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Sacroiliac lesions as a cause of chronic poor performance in competitive horses

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Summary

The histories and clinical signs in 11 horses with longstanding poor performance attributed to chronic sacroiliac damage are described. The main clinical feature was a lack of impulsion from one or both hindlimbs causing a restriction in the hind gait or lowgrade lameness. A temporary improvement was often achieved using anti-inflammatory medication, but eventually all the horses were killed because of unsatisfactory progress at exercise. Post mortem examination revealed that changes were confined to the sacroiliac joints. The macroscopic and histological findings varied considerably and in only two cases could the changes be classified histologically as arthrosis. In the other nine horses there was increased joint surface area or irregular outline associated with extensions of the joint on the caudomedial aspect. These changes were interpreted as indicating a chronic instability of the joint leading to restriction of hindlimb impulsion. The underlying cause of the problem was not ascertained but the significance and possible pathogenesis of the lesions are discussed.

Introduction

IN recent years considerable importance has been attributed to injury of the sacroiliac joint as a cause of lowered performance in horses (Adams 1969; Rooney, Delaney and Mayo 1969; Rooney 1977, 1981; Koch 1980; Jeffcott 1982). However, the exact nature of the changes involved are not yet clear. There have been a few reports of post mortem changes (Rooney *et al* 1969; Jeffcott 1982) but these can by no means be described as definitive. There are several reasons for the lack of basic knowledge on the sacroiliac joint of the horse: (a) Clinical diagnosis of sacroiliac damage is imprecise because of its inaccessible anatomical location; (b) the biomechanics of the joint are not fully understood; (c) the clinical signs are rarely severe enough to warrant euthanasia, even though the incidence of the condition may be quite high, and post mortem examination is therefore seldom possible; (d) the joint is difficult to examine effectively at autopsy; (e) until recently, little has been known about the normal macroscopic and histological appearance of the joint (Dalin and Jeffcott 1985a, b; Ekman, Dalin, Olsson and Jeffcott 1985).

The purpose of this study is to report on the clinical findings of 11 competitive horses with suspected chronic sacroiliac damage and to correlate the clinical signs with the post mortem changes in the joint.

Materials and methods

A total of 11 horses of varying breed, age and size with a history of poor performance at exercise, usually with low grade hindlimb lameness, were investigated (Table 1). A full history was obtained for each case and clinical examination carried out with particular reference to examination of the back (Jeffcott 1981). Elimination of thoracolumbar, stifle, hock and distal limb problems was performed by appropriate flexion tests, local anaesthetic nerve blocks and radiography. Radiographic examination of the thoracolumbar, lumbosacral and pelvic areas was performed on six cases (Jeffcott 1982) and linear tomography of the lumbosacral region on four cases (Jeffcott 1983a, b).

Post mortem examination was carried out as described by Dalin and Jeffcott (1985a) including morphometric measurements of the sacral and iliac joint surfaces (Dalin and Jeffcott 1985b). Histological examination was done in seven horses and in six of these radiographic examination of slabs from the sacroiliac joints was performed.

Statistical analyses were carried out by standard methods and included the calculation of significance by Student's *t* test for paired observations.

Results

Clinical findings

The horses were predominantly geldings of middle age (five to 12 years old) and large body size (Table 1). They were all competitive animals; four horses were mainly used for general riding purposes but did compete in some showing and amateur equestrian competitions. Only two of the horses were less than 155 cm in height and these were both Standardbred harness racing horses; the others were Thoroughbreds or Warmblood types. No consistent history of trauma could be incriminated as being the inciting cause of lameness in any of the cases in this series.

There was always a prolonged history of poor performance at exercise. The estimated mean time for the duration of

TABLE 1: Breed, use, sex, body weight and height of 11 horses examined with sacroiliac damage

Case number	Breed	Use	Age (years)	Sex	Body weight (kg)	Height at withers (cm)
1	SWB	Hack	9	G	590	167
2	STB	Trotter	6	G	433	155
3	TB	Dressage	7	M	490	160
4	SWB	Hack/Dressage	6	G	600	166
5	Holst	Hack	5	G	550	164
6	STB	Trotter	7	M	–	152
7	SWB	Hack/Dressage	7	G	–	170
8	SWB	Hack	12	G	610	172
9	TB	Eventer	7	G	594	162
10	TB	Eventer	8	G	560	163
11	SWB	Hack	6	M	590	168

SWB Swedish Warmblood TB Thoroughbred G Gelding
STB Standardbred Holst Holsteiner M Mare

clinical signs was about 14 months (Table 2). Mild hindlimb lameness (ie, swinging type) was usually present and was frequently most noticeable at a slow trot. This was often associated with some stiffness of the hind action at the commencement of exercise. The lameness was often difficult to define precisely. It tended to be unilateral but from time to time the opposite hindlimb was also affected. The lameness was seen from behind as an unlevel action with restriction in hindlimb stride and poor impulsion from the quarter. There was commonly mild abduction of the affected limb during mid stride followed by bringing the leg underneath the body before landing (ie, plaiting). Some restriction in hock flexion was frequently suspected, often with dragging of the toe during the cranial phase of the stride.

The response to hock flexion was variable but in no case was there a dramatic and positive response. Radiographic examination of the hocks was carried out in nine cases and no signs of any changes resulting from bone spavin were demonstrated. When viewed from behind the majority of horses showed some asymmetry of the hindquarters with the tuber coxae and tuber sacrale lower on the side the animal was lame on. However, this was not consistent in all cases. Atrophy of the gluteal muscles was another frequent finding and this was usually more pronounced on the lame quarter. Often associated with this was a greater prominence of the tips of the caudal lumbar spinous processes and tuber sacrale, referred to colloquially as a 'jumper's bump'. Palpation of the distal lumbar spine and lumbosacral region was frequently resented, although none of the horses showed a marked pain response. There was usually some reduction in the normal range of spinal flexibility to dorsiflexion (ie, dipping), ventroflexion (ie, arching) or lateral flexion of the back. In most cases, no obvious difficulties turning in a tight circle or moving backwards were seen.

Rectal palpation of the skeletal structures in the pelvic canal was normal and no pain could be elicted from the iliopsoas muscles. Clinical pathological examination, including analysis of the haematological profile and the enzymes aspartate amino transferase and creatine kinase, did not indicate the presence of any active skeletal muscle damage.

The Standardbreds encountered problems when performing at speed, particularly on the turns where they tended to lose their action and go out of trot into transitional gait or gallop. **Case 2** was exercised on a treadmill (Fredricson *et al* 1983) and showed poor hindlimb impulsion and difficulty maintaining the trotting gait at speeds greater than 7 m/sec. A marked improvement was obtained up to speeds of 9.5 m/sec when the animal was maintained on therapeutic levels of phenylbutazone. There was a rapid deterioration in performance once medication was withdrawn.

The riding and jumping horses invariably performed at their worst at slower speeds, particularly during ground work and dressage movements. They showed stiffness of the back and

TABLE 2: Presence of certain clinical features and radiological findings in 11 horses with sacroiliac damage

	Case number										
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**
Duration of signs (months)	>24	>18	>10	>8	>10	>12	>5	>12	>20	>18	>12
Poor performance at exercise	+	+	+	+	+	+	+	+	+	+	+
Restricted hindlimb impulsion or stiffness	+	+	+	+	+	+	+	+	+	+	+
Stiff back at exercise	+	+	+	+	+	–	+	+	+	–	+
Plaiting with hind feet	–	–	+	–	–	+	–	–	+	+	+
Scuffing or dragging hind toes	–	+	+	+	–	+	+	+	+	+	–
Mild hindlimb lameness	RH	RH	BH	RH	LH	LH	LH	LH	RH	–	LH
Response to hock flexion	+	±	+	+	+	±	+	±	–	–	+
Asymmetry of hind quarters	+	+	+	–	+	+	+	+	±	–	+
Gluteal atrophy	+	+	+	–	+	+	+	+	–	+	+
Discomfort in lumbar or sacral spine	+	+	+	+	–	+	+	+	±	–	±
Changes noted on plain X-rays of sacroiliac region	–	–	–	–	ND	ND	ND	ND	+	+	ND
Changes noted at linear tomography	+	+	–	–	ND	ND	ND	ND	ND	ND	ND
Temporary response to phenylbutazone medication	+	+	ND	+	ND	ND	ND	+	+	+	ND

\+ Present ND Not done RH Right hindlimb
– Absent LH Left hindlimb
± Equivocal BH Both hindlimbs

TABLE 3: Summarised results of the macroscopic post mortem findings in the sacroiliac joints of 11 horses and the correlation with the history of hindlimb lameness

		Abnormal joint outline	Cartilage erosion	Sacral asym-metry	Presence of articular extension	Clinical signs of lameness
Case 1	Left	+ +	–		*Type 1*	–
	Right	+ +	–		*Type 1*	RH
Case 2	Left	+ +	–		*Type 2*	–
	Right	+	–		*Type 1*	RH
Case 3	Left	–	–		–	LH
	Right	–	+ +		*Type 4*	RH
Case 4	Left	–	–		*Type 3, 4*	–
	Right	–	–		–	RH
Case 5	Left	+	–		*Type 3*	LH
	Right	+	–		*Type 3*	–
Case 6	Left	+	NR	+	*Type 1*	LH
	Right	–	NR		–	–
Case 7	Left	+ +	NR	+	*Type 1*	LH
	Right	+ +	NR		*Type 1*	–
Case 8	Left	+	–	+	*Type 3, 4*	LH
	Right	+	–		–	–
Case 9	Left	+ +	–	+	*Type 2*	–
	Right	+ +	–		*Type 1*	RH
Case 10	Left	+ +	–	+	*Type 2*	–
	Right	+	–		*Type 1*	–
Case 11	Left	–	+	+	–	LH
	Right	–	–		–	–

\+ Present
– Absent
NR Not recorded
RH Right hindlimb
LH Left hindlimb

were often unable to work on one diagonal and seemed to lack impulsion from one or both hindlimbs. In six cases a temporary and partial improvement was obtained following medication with therapeutic doses of phenylbutazone (Table 2).

Radiographic examination of the thoracolumbar spine, pelvic and lumbosacral regions was performed in six horses. No lesions of clinical significance were found in the thoracolumbar spine or pelvis. Some changes were detected in the sacroiliac region of **Cases 9** and **10** but not in **Cases 1** to **4.** These findings were fairly non-specific and consisted of apparent increase of the normal joint space with irregular outline and apparent enlargement at the caudal edge of the wing of sacrum. Linear tomography was performed in **Cases 1** to **4** from which some enlargement of the caudal aspect of the sacroiliac joint was suspected (Table 2).

Poor performance and abnormal hindlimb action were consistent finding but did not appear to be progressive. There were times when a temporary improvement was seen in some horses with or without anti-inflammatory medication, but all the animals were ultimately euthanased at their owners' request because of consistently poor performance.

Macroscopic findings

Changes were confined to the sacroiliac region (Table 3). On dissection no laxity of the joints could be demonstrated. The ventral sacroiliac ligaments showed no detectable gross changes, but asymmetry of the ilium or wings of sacrum was found in six cases (Figs 1 and 2).

The sacroiliac joint surfaces showed evidence of articular

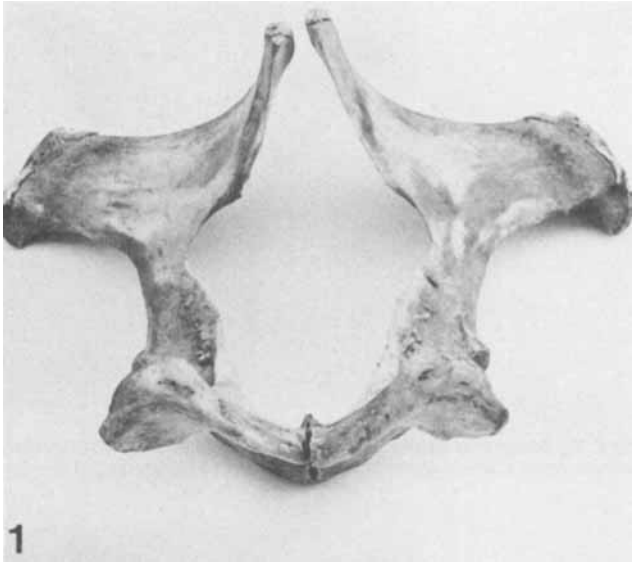

Fig 1. Macerated pelvis from a seven-year-old Standardbred mare **(Case 6)** *showing obvious asymmetry between the two wings of ilium*

cartilage degeneration but the extent was essentially similar to that seen in normal horses (Dalin and Jeffcott 1985a). In two animals **(Cases 3** and **11)** there were erosive lesions of the articular cartilage which affected both the sacral and iliac surfaces (Fig 3). There was a high incidence of abnormal joint outline with extension of the joint surfaces and peri-articular spur formation was seen in three cases (Figs 4 and 5). The extensions of the joints could best be described as follows.

Type 1 was seen as a prominent articular extension at the caudal edge of the wing of sacrum apparently covered by articular hyaline cartilage and surrounded by joint capsule. This triangular-shaped sacral structure fitted into a depression (ie, notch) in the opposing iliac surface which was covered by fibrocartilage (Figs 4 and 6).

Type 2 had a much more diffuse appearance involving extension of the sacral joint surface over a large area of the caudal edge of the wing of sacrum. This, too, was covered by hyaline cartilage and fitted into a large notch on the iliac surface (Figs 4 and 7).

Type 3 showed a large eminence on the medial aspect of the sacrum covered by articular cartilage, projecting dorsally into a notch in the iliac surface (Figs 4, 5 and 8). This gave the appearance of an unusual and pronounced concavity of the sacral surface of the joint.

Type 4 were small periarticular spurs close to the joint capsule and involving the insertion of the sacroiliac ligament (Figs 4 and 11).

Morphometric analyses

The results of the measurements of the surface area, length, width and width/length ratio of the sacroiliac joint surfaces are shown in Table 4. From comparisons with results obtained from a series of 41 normal horses (Dalin and Jeffcott 1985b) it was found that the sacral articular areas in the *Type 1* and *2* cases (ie, **Cases 1, 2, 9** and **10)** were significantly larger than expected (Table 5). In one case, the area was more than double the normal size. From the measurements of length and width

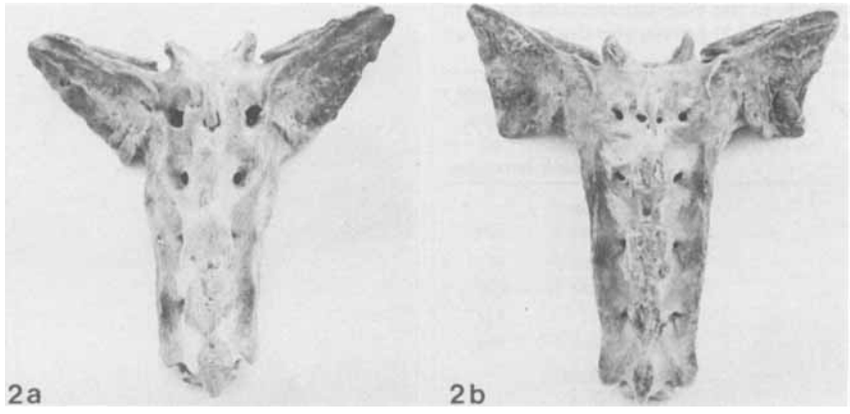

Fig 2. (a) Macerated sacrum from a seven-year-old Standardbred mare **(Case 6)** *showing asymmetry of the wings of the sacrum. (b) Macerated sacrum from a seven-year-old Swedish Warmblood gelding* **(Case 7)** *showing* Type 1 *extensions at the caudal edge of both sacral wings*

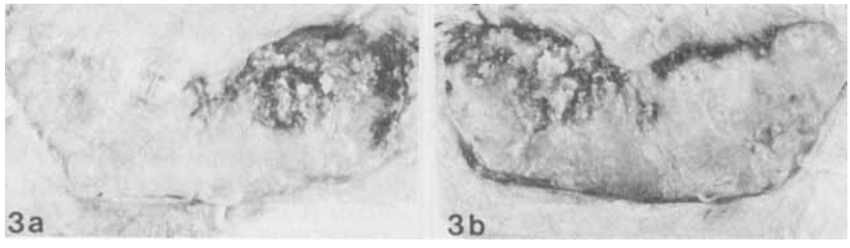

Fig 3. Right sacroiliac joint surfaces from a seven-year-old Thoroughbred mare **(Case 3)** *showing extensive erosion of both sacral (a) and iliac (b) articular surfaces*

of the joint surfaces it was seen that enlargement of the joint surface was caused primarily by a widening of the joint surfaces. The horses with erosions and *Types 3* and *4* changes were all within the normal range of joint surface measurements.

Radiography of slabs

In two horses **(Cases 3** and **11)** the subchondral bone of ilium and sacrum had a very irregular surface and cyst-like formations of varying sizes were present. The *Types 1, 2* and *3* articular extensions **(Cases 1, 2, 4** and **5)** consisted of apparently normal spongy bone (Fig 5b).

Histological findings

The histological appearance of sacral and iliac articular cartilage and the subchondral bone was much the same as seen in normal horses of the same age (Ekman *et al* 1985).

There were marked changes in the subchondral bone in only two of the horses **(Cases 3** and **11).** These changes consisted of large cavities (so-called pseudocysts; Olsson, Reiland, Pettersson and Strömberg 1983) filled by granulation tissue (Fig 9) with areas of chondroid metamorphosis. Multiple areas of bone marrow fibrosis were also present. In addition there was severe degeneration of the articular cartilage as demonstrated by fraying, clefts and clusters of cells. In large areas the entire cartilage was lost (Fig 9).

The *Types 1, 2* and *3* articular extensions consisted of apparently normal bone covered with cartilage. When there were changes in this cartilage they did not differ from those seen in the preformed articular cartilage (Fig 10).

Mature osteophytes *(Type 4)* were found at the caudal or cranial edges of the sacral articular cartilage in three cases **(Cases 3, 4** and **8)** (Fig 11).

Discussion

The terminology concerning chronic sacroiliac disease in the horse has been confused because of the lack of thorough pathological studies. Terms used to describe the same clinical syndrome have included sacroiliac subluxation or dislocation (Adams 1969), sacroiliac strain or instability (Jeffcott 1980, 1982) and sacroiliac arthrosis (Rooney 1977; Jeffcott 1983b). The chronic sacroiliac lesions reported in the present study could possibly have arisen from a trauma producing asymmetry of the ilium and sacrum over a prolonged period. No macroscopic damage to the ventral sacroiliac ligaments was found as has been recorded in acute trauma to this joint (Rooney *et al* 1969). The present findings cannot be classified as subluxation or dislocation and the indication for manipulative treatment (Herrod-Taylor 1967) must therefore be seriously questioned. The condition can be partially or temporarily alleviated by anti-inflammatory medication but not with any lasting success.

The history and findings associated with chronic sacroiliac damage are characteristic, although diagnosis must still largely be made by elimination of all other causes of hindlimb lameness and back injury. Because effective local anaesthesia of this joint is impractical, linear tomography is the only available objective diagnostic aid (Jeffcott 1983a, b), and this is only useful for demonstrating the joint extensions *(Types 1* and *2).* Nevertheless, the present data should provide a sound basis for tentative diagnosis and prognosis. The incidence of chronic sacroiliac damage in competitive horses and racehorses is probably quite high (Rooney 1977; Dalin, Magnusson and

TABLE 4: Morphometric results of the sacroiliac joint surfaces of nine horses with sacroiliac damage

		Surface area (cm^2)	Surface length (cm)	Surface width (cm)	Width/ length
Case 1	LS	22.9	9.6	5.5	0.57
	LI	24.3	9.9	5.7	0.58
	RS	26.4	9.5	6.5	0.68
	RI	27.9	9.3	5.7	0.61
Case 2	LS	20.4	7.4	4.7	0.64
	LI	23.2	8.0	4.9	0.61
	RS	16.1	8.9	4.1	0.46
	RI	18.4	8.7	4.7	0.54
Case 3	LS	16.4	7.5	3.6	0.48
	LI	18.6	8.1	3.5	0.43
	RS	18.7	8.2	3.7	0.45
	RI	19.7	7.8	3.6	0.46
Case 4	LS	14.3	8.7	2.4	0.28
	LI	14.0	8.5	2.6	0.31
	RS	11.3	7.2	2.2	0.31
	RI	11.6	7.5	2.2	0.29
Case 5	LS	14.3	7.9	3.5	0.44
	LI	11.9	7.5	2.7	0.36
	RS	10.8	7.5	2.5	0.33
	RI	10.4	6.6	2.7	0.41
Case 8	LS	15.3	8.7	3.2	0.37
	LI	18.9	9.2	3.8	0.41
	RS	14.7	8.6	3.0	0.35
	RI	15.3	8.7	3.2	0.37
Case 9	LS	20.2	8.1	4.9	0.60
	LI	NR	NR	NR	NR
	RS	17.0	7.7	4.6	0.60
	RI	NR	NR	NR	NR
Case 10	LS	31.8	10.5	5.0	0.48
	LI	27.7	10.0	5.0	0.50
	RS	23.0	9.3	4.0	0.43
	RI	18.5	8.0	4.1	0.51
Case 11	LS	16.9	8.4	3.5	0.42
	LI	18.7	8.4	3.5	0.42
	RS	14.6	7.9	2.8	0.35
	RI	17.3	8.8	2.6	0.30

LS Left sacrum RS Right sacrum NR Not recorded
LI Left ilium RI Right ilium

TABLE 5: Cases with articular extensions *(Types 1* and *2)* with a comparison between expected values (Dalin and Jeffcott 1985b) and morphometric results of the sacral joint surfaces

Variable / case number	Expected size (Dalin and Jeffcott 1985b)	Measured size Most affected side	Measured size Least affected side
Area (cm^2)			
Case 1	16.1	26.4	22.9
Case 2	12.9	20.4	16.1
Case 9	16.2	20.2	17.0
Case 10	15.5	31.8	23.0
All cases, $\bar{x}$ (sd)	15.2 (1.55)	24.7* (5.54)	19.8* (3.71)
Surface length (cm)			
Case 1	8.3	9.5	9.6
Case 2	7.4	7.4	8.9
Case 9	8.4	8.1	7.7
Case 10	8.2	10.5	9.3
All cases, $\bar{x}$ (sd)	8.1 (0.46)	8.9^{ns} (1.39)	8.9^{ns} (0.83)
Surface width (cm)			
Case 1	3.4	6.5	5.5
Case 2	3.1	4.7	4.1
Case 9	3.4	4.9	4.6
Case 10	3.4	5.0	4.0
All cases, $\bar{x}$ (sd)	3.3 (0.15)	5.3** (0.83)	4.6* (0.69)
Width/length ratio			
Case 1	0.41	0.68	0.57
Case 2	0.42	0.64	0.46
Case 9	0.40	0.60	0.60
Case 10	0.41	0.48	0.43
All cases, $\bar{x}$ (sd)	0.41 (0.008)	0.60* (0.086)	0.52* (0.083)

ns No significant difference
* $P<0.05$
** $P<0.01$

Thafveln 1985a), and many cases may remain undiagnosed. Jeffcott (1980) in his series of 443 referred back cases reported an incidence of 15 per cent of chronic sacroiliac strain. Presumably, there must be a range of severity of the lesions occurring, so that many horses with only mild damage appear to perform to the satisfaction of their trainers or riders and therefore go unnoticed.

The most consistent clinical feature was a longstanding history of reduced performance at exercise. This was more readily noticed in riding horses at slow speeds, whereas in the harness racing horses it was only evident at racing pace. The condition is not confined to these two types of horses and is an important clinical entity in galloping and National Hunt horses (Jeffcott 1980). The correlation of the side of lameness with the sacroiliac lesion was usually, but not always, made correctly. This was probably due to the difficulty of precisely defining the lameness and the leg primarily involved. In addition, the condition probably always causes some abnormality of gait of both hindlimbs.

It is interesting that the clinical picture once recognised is not usually progressive. Treatment appears to be very difficult and is usually aimed at overcoming the sacroiliac damage by progressively building up the muscles of the quarters and back. Improvement in muscular tone and fitness tends to counteract the clinical signs of poor hindlimb impulsion (Jeffcott 1982). In mild cases this type of management has been successful, although once fit the horse must be kept fit all the time and not allowed to rest or it would lose muscle tone and return to the original state.

The histopathological findings were surprising. There was evidence of cartilaginous degeneration in all cases, but only to the same extent as that seen in normal horses (Dalin and Jeffcott 1985a; Ekman *et al* 1985). In only two horses was there substantive histological evidence of an arthrosis (**Cases 3** and **11**). The most significant gross finding was alteration or enlargement of the articular surfaces to produce a widening of the joints or gross uneveness. These extensions of the joint have previously been referred to as 'spur formations' (Jeffcott 1982, 1983b), but this has now been shown to be erroneous because they cannot be classified histologically as osteophytes. Similar enlargements of the joint surfaces were not seen in the normal horses (Dalin and Jeffcott 1985a) and their pathogenesis is uncertain. It may be that they arise from prolonged mild instability of the joint resulting in a gradual remodelling and consequent enlargement of the joint surfaces. This may have an underlying congenital origin exacerbated by the type of work involved or some traumatic incident.

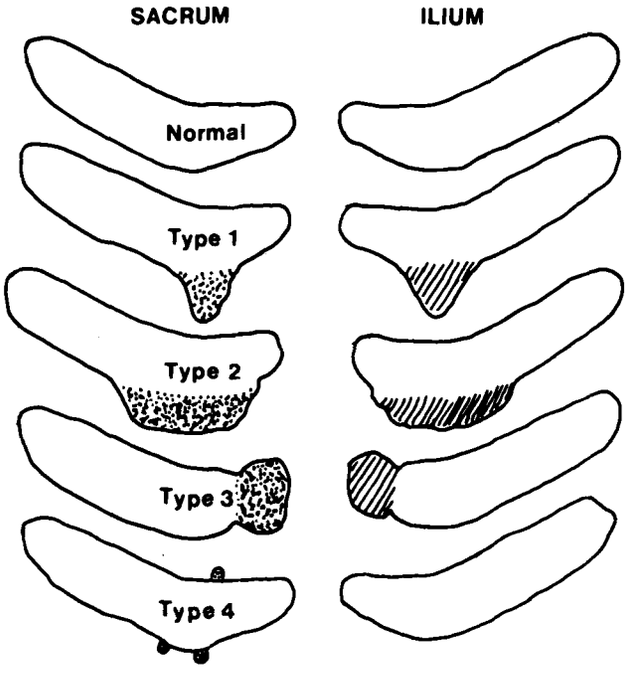

Fig 4. Diagrammatic representation of the articular surfaces of the left sacroiliac joint to illustrate the different features of extensions of the articular surfaces and osteophyte positions in horses exhibiting signs of chronic sacroiliac disease. The dotted shading indicates the extent of the areas affected on the sacral surface and the hatched areas on the iliac surface denotes the articular notches

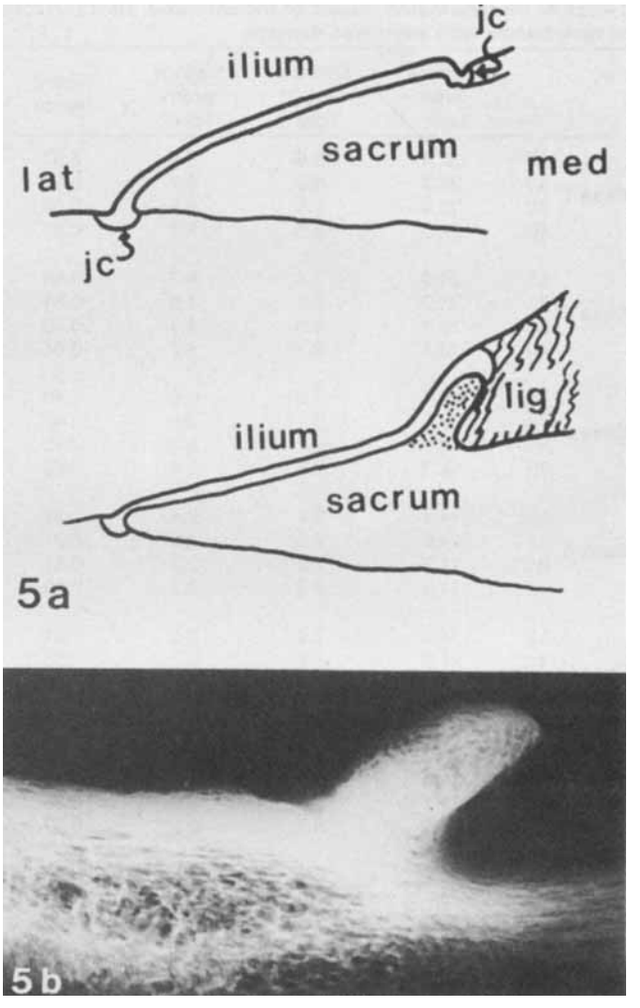

Fig 5. (a) Diagrammatic cross-sectional views through the mid portion of the sacroiliac joint to show the contour of the sacral and iliac articular surfaces in normal (upper) and in Type 3 *lesions (lower). This gives the sacral surface a concave appearance rather than the normal flattened outline. (jc joint capsule). (b) Radiograph of a slab from the left sacral wing of a five-year-old Holstein gelding* **(Case 5)** *with a* Type 3 *lesion. The prominent extension is directed in about 40 degrees angulation from the normal plane of the joint surface*

The pathological picture of chronic sacroiliac disease has not been completely elucidated, but it is clear that arthrosis is not nearly as prevalent as has been suggested by Rooney (1977). The mechanics of sacroiliac movement are largely unknown and the limits of stress to which the joint is subjected during exercise have not been measured. Rooney (1981) has put forward a theoretical basis for the forces acting on the sacroiliac region that lead to articular cartilage degeneration (ie, arthrosis). However, it seems as if his pathological

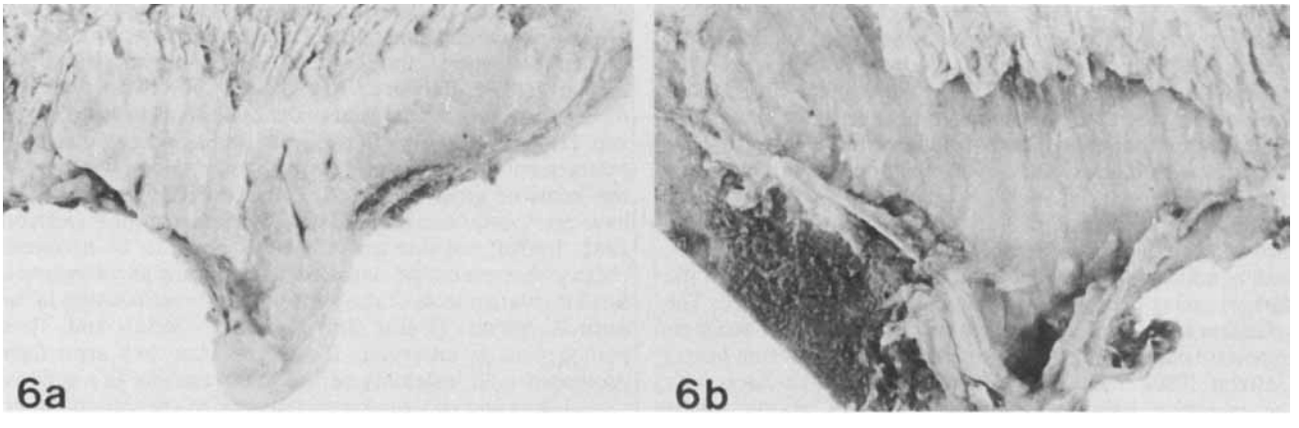

Fig 6. Sacroiliac articular extension, Type 1, *in a nine-year-old Swedish Warmblood gelding* **(Case 1)** *showing a large triangular-shaped extension on the caudal aspect of the right wing of the sacrum (a) and the corresponding notch on the articular surface of the right ilium (b)*

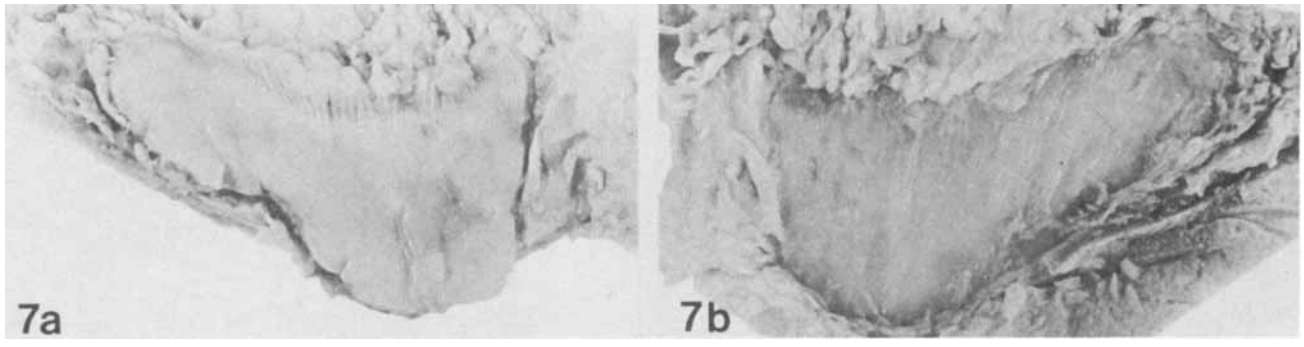

Fig 7. Sacroiliac articular extension, Type 2, *in a six-year-old Standardbred gelding* **(Case 2)** *showing widespread extension of the caudal aspect of the left articular surface (a) resulting in increased surface area of both sacral and iliac (b) surfaces*

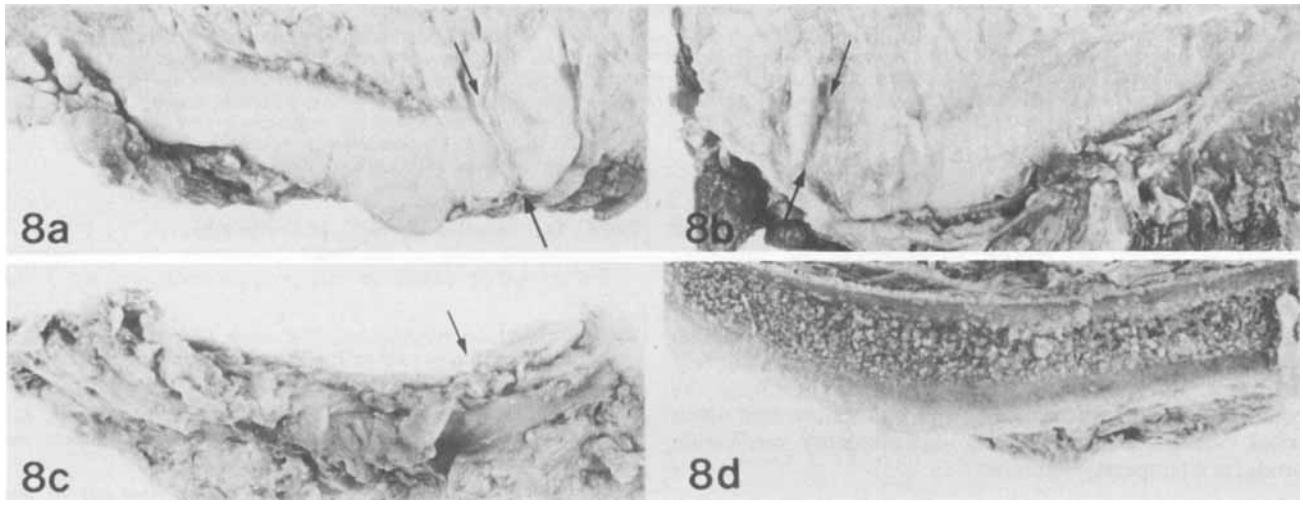

Fig 8. (a) Large dorsal eminence covered by articular cartilage, Type 3, *on the most medial aspect (arrows) of the left sacral joint surface of a five-year-old Holstein gelding* **(Case 5).** *(b) A depression on the iliac surface (arrows) corresponding to the dorsal eminence in Fig 8a. (c) Caudocranial view of the same area as in Fig 8a showing the concavity of the sacral surface caused by the dorsal eminence on the medial aspect of the sacral wing (arrow). A radiograph of a slab from this area is shown in Fig 5b. (d) Caudocranial view of a section through the same area as in Fig 8b showing the convex iliac surface*

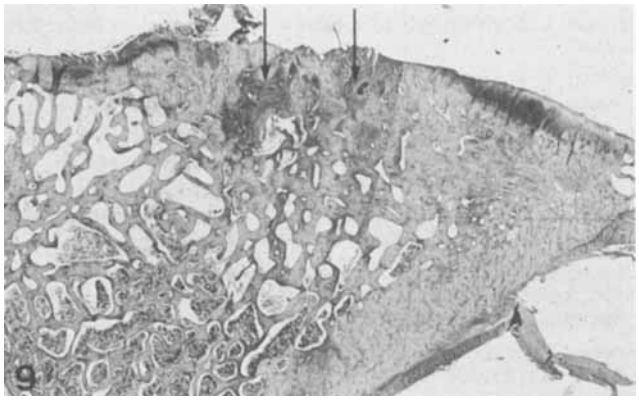

Fig 9. Sagittal section of the caudal part of the left sacral wing from a seven-year-old Thoroughbred mare **(Case 3)** *showing obvious pathological changes. In the centre, the joint cartilage is completely lost and large so-called pseudocysts (arrows) filled by granulation tissue are seen in the subchondral bone. To the right, the surface of the joint cartilage is slightly frayed and to the left, the cartilage is disintegrating. The bone trabeculae of the subchondral bone are thick and plump. Haematoxylin and eosin × 4*

interpretation of the lameness involved is incorrèct and Crawford (1982) has questioned his theoretical analysis. It is important to remember that this joint is probably never fully loadbearing and the forces to which it is subjected are more shearing in type than compressive (Dalin and Jeffcott 1985a; Ekman *et al* 1985). This would account for the articular degeneration that occurs with normal ageing (Dalin and Jeffcott 1985a; Ekman *et al* 1985) and if unduly stressed by exercise could lead to mild articular instability which could result in a gradual remodelling with enlargement of the joint. The movements of the sacroiliac joint in man have been investigated *in vivo* (Beal 1982; Bellamy, Park and Rooney 1983), although the results are somewhat contradictory. In any case data are not meaningful because it is wrong to draw analogies between two species that have such a different posture and means of locomotion. Sacroiliac movement in the normal horse seems to be extremely limited (Getty 1975; Dalin and Jeffcott 1985a).

The underlying cause of acute damage to the sacroiliac joint and the ventral sacroiliac ligaments is undoubtedly traumatic (Rooney *et al* 1969). It is also clear that some of these cases may progress to the chronic state (Jeffcott 1980). The pathogenesis of the apparently spontaneous or insidious cases

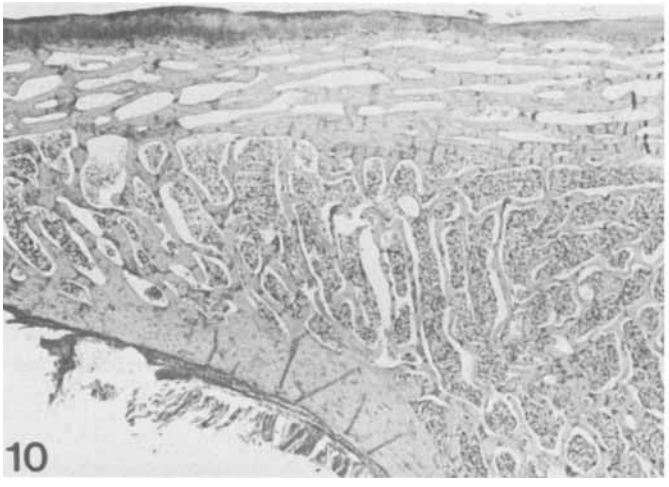

Fig 10. Sagittal section of the left sacral wing from a nine-year-old Swedish Warmblood gelding **(Case 1).** *To the left is the base of an articular extension* (Type 1) *with normal bone and with cartilage showing only slightly irregular stainability. The subchondral bone trabeculae are arranged parallel with the joint surface. Haematoxylin and eosin × 4*

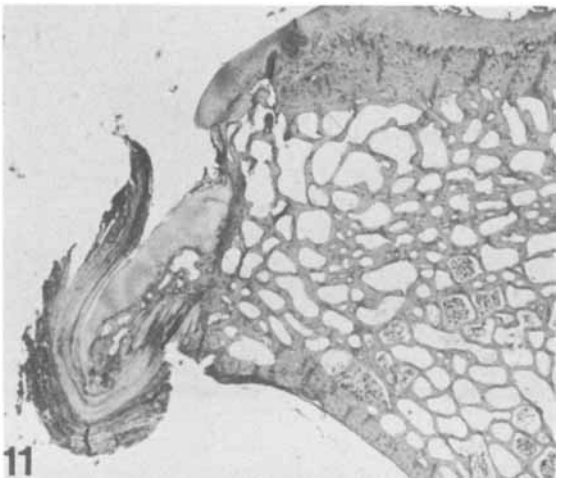

Fig 11. Sagittal section of the caudal edge of the left sacral wing from a 12-year-old Swedish Warmblood gelding **(Case 8).** *To the left, outside the preformed articular surface, there is a small osteophyte covered by a normal hyaline cartilage. The osteophyte has a mature appearance and it seems to exert pressure on the fibrous joint capsule, which is pushed in a ventral direction. Haematoxylin and eosin × 4*

of sacroiliac damage needs further investigation. This would require a means of precise measurement of sacroiliac movement according to gait and speed. Another important factor is to determine the extent and origin of the pain involved. It may be that the joint extensions at the caudal aspect of the sacrum can create pressure on the adjacent obturator nerves. If this were true, it might create the equivalent situation of sciatica or lumbago seen in man which could perhaps explain why anti-inflammatory medication produces a temporary response.

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