identified or reported functional intrinsic mechanism for removal of disrupted collagen within synovial locations. The authors hypothesise that debridement facilitates the development of an inert scar over the affected area, and this is supported by observations at second-look arthroscopic procedures (Firth et al. 1991; Kannegieter and Colgan 1993; Smith and Wright 2011b).

The goals of treatment with surgery differ from methods used in other reports of similar cases. Boswell (2009) and Dyson et al. (2005) managed cases conservatively by providing physical support and a change of loading characteristics with farriery techniques. This was combined with a period of rest and controlled exercise, common to management of many tendon and ligament disorders. The goal of treatment was to allow intrinsic healing of the injured tendon, in a mechanically optimized environment. Bell et al. (2009) and Gutierrez-Nibeyro (2010) medicated affected bursae with corticosteroids, with the aim of suppressing inflammation and providing symptomatic relief. Although reasonable short-term success was achieved with the latter, long term success was poor. Outcome of cases undergoing surgical debridement compares favourably to those reported in these other series (Dyson et al. 2005; Bell et al. 2009; Boswell 2009; Gutierrez-Nibeyro et al. 2010).
In conclusion, endoscopy of the navicular bursa offers diagnostic information that cannot currently be obtained with other modalities and permits rationalized targeted intervention. It is minimally invasive, associated with little patient morbidity, and appears to have merit in management of clinical cases.

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References


Figure 1: a) Endoscopic view of a tear of the DDFT. Torn fibres have recoiled proximally and gathered as a clump. A transthecal approach allows the arthroscope to be positioned in the distal DFTS, providing the best perspective of the tear.  b) Interference of arthroscope and instrument when operating in minimised by using a transthecal approach. N = navicular bone; T = torn fibres; L = T-ligament; G = granuloma

Figure 2: a) Endoscopic view of a linear tear of the DDFT. Torn fibres have recoiled proximally and gathered as a clump. There is haemorrhagic staining of the disrupted fires. b) Following debridement a trough shaped defect can be seen in a linear configuration in the DDFT.

Figure 3: Endoscopic view of a tear of the DDFT. Torn fibres have recoiled proximally, and developed a granulomatos cap of tissue.

Figure 4: Endoscopic view of a tear of the DDFT. Torn tendon fibres have recoiled proximally and adhered in a transverse orientation across the bursa, to the contralateral lobe of the DDFT. A = adhesion
**Manufacturer's address**

1 GE Healthcare, Buckinghamshire, UK.
2 Hermes Medical Solutions, Stockholm, Sweden
3 Philips, Guildford, UK.
4 Hallmarq, Guildford, UK.
5 Karl Storz, Tuttlingen, Germany.
6 Sontec, Englewood, Colorado, USA.
7 Scanlan, St Paul, Minnesota, USA.
8 Dyonics, Smith & Nephew, London, UK.
9 Ethicon, Edinburgh, UK.
10 Covidien, Dublin, Ire.
11 Pitman-Moore Ltd., Crewe, Cheshire, UK.
12 Cp-pharma, Burgdorf, Germany.
13 Arnolds, Shrewsbury, Shropshire, UK.
14 Bayer, Newbury, Berkshire, UK.
Endoscopic evaluation of the predictive value of standing low field MRI for injuries within the navicular bursa

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Key Words: navicular bursa; deep digital flexor tendon; MRI; endoscopy; surgery

WORD COUNT: 1911

Summary

Reasons for performing study: In recent years there has been increasing reliance on magnetic resonance imaging (MRI) for investigation of lameness localising to the foot in horses. Injuries are frequently identified within the navicular bursa, but to date there remains limited correlative information to directly visualised lesions.
Objectives: To correlate endoscopic findings in the navicular bursa of horses with lameness localising to this region, with surface abnormalities of the structures comprising the bursa detected pre-operatively by standing low field MRI. The authors hypothesise that MRI accurately predicts intra-bursal injuries, but that superior morphological characterization is provided by bursoscopy.

Methods: Horses which underwent standing low field MRI, and endoscopic examination of the navicular bursa, for the investigation of lameness were identified. Using endoscopic observations as the defining standard, the reliability of low field MRI for detection of injuries within the navicular bursa was evaluated.

Results: 51 feet from 39 lame horses were examined with standing low field MRI and endoscopy of the navicular bursa. For surface injuries to the dorsal border of the deep digital flexor tendon, MRI had reasonable sensitivity, specificity, and predictive values. MRI had poor specificity and positive predictive values for detection of lesions of the fibrocartilage of the navicular bone and adhesions within the navicular bursa.

Conclusion: Low field MRI plays a useful role in the investigation of lameness localising to the navicular region in horses, but also has limitations in accurately defining intra thecal lesions.
Potential relevance: The limitations in accuracy of standing low field MRI for diagnosing intra thecal injuries of the navicular bursa should be considered when recommending and interpreting the results of imaging in clinical practice.

Introduction

Lameness resulting from injuries involving the navicular apparatus are common. They are frequently and increasingly diagnosed in clinical practice with low field magnetic resonance imaging (MRI) performed in the standing patient (Mair et al. 2005a; Sherlock et al. 2007). Previous work has correlated gross and histopathology with high field MRI abnormalities in lame and non-lame horses (Murray et al. 2006; Blunden et al. 2009; Dyson et al. 2010). Little correlative information has been published for low field MRI (Murray et al. 2009), and for both high and low field MRI there is sparse information regarding surface injuries of the deep digital flexor tendon (DDFT), and other intra-bursal injuries which commonly are described with MRI in clinical practice (adhesions, soft tissue proliferation, navicular fibrocartilage lesions).

The aims of this study are to correlate endoscopic findings in the navicular bursa of horses with lameness localising to this region, with surface abnormalities of the structures comprising the bursa detected pre-operatively by standing low field MRI. The authors hypothesise that MRI accurately predicts intra-bursal injuries, but that superior morphological characterization is provided by bursoscopy.

Materials and Methods
Case selection: All horses referred to the authors which underwent standing low field MRI and endoscopy of the navicular bursa (n = 39), for the investigation of lameness, between 1999 and 2010 were identified.

MRI examination: All cases had both front feet examined under standing sedation with a 0.27 Telsa open C-shaped magnet and dedicated hoof radiofrequency coil (Hallmarq1) as described previously (Mair et al. 2005b). Images were obtained at 8 different clinics (Clinic a 12; b 11; c 8; d 3; e 2; f 1; g 1; h 1) and interpreted in all cases by clinicians experienced in low field MRI. Sequences obtained included gradient echo (GRE) T1 weighted, GRE T2*-weighted, fast spin echo (FSE) T2 weighted and FSE short tau inversion recovery (STIR), in sagittal, transverse and frontal planes.

DDFT lesions were classified as dorsal border fibrillation (irregular dorsal border of the DDFT within the navicular bursa), parasagittal splits (hyperintense signal extending from the dorsal to palmar margins of the tendon on T1 or T2* pulse sequences), dorsal border tears (disrupted dorsal border of the DDFT, with irregular tissue extending from this into the bursa) and core lesions (central hyperintense signal on T1 or T2* pulse sequences, within one lobe of the DDFT, without disruption of the tendon margin at the level of the lesion).

Surgical technique: All horses underwent bursoscopy by the first or second author under general anaesthesia as described previously (Smith et al. 2007; Smith and Wright 2011).
Statistical analysis: Using endoscopic observations as the defining standard, sensitivity, specificity, positive and negative predictive values were calculated to evaluate the reliability of low field MRI for detection of intrathecal tears of the dorsal border of the DDFT, lesions of the fibrocartilage of the navicular bone and adhesions within the navicular bursa.

Results

39 horses had 51 navicular bursae examined with both standing low field MRI and endoscopy.

Low field MRI

DDFT tendonitis was the most common principal lesion (44 feet, 86%). Lesions affected the medial (n = 30) and lateral (n = 28) lobes with similar frequency; lesion types seen at the level of or proximal to the navicular bone included dorsal border fibrillation (n = 15), parasagittal splits (n = 4) (Figure 1a), dorsal border tears (n = 18) (Figure 2a) and core lesions (n = 8). In addition, the same lesion at different levels appeared as both a core lesion and fibrillation (n = 4), split and tear (n = 3), core, split and tear (n = 1), core and tear (n = 2), or core and split (n = 2). 3 lesions continued distal to the navicular bone as parasagittal splits and 1 as a core lesion. Parasagittal splits (n = 3) and core lesions (n = 1) were also seen as sole lesions distal to the navicular bone. Lesions distal to the navicular bone affected the medial (n = 2) and lateral (n = 6) lobes of the DDFT.

Further abnormalities of the podotrochlear apparatus reported at MRI included, in 32 (63%) feet injuries of the navicular bone, manifested as altered signal intensity in T1,
T2* or STIR pulse sequences (n = 30) (Figure 1b), and / or erosions of the flexor surface of the bone (n = 4). Evidence of desmitis of the distal sesamoidean impar ligament (DSIL) was identified in 7 (14%), and desmitis of the collateral sesamoidean ligaments in 10 (20%) limbs. Adhesions within the bursa were reported in 11 feet (22%) from the DDFT to the CSL (n = 5), flexor surface of the navicular bone (n = 5), DSIL (n = 2), and contra-axial lobe of the DDFT (n = 1). In one bursa a mass was seen in the proximal recess.

In 28 limbs (55%) there were concurrent DDFT and navicular bone injuries. Twenty (39%) feet were reported to have multiple i.e. \( \geq 3 \) significant abnormalities of the intra thecal components of the podotrochlear apparatus.

Other observations: Common non-specific findings included distension of the DIP joint and navicular bursa, with or without synovial proliferation; minor remodelling of the proximal, distal and sloping (wings) margins of the navicular bone; thickening of the palmar (flexor) cortex of the navicular bone; loss of definition between the DDFT and surface of the navicular bone, CSL or DSIL. Less common non-specific findings included minor remodelling at the origin or insertion of the DIP joint collateral ligaments, and at the insertion of the DSIL and DDFT.

Endoscopic findings

Lesions of the DDFT were seen in 40/51 (78%) bursae. DDFT splits were seen in the lateral lobe in 2 bursae (1 proximal and 1 distal to the navicular bone). Dorsal border fibrillation was seen in the proximal recess of 9 bursae, affecting the medial (n = 2), lateral (n = 6) or both (n = 1) lobes of the DDFT. Tears of the DDFT were seen in the proximal recess of 33 bursa affecting the lateral (n = 11), medial (n = 11) or both
lobes (n = 11). Tears had a linear orientation in all cases and extended from the proximal bursa variable distances distally. In all cases, torn tendinous tissue extruded from the tears into the bursal lumen (Figure 1c, 2b). In 1 case the dorsal border of the lateral lobe of the DDFT was friable and with gentle probing ruptured to expose torn tendon tissue. Large strands of torn tendon were found adhered (n = 15; 29%) to the opposing surface of the navicular bone (n = 2), CSLs (n = 7), the contralateral lobe of the DDFT (n = 3) or the T ligament (n = 3). Cartilaginous defects were seen in 4 bursae (8%) (Figure 1d) of which 2 were partial thickness and 2 were full thickness.

The sensitivity, specificity, positive and negative predictive values of low field MRI for each lesion identified endoscopically are displayed in Table 1.

Discussion

The results of this study suggest that standing low field MRI provides reasonable predictive values for detection of dorsal border disruption of the DDFT, although results of such should be interpreted with the understanding that currently both false positive and false negative results will be obtained. Non-the-less it is able to detect lesions that are deeper than surface level, and which may be prognostically important before considering endoscopy.

Cases in the current series demonstrate that endoscopy identifies with greater accuracy, and provides superior morphological characterisation of, lesions within the navicular bursa, compared to low field MRI. Endoscopy therefore has a valuable role both in diagnosis and in determination of prognosis. As such it should be considered in all cases when lameness has been localised to the navicular bursa, particularly as it
also offers concurrent therapeutic potential (Smith et al. 2007; Smith and Wright 2011).

The reliability of standing low field MRI for detection of fibrocartilage lesions was poor, and this is consistent with previous work in cadaver limbs with high field MRI (Schramme et al. 2009). The prevalence of lesions, particularly of the fibrocartilage, was low, which contributes to the rates of false positives seen (Bayes and Price 1763). Numbers are too small to draw conclusions regarding the causes of misinterpretation. However, our findings support the conclusions of Schramme et al. (2009) that MRI cannot reliably be used for detection of fibrocartilage lesions. These authors demonstrated improved reliability for detection of fibrocartilage lesions using saline magnetic resonance bursography.

Similarly low predictive values were seen for detection of adhesions. Saline magnetic resonance bursography may also have application for improving the rate of detection of adhesions and surface lesions of the DDFT. As yet, this is speculative. It could be given further consideration in clinical practice but is clearly invasive. Murray et al. (2006) demonstrated good correlation in 51 cadaver limbs between high field MRI and histology and macroscopic detection of adhesion formation, for lesions of the DDFT and navicular bursa. In a study comparing low and high field MRI in 10 cadaver limbs (Murray et al. 2009), both systems detected the majority of lesions, but high field MRI had greater accuracy and some details of lesions were clearer. In current clinical practice in the UK, low field MRI is used much more frequently than high field MRI for investigation of lameness localising to the feet. Clinicians should to be aware of potential differences in information that can be obtained from high vs. low field MRI, and suitable consideration given when choosing which modality to use. High field MRI has not been endoscopically validated, but the results of this
study suggest that a degree of caution should currently be exercised when surface
lesions in the navicular bursa are diagnosed by standing low field MRI.

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and interpreted the MRIs.

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magnetic resonance imaging represent histologic findings in the equine


TABLE 1: Sensitivity, specificity and predictive values for detecting lesions within the navicular bursa with standing low-field MRI

<table>
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<tr>
<th></th>
<th>Dorsal border DDFT injury</th>
<th>Dorsal border DDFT split or tear</th>
<th>Fibrocartilage injury</th>
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<td>NPV (%)</td>
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<td>88</td>
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<td>69</td>
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PPV = positive predictive value; NPV = negative predictive value
Figure 1: Transverse T1W 3D HR images a) demonstrating an oblique parasagittal split within the medial lobe of the DDFT (arrow), which is enlarged. The dorsal surface of the medial lobe is disrupted. b) A large area of decreased signal within the navicular bone (arrows) was identified with MRI. Corresponding endoscopic images c) linear tear of the medial lobe of the DDFT, with torn fibres extruded into the bursal lumen, and adhered to the opposing surface of the bursa. d) a small circular depression is present in the sagittal ridge of the navicular bone. N = navicular bone; T = disrupted DDFT; D = depression (Further endoscopic images and MRI slices of the navicular bursa are published online).

Figure 2: a) Transverse T1W 3D HR image demonstrating dorsal border disruption and hyperintense signal of the medial lobe of the DDFT (arrow). b) Corresponding endoscopic image demonstrating a linear tear in the medial lobe of the DDFT, with recoiled fibres in the proximal bursa. A fibrous adhesion crosses the bursa proximal to the torn fibres. A = adhesion (Further endoscopic images and MRI slices of the navicular bursa are published online).

**Supplementary information**

Figure 1: e-p) Transverse T1W 3D HR images of the navicular bursa. Lesions identified were an erosive lesion in the sagittal ridge of the navicular bone, large area of increased signal in the spongiosa of the navicular bone, an oblique parasagittal split within the medial lobe of the DDFT, and mild swelling of the
dorsal border of the medial lobe proximal to the navicular bone. q) Endoscopic view of the tear in the medial lobe of the DDFT, extending distally opposite the navicular bone in a linear orientation.

Figure 2: c-g) Transverse T1W 3D HR images of the navicular bursa. Lesions identified were enlargement and dorsal border disruption of the medial lobe of the DDFT, and fibrillation of the lateral lobe of the DDFT, in the proximal bursa, which was markedly distended. h) Endoscopic view of a linear tear in the lateral lobe of the DDFT, with adjacent fibrous adhesions. i) The tear in the medial lobe extends distally opposite the palmar surface of the navicular bone, and further torn fibres can be seen extruded.