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General Articles

Physiological stimuli of thirst and drinking patterns in ponies

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Summary

The stimuli that elicit thirst were studied in four ponies. Nineteen hours of water deprivation produced an increase in plasma protein from 67 ± 0.1 g/litre to 72 ± 2 g/litre, a mean (± se) increase in plasma sodium from 139 ± 3 to 145 ± 2 mmol/litre and an increase in plasma osmolality from 297 ± 1 to 306 ± 2 mosmol/litre. Undeprived ponies drank 1.5 ± 0.9 kg/30 mins; 19 h deprived ponies drank 10.2 ± 2.5 kg/30 mins and corrected the deficits in plasma protein, plasma sodium and plasma osmolality as well as compensating for the water they would have drunk during the deprivation period.

In order to determine if an increase in plasma osmolality would stimulate thirst, 250 ml of 15 per cent sodium chloride was infused intravenously. The ponies drank when osmolality increased 3 per cent and when plasma sodium rose from 136 ± 3 mmol/litre to 143 ± 3 mmol/litre. Ponies infused with 15 per cent sodium chloride drank 2.9 ± 0.7 kg; those infused with 0.9 per cent sodium chloride drank 0.7 ± 0.5 kg. In order to determine if a decrease in plasma volume would stimulate thirst, ponies were injected with 1 or 2 mg/kg bodyweight (bwt) frusemide. Plasma protein rose from 68 ± 2 g/litre pre-injection to 75 ± 2 g/litre 1 h after 1 mg/kg bwt frusemide and to 81 ± 1 g/litre 1 h after 2 mg/kg bwt frusemide. Ponies treated with 1 mg/kg frusemide voided 5.3 ± 0.81 kg urine and drank 3.0 ± 2 kg water. Ponies injected with 2 mg frusemide voided 6.4 ± 5 kg urine and drank 4.3 ± 1.4 kg water. Controls injected with 0.9 per cent sodium chloride voided 1.5 ± 0.5 kg urine and drank 0.9 ± 0.6 kg water. When food and water were freely available the ponies drank 27 ± 8 mins/day, 89 per cent of which occurred within a period from 10 mins before to 30 mins after feeding. They drank 8.7 ± 0.9 kg/day at a rate of 552 ± 232 ml/min. It was concluded that ponies usually drink periprandially and when challenged with either an increase in the osmotic pressure of their body fluids or a decrease in the volume of their body fluids they responded by increasing their water intake.

Introduction

THE drinking behaviour of horses has not been studied in detail. A recent review (Hinton 1978) indicated the paucity of information. Fonnesbeck (1968) has observed the effect of different diets on water intake of horses. Most studies of free ranging horses have been made in the arid American west where horses drink only once a day or once every two days (Pellegrini 1971; Feist and McCullough 1976). The physiological response to water deprivation has received more attention. Tasker (1966, 1967a) and Carlson, Rumbaugh and Harrold (1979) have observed the physiological effects of water deprivation and the compensatory drinking that follows. Other Equidae have received more attention. The classical studies of Dill and his co-workers (Dill, Yousef, Cox and Barton 1977; Yousef, Dill and Mayes 1970) on the burro and those of Maloiy (1970) and Maloiy and Boarer (1971) on the Somali donkey have indicated how well asses can withstand dehydration and how precisely they can compensate.

The purpose of this experiment was to investigate the physiological stimuli of thirst and drinking patterns in the horse. Fitzsimons (1972) has implicated two stimuli of thirst: an increase in osmotic pressure of the plasma and a decrease in blood volume. An attempt was made to determine whether horses responded to these stimuli by drinking and to compare the threshold change in plasma that accompanied the onset of drinking in the horse with that observed in other species.

Materials and methods

Stimuli of thirst

Four pony geldings (185 to 220 kg) were used. They were housed in box stalls and fed 1.5 kg of alfalfa hay and 0.5 kg grain twice a day. Water was available from buckets *ad libitum* except as specified below.

The experiments were run in a Latin square design in which a pair of ponies were taken to a separate building and placed in 1.2 × 1.6 m stocks. One pony served as the experimental and the other as the control subject. Forty-eight hours later the experiment was repeated with the roles of the ponies as experimental and control reversed. Experiments were carried out in late morning. Each pony served once a week as control and once as experimental. Each animal was tested three times with each treatment: water deprivation, sodium chloride infusion and frusemide (Lasix; Hoechst) 1 mg/kg bodyweight (bwt). The ponies were tested only once with 2 mg/kg bwt frusemide.

Blood samples were collected from the jugular vein into tubes containing 143 iu lithium heparin. Osmolality was measured using a freezing point depression osmometer. Plasma protein was measured by means of a refractometer. Sodium was determined using a flame photometer.

Water deprivation. — The ponies were water deprived for 19 h. Water intake of the undeprived ponies was measured. Blood samples were taken before (0 time) and at 15, 30 and 60

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mins after water was made available. The latency to drink and the amount of water drunk in 30 mins was measured.

Sodium chloride infusions. — A polyethylene catheter (inner diameter 0.023 cm, outer diameter 0.038 cm) was introduced into the jugular vein through a 15 gauge needle and connected via silastic tubing to a peristaltic infusion pump that infused at a rate of 25 ml/min. A total of 250 ml of 15 per cent sodium chloride were delivered to the experimental ponies. Control ponies received 250 ml of 0.9 per cent sodium chloride. Water was made available to the ponies as the infusion was started. Blood samples were taken before infusion, at the time the pony took its first drink and at the end of the infusion (10 mins) and 30 mins after the time of the infusion.

Frusemide. — The ponies were fitted with urine collecting harnesses and urine was collected for the duration of the experiment. Blood samples were taken immediately before administration of 1 or 2 mg/kg bwt frusemide or a control injection of 1 ml/50 kg bwt 0.9 per cent sodium chloride. Blood samples were also taken at 30 mins, 1, 2 and 3 h post injection.

Paired tests were used to compare control and experimental values for each pony's mean intake, plasma protein, sodium, and osmolality at each time measured. All values given are mean ± standard error of the mean (sem).

Drinking patterns

Four Shetland type ponies (two of the ponies that served in the stimuli of thirst experiment and two different ponies) were studied. Each pony was kept in a 2.1 × 2.9 m box stall in a room with no other animals present. The isolation was necessary to avoid social facilitation of feeding and drinking. Feed and water were available *ad libitum* from buckets over which photoelectric cells and light beams were positioned. Each eating and drinking episode was recorded on an event recorder (Esterline Angus). Water and feed were weighed and refilled twice daily. The ponies were fed three diets, a grain diet composed of corn, oats, bran and soybean meal (342 kcal/kg), the grain diluted with 25 per cent sawdust by weight (2.57 kcal/g) and the grain diluted with 50 per cent sawdust by weight (1.71 kcal/g). A mixture of 50 per cent molasses in water was added at a rate of 250 ml/kg of diet to prevent the ponies from separating the feed into its components. The details of the effect of the calorie dilution on the food intake of the ponies is reported elsewhere (Houpt, Laut, Kirk and Carter 1982). Drinking was considered to be periprandial if it occurred within the period 10 mins before or 30 mins after eating (Houpt, Baldwin and Houpt 1983).

Results

Stimuli of thirst

Water deprivation. — Fig 1 illustrates the effect of 19 h water deprivation on subsequent water intake. Deprived ponies drank with a latency of 10.3 ± 5.9 secs; undeprived ponies drank with a latency of 1937 ± 1520 secs. The undeprived ponies drank 1.5 ± 0.9 kg/30 mins and the deprived ponies drank 10.2 ± 2.5 kg/30 mins ($t = 5.19$, $P<0.05$). The effects of water deprivation and rehydration on plasma protein, osmolality and sodium are shown in Table 1. All were significantly elevated following overnight deprivation but returned to control levels within 15 mins after water was made available. There was a significant correlation between the change in osmotic pressure and the amount drunk ($r = 0.73$; $P<0.05$). The ponies overcompensated slightly for the deprivation by drinking more than their control overnight intake (7.2 ± 1.3 kg/19 h). This is reflected in the plasma osmolality that fell slightly, but not significantly, below the control level after 1 h access to water.

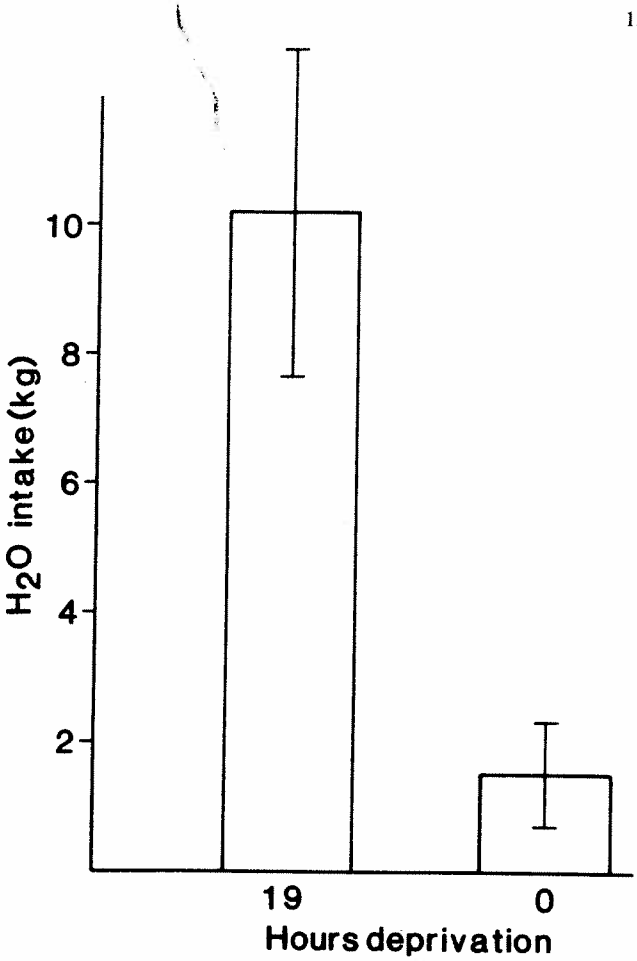

Fig 1. Water intake of ponies following water deprivation. Four ponies were deprived of water for 19 h and undeprived (0 h) before being offered water for 30 mins. The vertical lines represent the se of the means of three replications on each pony under eact condition

Sodium chloride infusion. — Fig 2 illustrates the effect of infusion of a hypertonic solution on water intake. The ponies infused with 15 per cent sodium chloride drank 2.9 ± 0.7 kg and the ponies infused with 0.9 per cent sodium chloride drank 0.7 ± 0.5 kg ($t = 3.98$, $P<0.05$). The effect of infusion of a hypertonic solution on plasma protein, sodium and osmolality is shown in Table 2. Plasma sodium was significantly increased at the end of the infusion and plasma protein had fallen indicating that water had moved from cells into the blood in response to the hypertonicity of the blood.

TABLE 1: Effect of water deprivation on mean (± sem) values of plasma protein, sodium and osmolality

	Plasma protein (g/litre)		Sodium (mmol/litre)		Osmolality (mosmol/litre)	
Time (min)	19h deprived	0 h deprived	19 h deprived	0 h deprived	19 h deprived	0 h deprived
0	71.8 ± 2.1*	67.5 ± 1.2	145 ± 2*	139 ± 3	306 ± 1.5*	297 ± 1.4
15	69.8 ± 2.4	65.6 ± 1.5	39 ± 3	140 ± 4	300 ± 5.6	293 ± 1.6
30	69.1 ± 2.1	65.4 ± 1.7	138 ± 2	137 ± 3	289 ± 2.7	291 ± 2.7
60	68.2 ± 2.2	66.0 ± 1.1	137 ± 2	137 ± 2	289 ± 2.0	290 ± 1.3

* Significantly greater than undeprived

TABLE 2: Effect of sodium infusion on mean (± sem) values for plasma protein, sodium and osmolality

Time	Plasma protein (g/litre)		Sodium (mmol/litre)		Osmolality (mosmol/litre)	
	15% sodium chloride	0.9% sodium chloride	15% sodium chloride	0.9% sodium chloride	15% sodium chloride	0.9% sodium chloride
0	67.9 ± 1.2	67.5 ± 0.7	136 ± 3	134 ± 1	293 ± 2.1	293 ± 1.6
1st drink	63.8 ± 1.8	(65.9 ± 1.2)*	140 ± 3	(141 ± 2.5)*	301 ± 2.9	(299.5 ± 6.0)*
Post infusion	59.0 ± 1.4†	63.9 ± 1.0	143 ± 2†	137 ± 2	303 ± 3.7	291 ± 1.2
30 mins post infusion	61.3 ± 1.6	64.7 ± 1.2	140 ± 2	133 ± 3	296 ± 2.86	291 ± 2.1

()* Values of the two control ponies that drank
† Significantly greater than 0.9 per cent sodium chloride infused

Frusemide. — Fig 3 illustrates the effect of frusemide on water intake. The ponies drank 3.0 ± 2 kg when injected with 1 mg/kg frusemide and 4.3 ± 1.4 kg when injected with 2 mg/kg frusemide. The latter was significantly greater than control intake of 0.9 ± 0.6 kg ($t = 3.53$, $P<0.05$). The effects of 1 mg/kg frusemide on plasma protein sodium and osmolality are given in Table 3. Plasma protein inereased significantly through the third hour. Plasma protein fell even further when 2 mg/kg frusemide was injected. The pre-injection plasma protein was 71 ± 3.0 g/litre; that at 30 mins was 80 ± 2.0 g/litres; that at 1 h was 81 ± 1.0 g/litre; that at 2 h was 80.8 ± 3.0 g/litre; and that at 3 h was 78 ± 2.0 g/litre. All of the post injection values were significantly higher than the control values ($t = 4.40$, $P<0.05$). There was a significant correlation between the amount of urine voided and the amount of water drunk ($r = 0.72$, $P<0.05$). The ponies treated with 1 mg/kg frusemide voided 5.3 ± 0.81 kg urine; 0.9 per cent sodium chloride injected ponies voided 1.5 ± 0.5 kg urine. When injected with 2 mg/kg frusemide the ponies voided 6.4 ± 5 kg urine; when injected with 0.9 per cent sodium chloride they voided 0.8 ± 0.6 kg urine.

Drinking patterns

The amount drunk, the time spent drinking, the rate of drinking and the percentage of drinking that occurred in association with feeding are shown in Table 4. The ponies

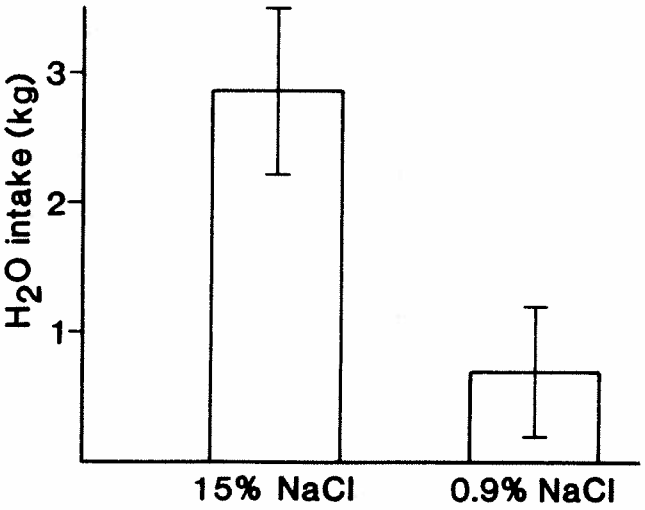

Fig 2. Water intake by ponies in response to sodium chloride infusion. Four ponies were infused with 250 ml 15 per cent sodium chloride and 0.9 per cent sodium chloride. Water was available during and for 30 mins after the infusion. The vertical lines represent the sem of the means of three replications on each pony under each condition

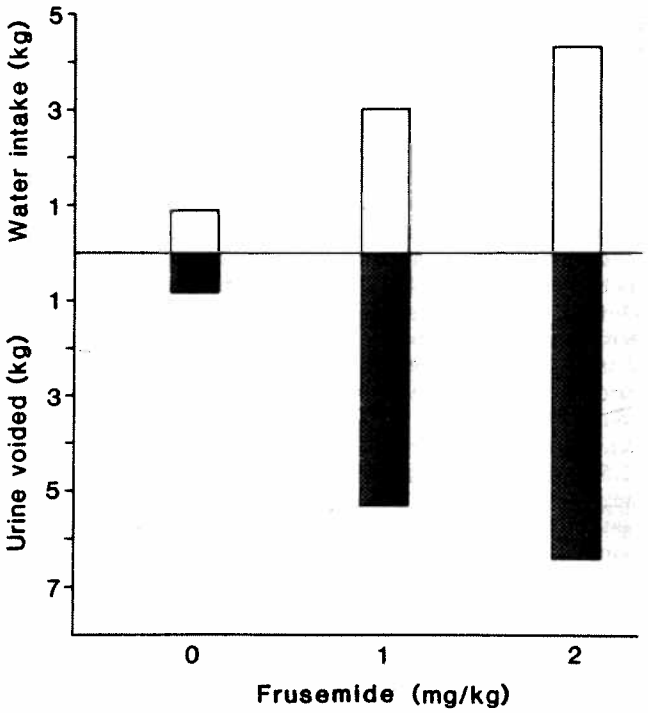

Fig 3. Water intake and urine output by ponies in response to frusemide. The upper half of the diagram represents water intake; the lower half represents urine output. The four ponies were treated with 0.9 per cent sodium chloride (0), 1 mg/kg and 2 mg/kg frusemide

TABLE 3: Effect of frusemide on mean (± sem) values for plasma protein, sodium and osmolality

Time (mins)	Plasma protein (g/litre)		Sodium (mmol/litre)		Osmolality (mosmol/litre)	
	Frusemide†	0.9% sodium chloride	Frusemide†	0.9% sodium chloride	Frusemide†	0.9% sodium chloride
0	67.9 ± 1.7	68.0 ± 1.1	139 ± 2	139 ± 2	294 ± 1.3	297 ± 2.3
30	75.9 ± 1.7*	67.3 ± 0.9	137 ± 2	141 ± 2	295 ± 3.2	292 ± 2.4
60	74.5 ± 2.2*	66.9 ± 1.2	137 ± 2	143 ± 3	293 ± 2.6	295 ± 2.5
120	72.3 ± 1.6*	67.1 ± 1.0	136 ± 4	143 ± 3	292 ± 2.4	294 ± 1.7
180	69.6 ± 2.3	66.9 ± 1.7	137 ± 2	141 ± 4	292 ± 3.5	292 ± 1.9

* Significantly ($P<0.06$) different from control (0.9 per cent sodium chloride)
† The dose of frusemide was 1 mg/kg

TABLE 4: Mean (± sem) values for daily water intake and time spent drinking by four ponies

Diet	Drinking time min/24 h	Intake kg/24 h	Drinking rate ml/min	Percent periprandial*
Grain + 0% sawdust	27 ± 8	8.7 ± 0.9	552 ± 232	89
Grain + 25% sawdust	26 ± 8	11.3 ± 1.6	678 ± 248	89
Grain + 50% sawdust	21 ± 9	11.1 ± 1.8	860 ± 284	86

* Periprandial refers to all drinking occurring within a period 10 mins before or 30 mins after feeding

drank 8 to 11 kg of water per day. The amount consumed rose when indigestible material was added to the diet but there was no significant correlation between the proportion of sawdust and the amount consumed. The time spent drinking varied from 27 mins/day on the grain diet to 21 mins/day on the 50 per cent sawdust diet. The rate of drinking rose in proportion to the amount of indigestible material in the diet. The majority (86 to 89 per cent) of the water intake took place in association with feeding.

Discussion

Stimuli of thirst

Horses are able to correct a deficit in body fluid whether the cause is simple water deprivation, an increase in sodium concentration or a decrease in extracellular plasma volume. An overnight water deprivation resulted in a significant increase in both osmotic pressure and plasma protein. The latter reflected the fall in plasma volume. The ponies compensated for deprivation by drinking an amount equal to or greater than the amount they consumed overnight when water was freely available. The ingested water functioned to restore plasma osmotic pressure and blood volume to normal within an hour. These results are similar to those obtained in studies of donkeys (Yousef *et al* 1970; Maloiy 1970).

Changes seen after 19 h water deprivation are similar to those seen in other species. The ponies showed a 3 per cent increase in plasma osmolality whereas humans showed a 3 per cent increase (8 mosmol) and dogs a 4 per cent increase (10 mosmol) following 24 h water deprivation (Ramsey, Rolls and Wood 1977; Rolls *et al* 1980). The 6 per cent increase in plasma protein observed in the ponies is similar to the 6 per cent increase, from 73 to 77 g, found in 24 h water deprived humans (Rolls *et al* 1980). This represents slightly less than a 6 per cent increase in plasma volume in both species.

The threshold for osmotically induced thirst appears to be an 8 mosmol increase or a 3 per cent increase. Dogs drink when the osmotic pressure of their plasma is 10 mosmol above normal, a 3 to 6 per cent increase (Wood, Rolls and Ramsey 1977). A 2 to 3 per cent increase in osmolality is sufficient to stimulate thirst in rats (Fitzsimons 1972).

The deficits produced in this study are similar to those expected in field conditions where wild horses have been observed to travel long distances for a daily drink (Feist and McCullough 1976; Berger 1977; Miller and Denniston 1979). Presumably under these arid conditions there is an increase in osmotic pressure and a decrease in blood volume. Less severe water deprivation would be expected to lead first to an increase in plasma osmotic pressure since the circulation is protected at the expense of tissue, gastrointestinal and cellular fluid. An increase in osmotic pressure may also account for the tendency of horses to drink at the time they eat. This behaviour, periprandial drinking, is probably caused by the movement of fluid into the gastrointestinal tract, as the intestinal osmotic pressure will increase as the ingested food is digested.

Hypovolaemia or a decrease in blood volume would be expected to occur in a variety of clinical situations: blood loss, diarrhoea (Tasker 1976b), heavy sweating as in endurance horses, or, as indicated in this study, large urinary losses secondary to diuretic treatment.

Hinton (1977) has postulated that horses participating in endurance rides may fail to drink despite considerable fluid loss because there is no change in osmotic pressure but only an isosmotic loss of water and electrolytes in sweat. These results indicated that horses will drink in response to an isosmotic loss of blood volume. The decrease of plasma volume sufficient to stimulate thirst is 6 per cent. Laboratory rodents can also be stimulated to drink by decreasing blood volume. This is usually accomplished experimentally by injection of a hyperoncotic colloid. Frusemide has been shown to reduce plasma volume and increase water intake in sheep (Zimmerman and Stricker 1978) at a dose of 10 mg/kg and in rats (Rabe 1975). The dose necessary to stimulate significantly intake in this small group of horses (2 mg/kg) is on the high end of the recommended clinical dose of frusemide.

Drinking patterns

The amount consumed by *ad libitum* drinking ponies is similar to that reported previously (Tasker 1967a; Fonnesbeck 1968). What is of most interest is the rapid rate of drinking, from 500 ml to almost a litre/min in undeprived animals. It is also of interest that the ponies drank most water in association with feeding. This may be an attempt to moisten a dry diet by animals that eat grass under natural conditions. Waring (1983) has reported an exaggerated case of periprandial drinking in which a horse dunked its hay into the water bucket. Other species of animals are also periprandial drinkers. For example both pigs (Houpt *et al* 1983) and rats (Kissileff 1969) drink in association with meals. Management practices should accommodate the horse's drinking patterns by providing water *ad libitum* or in association with meals.

In summary, horses respond to both osmotic and hypovolaemic challenges by increasing their water intake and thereby correcting the deficit. The equine practitioner should be aware that some common pharmacological manoeuvres, eg, administration of frusemide before performance, can have other side effects such as the behavioural and physiological ones noted here.

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BOOK REVIEW

Proceedings of the fifth Bain-Fallon memorial lectures: Equine lameness by K. Johnson, R. Rose, L. Jeffcott, A. Gabel and R. Sande. Available from Australian Equine Veterinary Association, 134-136 Hampden Road, Artarmon, NSW 2064, Australia

THIS book is a compilation of contributions by authors who originate from Australia (Johnson and Rose), Great Britain (Jeffcott) and USA (Gabel and Sande). Much of the information has been documented more extensively elsewhere, but is usefully presented here in a summarised, practical form. Some recent previously unpublished data relating to follow up statistics for various treatment methods are presented but, unfortunately, much of this concerns Standardbred race horses, and is of limited application for the majority of British clinicians.

Radiography of the limbs is discussed in detail and there are many useful practical recommendations for patient positioning and angling of the X-ray beam. Radiographic interpretation is covered in depth and the importance of a systematic approach in film reading is emphasised; it provides an excellent review for anybody involved in reading X-rays. A high incidence of radiographic abnormalities revealed in a group of apparently normal horses is described and this chapter is essential reading for anybody involved in using radiology as part of the pre-purchase examination.

General principles of lameness investigation and diagnosis are discussed. Functional anatomy is described and related to the incidence of problems in various locations. Each limb is covered systematically, although not comprehensively, eg, there is little mention of laminitis. The problems relating to poor foot conformation and bad shoeing are alluded to frequently but not discussed in depth. Despite these limitations the authors present a great wealth of personal experiences.

There is little overlap by different authors, but occasionally differences of opinion are aired: for example, there is controversy as to whether lateral neck X-rays are best obtained in the standing position or in lateral recumbency, under general anaesthesia. In some instances more detailed descriptions would be beneficial. There are several references to the medial collateral ligament test, but there is no description of how it should be performed. There is a number of typographical errors, eg, 'I no longer do cunean tendonotomy because the surgery is not a reasonable treatment and *does* improve performance in the long run'. Surprisingly, there is little mention of a number of recent innovations in diagnostic techniques, eg, nuclear scanning and treatment methods and drugs such as sodium hyaluronate, but presumably this is because authors have wisely restricted themselves to personal experiences.

There are several controversial statements which provide food for thought. It is suggested that surgical arthrodesis of the distal intertarsal and tarsometatarsal joints as a treatment for bone spavin seldom renders a horse completely sound. Fractures of the distal tibia are said to carry a guarded prognosis for return to athletic exercise if treated conservatively. In one instance, it is suggested that non-steroidal anti-inflammatory drugs may 'protect horses from injury' but elsewhere the potential hazards of treatment with phenylbutazone are underlined by reference to a study in which synthesis of proteoglycans in normal articular cartilage was suppressed by phenylbutazone; it was concluded that an analgesic drug may mask the 'protective effect' of a painful synovitis and allow further mechanical damage to the articular cartilage.

Although it lacks illustration, the book is generally well presented, easy to read and provides a good list of references. For anybody interested in lameness and/or orthopaedic surgery, it makes valuable, interesting and at times thought provoking reading.

SUSAN J. DYSON