Case Report

Severe bilateral physitis with instability and Salter-Harris *type 1* fractures in two foals

M. A. Jackson*, M. Kummer, J. A. Auer, P. Ossent†, M. Paar‡ and A. E. Fürst

Equine Department and †Institute of Veterinary Pathology, Vetsuisse-Faculty, University of Zurich, Zurich, Switzerland; and ‡Clinic for Horses, Sottrum, Germany.

Keywords: horse; foal; physitis; Salter-Harris *type 1* fracture; metatarsus; computed tomography

Summary

This Case Report describes the clinical, radiographic, computed tomographic and *post mortem* findings in 2 foals with severe bilateral physitis of the distal third metatarsal bones (MtIII). The foals were admitted because of bilateral hindlimb lameness of several weeks' duration. Initial examination revealed that the physeal regions of the distal MtIII were enlarged, warmer than normal and painful on palpation. *Case 1* was treated conservatively and discharged but was readmitted 8 weeks later because of clinical deterioration. In both foals, radiographs showed plantar dislocation of both epiphyses through the physis and a Salter-Harris *type 1* fracture. The foals were subjected to euthanasia and a *post mortem* examination.

Introduction

Physitis is a disturbance of enchondral ossification in the growing horse characterised by enlargement of the physeal region of the long bones and often accompanied by heat, pain and swelling (Baxter and Turner 2002). 'Physeal dysplasia' is probably a more appropriate term because initially there is no active inflammation (Firth 1990). There are few published studies on physitis. Some authors consider it to be part of the developmental orthopaedic disease (DOD) complex, characterised by alterations in bone growth or development (Baxter and Turner 2002). Although the exact cause of physeal dysplasia has not been identified, the aetiology is most likely multifactorial and includes rapid growth, trauma to the ossification centres, genetic predisposition and nutritional imbalances. There is also speculation that physitis is a manifestation of osteochondrosis (White 1980). The disease manifests mainly between 4–8 months and one and 2 years of age (Baxter and Turner 2002). The distal third metacarpal/metatarsal bones (McIII/MtIII) and the distal radius are most commonly affected clinically, followed by the distal tibia. Usually both front or both hindlimbs and, occasionally, all 4 limbs are affected. Clinical signs include lameness, stiffness, swelling and pain on deep palpation. The most common radiographic finding is an irregular and widened physis (Firth 1990). Histologically, the disease is characterised by arrested ossification, an increase in height of the physeal cartilage and fissures between the physeal cartilage and adjacent metaphyseal region. These findings may indicate impaired metaphyseal blood supply (Firth 1990). Treatment of physitis consists of stall rest, a nonsteroidal anti-inflammatory drug (NSAID) and reduction of the energy content of the diet, adequate mineral supplementation in the feed including a correct balance of calcium and phosphorous. The prognosis is usually good, although cases of angular limb deformities following premature closure of the affected growth plates have been reported (Firth 1990; Ellis 2003).

The present report describes the clinical, radiographic, computed tomographic, *post mortem* and histological findings in 2 foals with severe bilateral physitis in the distal MtIII with secondary instability and Salter-Harris *type 1* fractures.

Case details

Case 1

History, clinical findings and treatment

A 3-month-old Warmblood filly was admitted to our clinic because of bilateral hindlimb lameness of one month duration and difficulty in rising. The lameness was worse early in the morning and after periods of rest. There was no history of trauma. The clinical examination revealed moderate bilateral hindlimb lameness (*grade 2/5*, American Association of Equine Practitioners lameness scale, *grades 0–5*) and stiffness in the hindlimbs. Lameness was worse when the foal was turned in a circle on a hard

*Corresponding author email: mjackson@vetclinics.uzh.ch

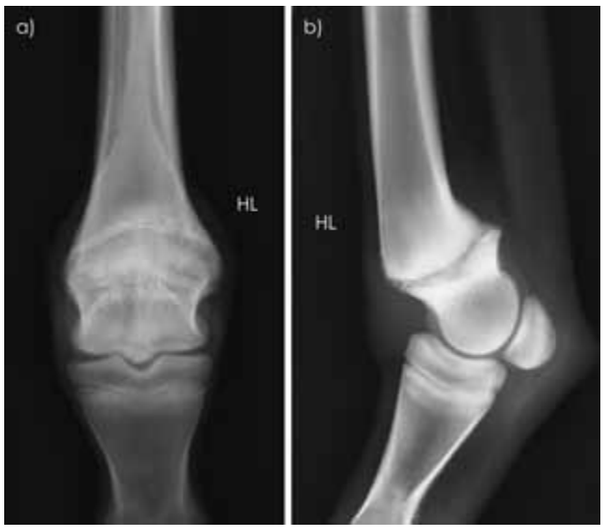

Fig 1: a) Dorsoplantar view of the left hind fetlock of* Case 1 *at initial examination, with irregular growth plate thickness and sclerosis of the surrounding bone. b) Lateral view of the left hind fetlock of* Case 1 *at initial examination. Mild caudal subluxation of the epiphysis along the growth plate is evident.

surface. Both physeal regions of the distal MtIII were enlarged and warmer than normal and deep palpation was painful. The forelimbs were normal. The rectal temperature and results of a complete blood cell count and biochemical profile were within normal limits. Radiographs showed an irregular growth plate of the distal MtIII in both hind legs and sclerosis of the surrounding bone (**Fig 1a**), which confirmed the diagnosis of physitis. On lateral views, mild plantar dislocation of both epiphyses was evident (**Fig 1b**). Radiographs of the distal McIII and the distal radius of both forelimbs showed normal growth plates. The foal was treated with an NSAID (phenylbutazone, 2.2 mg/kg bwt *per os* q. 12 h for one week and q. 24 h for 2 weeks), mineral supplementation and stall rest for 4 weeks, followed by turnout in a small paddock for another 4 weeks. The dam's grain intake was reduced.

Follow-up

The foal was discharged and the lameness improved initially, but 2 months later, she was referred to our clinic again because of sudden onset of severe lameness. Both hind pasterns were upright and the physeal regions of both distal MtIIIs were larger than at the time of discharge 2 months previously (**Fig 2a**). The foal was severely lame in both hindlimbs at walk. The growth plates were warmer than normal and painful on palpation and flexion of both fetlock joints also elicited pain. During manipulation of the fetlocks, there was no mediolateral or dorsoplantar instability. Radiographs of both hind fetlock joints showed extensive new bone formation and deformation of both distal MtIIIs with large radiolucent areas in the

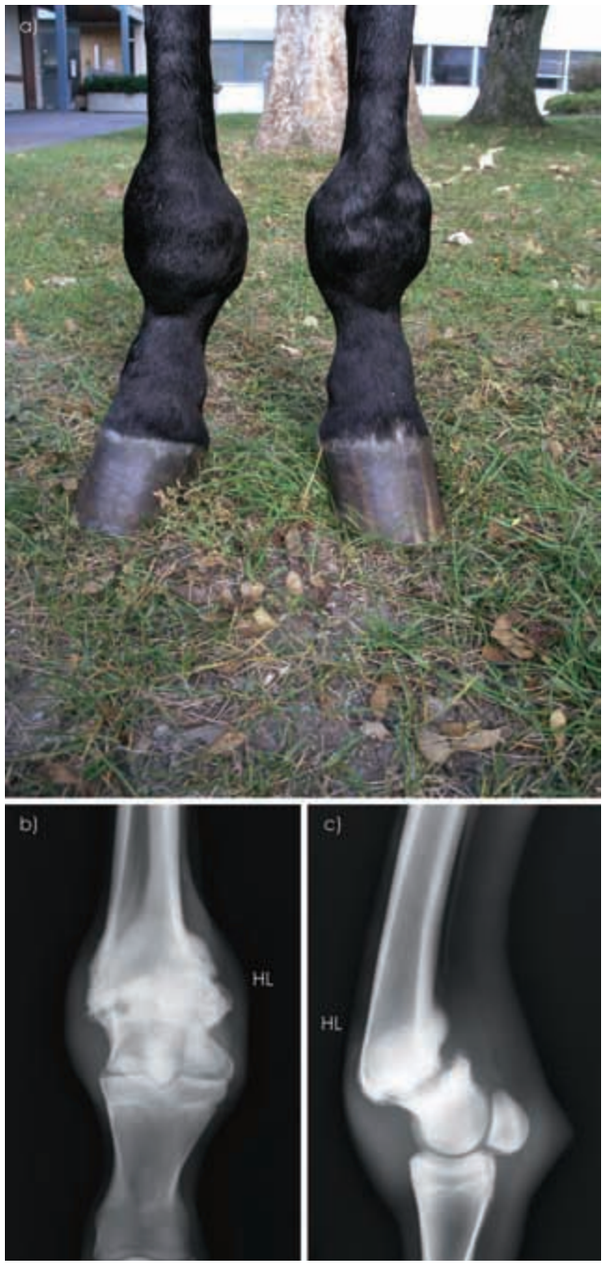

Fig 2: a) Enlargement of the distal metaphysis of both MtIII of* Case 1 *at 5 months of age. b) Dorsoplantar view of the left hind fetlock of* Case 1 *at 5 months of age. Severe bone proliferation and extensive areas of radiolucency in the growth plates are present. c) Lateral view of the left hind fetlock of* Case 1 *at 5 months of age. A caudal subluxation of the epiphysis with deformation of the distal MtIII is evident.

growth plates (**Fig 2b**). Furthermore, both epiphyses were displaced plantarly through the physis (**Fig 2c**). Computed tomography (CT) revealed a Salter-Harris *type 1* fracture of both distal metatarsal bones (**Figs 3a** and **b**). The foal was subjected to euthanasia because of a poor prognosis.

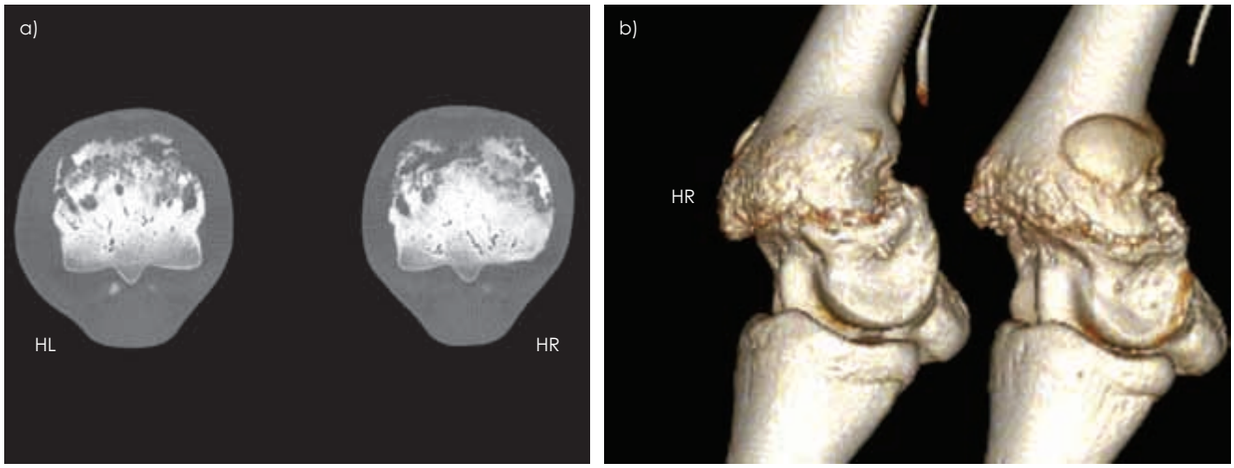

Fig 3: a) Computed tomography horizontal slice along the distal MtIII growth plate of Case 1 at 5 months. Note the extensive radiolucent areas in the growth plates of MtIII and the irregular bone proliferation dorsally. b) Computed tomography 3D reconstruction of both distal MtIII and proximal phalanx of Case 1 at 5 months of age. The caudal subluxation of the epiphyses along the growth plate as well as the massive periosteal proliferation around the growth plates are evident.

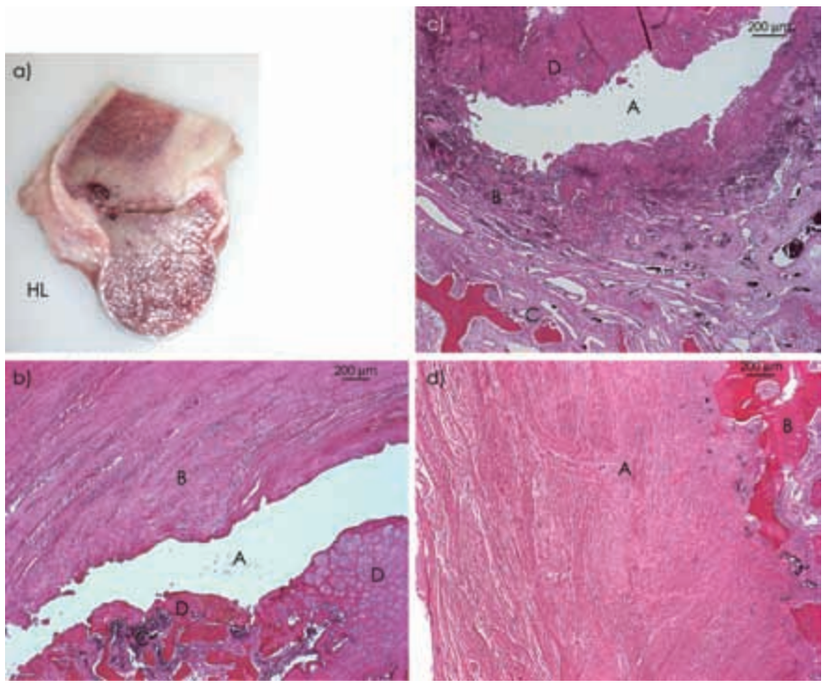

Fig 4: a) Sagittal histological section of the distal MtIII of Case 1, with plantar displacement of the epiphysis, separation between metaphysis and epiphysis along the growth plate with remnants of haemorrhage and accumulation of connective tissue. b) Histological section along the line of the fractured growth plate. Fracture gap (A), massive proliferation of fibrous tissue (B), areas of tissue necrosis (C) disseminated small cartilage islands (D). c) Histological section along the fracture line along the growth plate. Fracture gap (A), necrotic detritus (B), osteoclastic resorption of bone (C) and fibrous tissue bordering the fracture gap (D). d) The periosteum at the level of the physis (B) is massively thickened by fibrous connective tissue (A).

Post mortem *and histopathological examination*

Both distal MtIIIs were collected at necropsy and cut longitudinally in the sagittal plane. Tissue samples were fixed in neutral buffered formalin, demineralised in 5% nitric acid for 2–3 weeks, sectioned and stained with haematoxylin and eosin. As expected from the CT images, the distal epiphyses were separated from the metaphyses and displaced plantarly (**Fig 4a**). Examination of histological sections showed islands of disorientated cartilage along the fracture line (**Fig 4b**). There were remnants of necrotic bone, osteoclastic resorption,

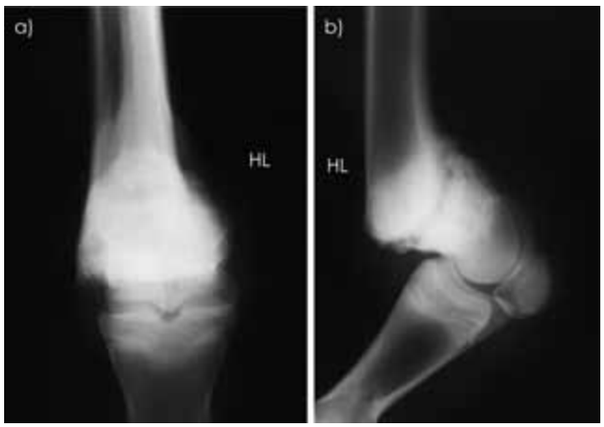

Fig 5: a) Dorsoplantar view of the left hind fetlock of Case 2 at initial examination, showing massive new bone proliferation at the level of the physis, massive periosteal proliferation and sclerosis of the surrounding bone. b) Lateral view of the left hind fetlock of Case 2 at initial examination, with marked caudal subluxation of the epiphysis along the growth plate.

abundant dense fibrous tissue and loose connective tissue proliferation (**Fig 4c**). Typical growth plate components and inflammatory cells were not seen along the fracture line. In the affected areas, the thickness of the periosteum was increased by fibrous connective tissue (**Fig 4d**).

Case 2

A 4-month-old Friesian filly was presented to the clinic because of severe bilateral hindlimb lameness of 5 weeks duration and intermittent fever. The owners reported that the filly had had difficulty rising since one month of age. The foal had been treated by the primary care veterinarian with antibiotics (trimethoprim/sulphamethoxazole, 30 mg/kg bwt *per os* q. 12 h) and an NSAID (phenylbutazone, 2 mg/kg bwt *per os* q. 12 h) for 2 weeks without improvement in the lameness. On clinical examination, the foal was severely lame in both hindlimbs and both physeal regions of the distal MtIII were enlarged, warmer than normal and painful on palpation. Fetlock manipulation elicited pain on flexion but no instability was detected. There was no fever and blood analysis revealed no abnormalities. Radiographs showed massive bone proliferation at the level of the growth plates with sclerosis of the surrounding bone and periosteal proliferation (**Fig 5a**). On lateral radiographic views, plantar displacement of both epiphyses through the growth plate was evident (**Fig 5b**). The owner elected euthanasia of the foal because of a poor prognosis.

Discussion

The growth plate separates the epiphysis from the metaphysis and provides rapid increase in bone length for a limited period of time by means of enchondral ossification (Dupuis *et al*. 1997). Cessation of metatarsal growth occurs by approximately 100 days of age, although the physis remains open and potentially active until foals are about 10 months old (Fretz *et al*. 1983). Physitis and physeal dysplasia are terms used to describe a disturbance of enchondral ossification in the growth plates (Baxter and Turner 2002). The incidence of physitis involving the distal McIII and MtIII in foals is highest between 4 and 8 months of age (Coffman 1973), which coincides with the beginning of physeal closure. Possible causes of physitis include mechanical stress of the physis attributable to excessive bodyweight or strenuous exercise and conformational abnormalities (Bramlage 1993). Both fillies described in this report underwent rapid growth; however, several studies have shown that overfeeding and rapid growth alone are not sufficient to cause physitis (Williams *et al*. 1982). A genetic predisposition and several other factors are thought to sensitise the physis to trauma or compression (Williams *et al*. 1982). Another possible cause of physitis is structurally deficient bone, which results in cartilage retention and poor trabecular bone formation and eventually inflammation of the physis when the deficient bone collapses (Bramlage 1993). In our 2 cases, it is conceivable that instability of an abnormal growth plate led to a shear fracture through the physeal cartilage (Salter-Harris *type 1* fracture). In *Case 1*, histological examination revealed massive new bone formation and fibrous tissue in the space between the separated metaphysis and epiphysis. This was most likely the result of continuous shear forces that acted in the physis secondary to cranial displacement of the metaphysis, in combination with resorption and further deterioration of the abnormal growth plates.

A similar condition that involves separation of the proximal femoral epiphysis has been reported in dogs, swine and humans. This condition is not associated with trauma and in adolescent human beings is recognised as a distinct clinical syndrome referred to as slipped capital femoral epiphysis (SCFE; Moores *et al*. 2004). This syndrome occurs most commonly during periods of rapid growth, when the stability of the physis is relatively low because of a mismatch between a high volume of hypertrophic cells and a comparatively low volume of interstitial matrix (Moores *et al*. 2004). It is bilateral in 20–30% of cases (Dupuis *et al*. 1997). Slipped capital femoral epiphysis may be classified as preslip, acute or chronic and stable or unstable. The displacement of the epiphysis is gradual but the epiphysis does not become separated from the femoral neck. This condition is considered stable when the capital epiphysis remains firmly attached to the femoral neck (Moores *et al*. 2004). Both foals described in this report had a gradual displacement of the epiphyses with increase in lameness and no history of trauma. Furthermore, although the occurrence of microinstability could not be assessed clinically, no instability was detected on gross manipulation of the distal metatarsal bones and fetlock joints. Therefore, the Salter-Harris *type 1* fractures diagnosed in the 2 foals appear to resemble chronic bilaterally stable SCFE.

Epiphysiolysis, which is considered a traumatic lesion associated with osteochondrosis in the growth cartilage, is a similar condition that occurs in swine (Grondalen 1974). Both foals had undergone rapid growth and it is conceivable that osteochondrosis may have played a role in their condition, especially in view of the bilateral occurrence of physitis. However, the growth plates of *Case 1* were examined histologically at an advanced stage of the disease and it was not possible to determine whether a pre-existing cartilaginous defect had been present. Therefore, the primary cause of epiphyseal displacement could not be determined. Histological examination at an earlier stage of the disease would be more revealing, although this is not feasible in a clinical situation.

In adolescent people with SCFE, the outcome of conservative treatment is good in most cases (Ordeberg *et al*. 1984), although continued displacement is seen in some patients. Therefore, surgical treatment involving stable fixation of the epiphysis and femoral neck using pins and/or screws is preferred (Loder *et al*. 2000). In the present report, in *Case 1* we used conservative treatment with an NSAID and mineral supplementation. In retrospect, early weaning of the foal and more severe restriction of exercise should have been instituted in this foal. Although the onset of displacement of the distal epiphyses had been diagnosed in *Case 1*, surgical fixation was not considered. The radiographic and CT examination 2 months later showed worsening of the displacement, lack of evidence of bone healing and separation along the physis, which was confirmed at *post mortem* examination. It is questionable whether stabilisation by internal fixation in the early stage of the disease, which is done in man with SCFE, should be considered in foals. In our opinion, a DCS plate or 2 transphyseal screws and a cast could stabilise the physis and prevent further displacement and fracturing and possibly prevent the development of angular limb deformity. Because growth at this levels ceases at approximately 100 days of age (Fretz *et al*. 1983), the implant would not be expected to interfere with longitudinal bone growth. However, surgical intervention should be carried out as soon as the dislocation is detected. Surgical stabilisation at a later stage of the disease is more likely to lead to implant failure or failure to correct the skeletal deformity.

Conclusions

This report describes gradual displacement of the distal epiphyses of MtIII in 2 foals. We propose that severe physitis led to instability of the growth plate and subsequently to a pathological fracture. The clinical manifestation of physitis in our 2 cases is similar to that of slipped capital femoral epiphysis in people, characterised by chronic displacement of the epiphysis without actual separation (Morscher 1968). Computed tomography yielded a definitive diagnosis of a Salter-Harris *type 1* fracture of the distal metatarsal bones. To ensure an early diagnosis of such fractures, foals with physitis that do not respond to conservative treatment should be monitored closely using serial radiographic examination. When early signs of plantar dislocation of the distal epiphysis are detected, surgical stabilisation of the bone using internal fixation should be considered to prevent further displacement and fracturing.

Acknowledgements

The authors would like to thank Dr Peter Moor for the referral of *Case 1*.

A special dedication from all the authors to Dr Pete Ossent, co-author of his last scientific work. Pete sadly passed away some months ago.

References

Baxter, G.M. and Turner, A.S. (2002) Diseases of bone and related structures. In: *Adams' Lameness in Horses*, 5th edn., Ed: T.S. Stashak, Lippincott Williams & Wilkins, Baltimore. pp 408-410.

Bramlage, L.R. (1993) Identification, examination, and treatment of physitis in the foal. *Proc. Am. Ass. equine Practnrs.* **39**, 57-62.

Coffman, J.R. (1973) Bone and muscle defect in foals. *Mod. Vet. Pract.* **54**, 53-54.

Dupuis, J., Breton, L. and Drolet, R. (1997) Bilateral epiphysiolysis of the femoral heads in two dogs. *J. Am. vet. med. Ass.* **210**, 1162-1165.

Ellis, D.R. (2003) Physitis. In: *Diagnosis and Management of Lameness in the Horse*, Eds: M.W. Ross and S.J. Dyson, W.B. Saunders, Philadelphia. pp 554-556.

Firth, E.C. (1990) Physitis. In: *Current Practice of Equine Surgery*, Eds: N.A. White and J.N. Moore, B Lippincott Company, Philadelphia. pp 534-536.

Fretz, P.B., Cymbaluk, N.F. and Pharr, J.W. (1983) Quantitative analysis of long-bone growth in the horse. *Am. J. vet. Res.* **45**, 1602-1609.

Grondalen, T. (1974) Osteochondrosis and arthrosis in pigs. Incidence in animals up to 120 kg live weight. *Acta vet. Scand.* **15**, 1-25.

Loder, R.T., Aronssen, D.D., Dobbs, M.B. and Weinstein, S.L. (2000) Slipped capital femoral epiphysis. *J. Bone Joint. Surg.* **82-A**, 1170-1188.

Moores, A.P., Owen, M.R., Fews, D., Coe, R.J., Brown, P.J. and Butterworth, S.J. (2004) Slipped capital femoral epiphysis in dogs. *J. small anim. Pract.* **45**, 602-608.

Morscher, E. (1968) Strength and morphology of growth cartilage under hormonal influence of puberty. *Reconstr. Surg. Traumatol.* **10**, 103-104.

Ordeberg, G., Hansson, L.I. and Sandstrom, S. (1984) Slipped capital femoral epiphysis in southern Sweden. Long term result with no treatment symptomatic primary treatment. *Clin. Orthop. Relat. Res.* **191**, 95-104.

White, S.L. (1980) Clinical manifestations and pathophysiology of osteochondrosis. *Proc. Am. Ass. equine Practnrs.* **26**, 69-80.

Williams, J.A., Collier, M.A. and Ross, M.W. (1982) Physitis in the horse. *Mod. Vet. Pract.* **63**, 407-413.