

Case Report

Splenectomy in a foal to control intra-abdominal haemorrhage caused by splenic rupture

M. A. MUURLINK*, J. P. WALMSLEY†, C. J. SAVAGE AND R. C. WHITTON

Equine Centre, University of Melbourne, 250 Princes Highway, Werribee, Melbourne 3030, Australia; and †The Liphook Equine Hospital, Liphook, Hampshire, UK.

Keywords: horse; splenectomy; intra-abdominal haemorrhage; splenic rupture; troponin I

Summary

Intra-abdominal haemorrhage in horses can be due to rupture of the spleen and may result in severe blood loss. The assessment and resuscitation of a foal that collapsed due to intra-abdominal haemorrhage is presented. Diagnosis of the source of the blood loss required exploratory laparotomy and confirmed splenic capsular rupture and haemorrhage. Splenectomy using a 17th rib resection approach was used to control the haemorrhage. Complications included acute cardiac muscle damage, which was monitored using echocardiography and troponin I levels. The successful surgical management of the ongoing blood loss in this foal suggests that splenectomy should be considered an option to control severe blood loss due to splenic rupture.

Introduction

Rupture of the spleen is uncommon in horses and may result in severe blood loss (Finocchio 1971; Pusterla *et al.* 2005; Blikslager and Wilson 2006). The clinical presentation is usually dominated by signs of acute blood loss and often the condition is rapidly fatal. The cause of the rupture is rarely determined in cases where there is no pre-existing splenic pathology, and in these cases it is presumed to be a result of trauma (Steiner 1981).

This case report presents the management and successful outcome of a foal with splenic rupture that presented with tachycardia and depression which progressed to recumbency and circulatory shock. Resuscitation, surgical management and post operative care are described. Complications encountered in this case included acute cardiac muscle damage. Its resolution as determined by clinical findings, echocardiography and plasma troponin I levels is presented.

Case details

History

A 10-week-old Thoroughbred colt weighing approximately 190 kg was presented to the referring veterinarian with intermittent pawing, inappetance and tachycardia of several hours duration. It was treated with flunixin meglumine, gentamicin sulphate and intranasal oxygen prior to referral.

Clinical findings

On presentation at the University of Melbourne Equine Centre the colt had pale moist mucous membranes, normal skin turgor, a heart rate (HR) of 88 beats/min and normal gastrointestinal sounds on auscultation. There were no external signs of trauma.

A venous blood sample revealed a slightly decreased packed cell volume (27%, reference range [rr] 31–44%), hypoproteinaemia (43 g/l, rr 52–68 g/l), a markedly elevated lactate (19.6 mmol/l, rr <2 mmol/l) and a metabolic acidosis (pH 7.187, bicarbonate 16.4 mmol/l, pCO₂ 45.1 mmHg). A full blood count and biochemical analysis subsequently performed revealed a leucocytosis (15.0 × 10⁹/l, rr 5.3–12.2 × 10⁹/l), characterised by a neutrophilia (10.0 × 10⁹/l, rr 2.8–9.2 × 10⁹/l) with a left shift (bands 0.3 × 10⁹/l, rr 0–0.15 × 10⁹/l) and a slightly elevated creatinine concentration (0.26 mmol/l, rr 0.11–0.21 mmol/l). (All reference ranges except troponin I and lactate levels from Koterba *et al.* 1990.) A blood clotting profile was not performed.

Frank blood was recovered on abdominocentesis, and transabdominal ultrasound examination revealed large amounts of swirling material of mixed echogenicity suggestive of haemoperitoneum.

During the ultrasound examination the foal collapsed into lateral recumbency. No heart sounds could be auscultated, mucous membranes were white and there were no palpebral reflexes. Resuscitation included intranasally delivered oxygen instituted at 6 l/min and circulatory support given with

*Author to whom correspondence should be addressed.

hypertonic sodium chloride (1 l 7% sodium chloride) followed by isotonic fluids (1 l 0.9% sodium chloride, followed by 1.5 l 0.45% sodium chloride with 2.5% glucose) delivered through a polyurethane (Milacath 14 gauge x 13 cm)¹ jugular vein catheter. Fresh blood from a universal donor, which was not cross matched, was also administered as it became available and a total of 3 l blood was given during resuscitation and surgery. During the period of resuscitation the HR varied from 80–154 beats/min and an electrocardiogram taken revealed ventricular tachycardia. The tachycardia was monitored for 15 min and then converted to sinus tachycardia with a lidocaine bolus (60 mg lidocaine i.v.) and a constant rate infusion of lidocaine (10 mg/min) was delivered until anaesthetic induction. During this period a HR of 100–130 beats/min was recorded. Diazepam was administered to the foal to facilitate restraint (29 mg in multiple small boluses i.v.).

A decision was made to perform an exploratory laparotomy given the foal's deteriorating clinical signs and the ongoing blood loss within the abdomen.

The lungs of the foal were preoxygenated using mask administered oxygen. Anaesthesia was induced with ketamine hydrochloride (300 mg i.v.). Propofol (60 mg i.v. to effect) was given to allow orotracheal intubation using an 18 mm cuffed endotracheal tube. Anaesthesia was maintained with isoflurane delivered in 100% oxygen using a circle breathing system. Intermittent positive pressure ventilation was performed throughout the procedure. During anaesthesia sinus rhythm was maintained with the HR varying from 60–80 beats/min. Mean blood pressure was monitored directly by catheterising the facial artery and maintained above 60 mmHg by alteration of vaporiser settings and i.v. infusions of Hartmann's solution, whole blood and phenylephrine. The foal was moved to lateral recumbency and was administered i.v. ketamine (100 mg) and propofol (50 mg to effect) to maintain adequate anaesthetic depth. Arterial blood gas analyses continued to reveal a metabolic acidosis, which was addressed using fluid administration and a small dose of i.v. sodium bicarbonate (8.4 g). Total anaesthetic time was 160 min. Anaesthetic recovery was uneventful.

Initially a ventral midline celiotomy was performed and the abdomen explored. A ruptured splenic capsule on its visceral surface with ongoing bleeding was identified and large blood clots were removed from the abdomen (**Figs 1, 2 and 3**). It was felt that adequate access to the major vessels of the spleen was not possible using the ventral approach. Endoscopically assisted splenectomy was not attempted due to time required for assembly and setup of equipment and concerns that the amount of blood in the abdomen may have obscured vision. The midline incision was closed with size 6.0 polyglactin 910 (Vicryl R) suture in simple continuous pattern in the *linea alba*, size 3.0 poliglecaprone 25 (Monocryl R) in simple continuous pattern subcutaneously, and stainless steel skin staples.

The foal was then moved into right lateral recumbency. A splenectomy was performed using a 17th rib resection as previously described (Roberts and Groenendyk 1978). Starting

from approximately the ventral margin of the *serratus dorsalis caudalis* muscle a 35 cm incision was made directly over the 17th rib. The superficial periosteum was incised. Periosteal elevators were used to elevate the periosteum and the rib was transected with an oscillating bone saw at the proximal and distal extent of the incision. A 30 cm section of the rib was removed and discarded. The deep periosteum and peritoneum was incised to provide access to the abdominal cavity. With the use of rib retractors, this approach provided adequate access to allow ligation of the splenic vessels with size 3.5 and 3.0 polydioxanone (PDS R) suture material. The pleural cavity was not penetrated. The spleen was removed and the incision closed with polydioxanone sutures in a simple continuous pattern in the deep periosteum, muscle and subcutaneous layers while staples were used for skin closure. Sterile adhesive plastic drapes were applied over both the lateral and ventral incisions for recovery and maintained for 2 days.

Benzyl penicillin (3 g) was given perioperatively. The foal was commenced on procaine penicillin (3200 mg i.m. every 12 h) and ceftiofur (1200 mg i.v. every 12 h), omeprazole (800 mg every 24 h *per os*) and flunixin meglumine (45 mg i.v. every 12 h).

No urine was passed during surgery or in recovery. Serum creatinine was slightly elevated (0.22 mmol/l) 30 min after recovery, although serum urea remained within the reference range. Following surgery i.v. Hartmann's solution with 2.5% glucose added was administered at a rate of 600 ml/h and the foal allowed nursing at hourly intervals. Urine was passed 50 min after recovery. The heart rate remained elevated over the next 12 h at 104–120 beats/min. At 12 h post surgery the foal had a packed cell volume of 28%, plasma total protein of 58 g/l, and a urine specific gravity of 1.007.

At 18 h post surgery an arrhythmia was auscultated and an electrocardiogram was performed revealing few normal QRS-T complexes demonstrating a multiform ventricular tachycardia which suggested multiple ventricular arrhythmic foci (**Fig 4**). The heart rate varied from 125–151 beats/min. An echocardiogram was performed and significant findings included variable echogenicity of the interventricular septum and reduced fractional shortening (19%).

A full blood count and biochemical profile performed at 18 h post surgery revealed serum creatinine concentration within the reference range, a left shift still evident (bands $0.6 \times 10^9/l$), elevated muscle enzymes AST 1673 u/l (rr 282–484 u/l) CK 16,470 u/l (rr 50–170 u/l) and mildly decreased albumin levels (27 g/l, rr 28–41 g/l). By this time the foal was considerably brighter and nursing well, although he had developed diarrhoea. Multiple faecal samples were collected but neither *Salmonella* nor *Clostridia* species were cultured, no parasite eggs or cysts were observed and rotavirus testing was negative. The diarrhoea was treated with i.v. and enteric fluid and electrolyte therapy and the oral administration of psyllium and yoghurt. The foal was administered 1 l of commercial hyperimmune plasma (Equiplas)² i.v. on Day 4 post surgery.

Venous blood cardiac troponin I levels were determined at 2, 6 and 11 days and at 3.5 and 5 months post surgery and revealed a marked elevation initially (18.04, 0.45, 0.06, 0.13

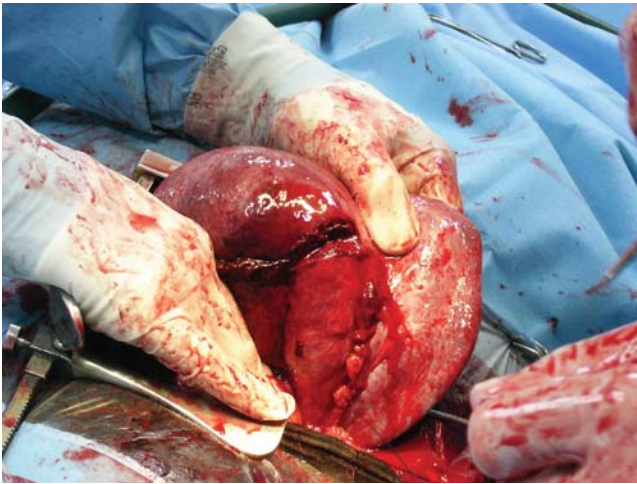


Fig 1: Intraoperative view: removing ruptured spleen from the abdomen.

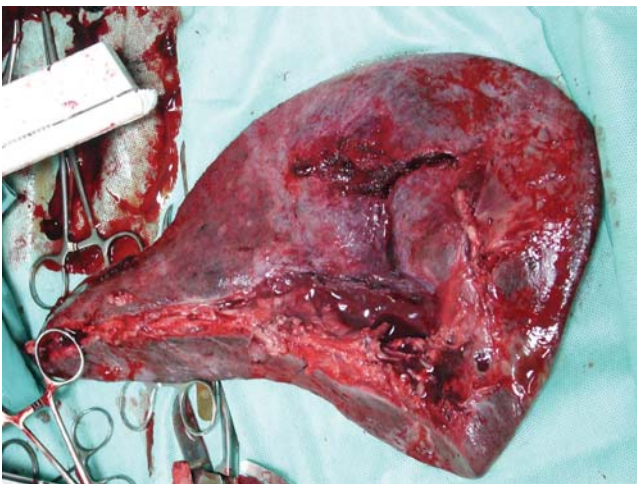


Fig 2: Spleen showing rupture of the splenic capsule on the visceral surface.

and $0.01 \mu\text{g/l}$, respectively; no reference ranges are available for foals of this age).

A significant leucocytosis had developed by 6 days post surgery ($27.3 \times 10^9/l$), which consisted of a neutrophilia with a left shift (neutrophils $17.5 \times 10^9/l$, bands 0.5×10^9), and a monocytosis ($1.7 \times 10^9/l$, rr $0.05\text{--}0.61 \times 10^9/l$). At discharge 11 days post surgery these values had increased with a leucocytosis ($38.8 \times 10^9/l$), characterised by a neutrophilia ($31.5 \times 10^9/l$) and a monocytosis ($1.9 \times 10^9/l$). At 2.5 months post surgery the foal still had a slight leucocytosis ($15.8 \times 10^9/l$, rr $6.6\text{--}14.6 \times 10^9/l$) with a mild neutrophilia ($8.8 \times 10^9/l$, rr $1.7\text{--}8.4 \times 10^9/l$). Fibrinogen levels remained within the reference range for foals of this age. During this time there was no haematological evidence of red blood cell regeneration with mean corpuscular volume and PCV remaining stable.

Mild flexural deformities of the fetlock joints developed in the foal's hindlimbs during hospitalisation; however this was not specifically treated. Mild incisional swelling at the rib resection site occurred and was monitored with diagnostic



Fig 3: Removal of large blood clots from the abdomen during surgery.

ultrasound and treated with poultices and hot packs. The wounds healed without significant dehiscence. Five days after surgery ceftiofur administration was discontinued and gentamicin sulphate ($1200 \text{ mg i.v. every } 24 \text{ h}$) commenced due to a mild fever (temperature 38.3°C to 39.3°C), thrombophlebitis of the catheterised jugular vein and some incision pain and swelling. The catheter tip was cultured but no growth ensued. The thrombophlebitis was treated with topical dimethyl sulphoxide and hot compresses. Gentamicin sulphate and procaine penicillin were continued for 6 days. At discharge the antibiotic was changed to doxycycline ($2 \text{ g per os every } 12 \text{ h}$) and this continued for a further 14 days.

An echocardiogram performed at 6 days post surgery revealed hyperechoic regions within the myocardium. The previously noted arrhythmia had been abolished and fractional shortening increased (34%). The heart rate had reduced to 72 beats/min and was normal on auscultation.

An examination performed at 5 months post surgery revealed a foal of normal weight (264 kg) and size for its age with normal hindlimb conformation. No significant abnormalities were observed on a full blood count and biochemistry evaluation. Physical and ultrasonographic examination determined the jugular vein thrombosis had resolved. An electrocardiogram revealed no abnormalities, while an echocardiogram showed one small area of hyperechogenicity in the median septum. Fractional shortening was 40%.



Fig 4: Electrocardiogram of the foal at 18 h post surgery showing polymorphic ventricular arrhythmia.

Discussion

Indications for splenectomy include splenomegaly, neoplasia, splenic infarction and abscess, splenic rupture and experimental purposes (Roberts and Groenendyk 1978; Blikslager and Wilson 2006). To our knowledge this is the first report of the successful use of splenectomy in a foal to control haemorrhage from a ruptured splenic capsule.

This foal presented with mild abdominal pain, tachycardia and pale mucous membranes, which is consistent with a previous review of cases of acute haemoperitoneum in horses (Pusterla *et al.* 2005). The most common clinical signs observed in the 19 adult horses with acute haemoperitoneum reviewed were tachycardia, tachypnoea, cold extremities, pale mucous membranes, prolonged capillary refill time and weakness. Colic was frequently noted, although abdominal distension was not often observed (Pusterla *et al.* 2005).

In the case described here the foal had a slightly low packed cell volume and total protein concentration. The mild leucocytosis with neutrophilia was consistent with a stress response. The recovery from the peritoneal cavity of frank blood is consistent with acute intra-abdominal haemorrhage. A packed cell volume of the recovered fluid from the abdominal cavity was not determined, although a value of >5% is considered suggestive of marked haemorrhage (Saxon 1994). The ultrasound finding of large amounts of swirling hyperechoic fluid in the peritoneal space was consistent with significant bleeding into this region. It has been reported that ultrasound evaluation in cases of acute haemoperitoneum may allow the visualisation of splenic haematoma and rupture of the capsule but this was not appreciated in this case. Ultrasonographic evaluation of splenic haematoma may be useful in monitoring cases treated conservatively (Reef 1998; Pusterla *et al.* 2005).

Studies in horses with experimentally produced haemorrhagic shock showed improvements in cardiac output, stroke volume, mean systemic and pulmonary arterial blood pressures, cardiac contractility, urine output and plasma expansion with the use of hypertonic saline compared to isotonic saline (Schmall *et al.* 1990). While there is some concern about the use of a small volume hypertonic saline infusions in the face of uncontrolled haemorrhage, it remains in current clinical use in man. Most studies showing increased blood loss and mortality with the use of hypertonic saline are experimental, with the fluid infusion starting immediately after the induction of uncontrolled haemorrhage. In clinical cases infusions are rarely made until after some time when there is already clinically apparent haemorrhagic shock. There seems to be a trend to decreased morbidity and mortality with the use of hypertonic saline in uncontrolled haemorrhage in human trauma patients and therefore its use in similar cases in the horse may be justified (Rocha-e-Silva and Poli de Figueiredo 2005). We chose to use hypertonic saline in the initial resuscitation of this foal to try to maintain adequate tissue perfusion during major intra-abdominal haemorrhage and haemorrhagic shock.

As soon as blood became available from a universal donor it was administered. Blood remains the fluid of choice in

haemorrhage and provides erythrocytes, leucocytes, platelets, clotting factors, albumin, globulin and other components, most of which are likely to be beneficial in severe haemorrhage. Careful selection of donors and good technique can minimise adverse reactions (Durham 1996).

When presented with a horse with acute haemoperitoneum the need for surgery is not clear. In this case we had not diagnosed the source of the haemorrhage prior to surgery. A study of 19 adult horses with acute haemoperitoneum found that 74% survived when medical treatment was attempted. However, of the 7 horses that had splenic haematoma and capsular tears diagnosed either by ultrasound or at *post mortem* 2 died (Pusterla *et al.* 2005). A report of 3 cases of apparent spontaneous ruptures of the spleen all resulted in fatality (Steiner 1981). This suggests the prognosis may be worse if splenic rupture is diagnosed.

Laparoscopic examination has been reported useful in the diagnosis of splenic bleeding and may have been able to localise the source (Mehl *et al.* 1998; Walmsley 1999). In this case the age and medical condition of the foal precluded standing laparoscopy, and a ventral midline laparotomy was performed to allow rapid evaluation of the abdomen and possible treatment.

A 17th rib resection technique was used as this allowed adequate access to the vasculature of the spleen and has the advantage that it rarely results in penetration of the pleural space. Alternative approaches for splenectomy include the ventral midline and the left paralumbar fossa; however, exposure of the vessels of the spleen is extremely limited with these approaches (Blikslager and Wilson 2006). While a 16th rib resection approach results in penetration of the pleural space, access to the major vessels of the spleen is improved (Rigg *et al.* 1987). Other techniques have been described involving either an intercostal approach or resection of the 18th rib or parts of multiple ribs. Laparoscopic assisted splenectomy has been reported in man; its use in horses is not yet established but could facilitate identification and ligation of splenic vessels (Blikslager and Wilson 2006).

Phenylephrine was administered during anaesthesia to assist in maintaining blood pressure and would have reduced the size of the spleen and facilitated splenectomy, but it is uncertain what effect it would have had on the ongoing haemorrhage through the splenic capsular tear. Prolonged antibiotic use in this foal was prompted by persistent mild fever, development of incisional swelling and pain, thrombophlebitis of the jugular vein and a marked leucocytosis characterised by a neutrophilia with a left shift. Unfortunately culture results were not available to direct antibiotic use. Broad spectrum antibiotics were used and chosen with regard to common sensitivity patterns seen within the hospital. A change to an oral antibiotic was made due to difficulties administering parenteral antibiotics. Pysllium may be of benefit in cases of colitis by promoting mucosal healing through the increased production of short-chain fatty acids within the colon, a source of enterocyte nutrition (Jones 2003). Hyperimmune plasma is commonly used with diarrhoea cases where there may be increased absorption of endotoxin.

The equine spleen has important functions, including storage of and a role in the turnover of erythrocytes and platelets, immunological functions and iron recycling. The equine spleen can store up to 60% of circulating erythrocytes and may be able to release some of this reserve in times of stress. The importance of splenic contraction and its effect on red cell indices during exercise is unclear (Pellegrini Masini *et al.* 2000). The effect of splenectomy on athletic ability in Thoroughbred racehorses has not been determined, although there have been reports of equestrian horses performing athletically after splenectomy (Roy *et al.* 2000; Westerduin *et al.* 2003).

Measurement of cardiac troponin levels in peripheral blood has been used in the diagnosis and management of acute cardiac damage in man (Collinson *et al.* 2001). Cardiac troponins form part of the regulatory mechanism for muscle contraction. Currently there is considerable research effort focused on establishing the significance of cardiac troponin I levels in horses and its usefulness in the diagnosis of cardiac disease (Phillips *et al.* 2003; Slack *et al.* 2005; Begg *et al.* 2006). In man, cardiac troponins are used to detect acute coronary syndromes, although elevated cardiac troponins may occur with a number of other cardiac and noncardiac conditions for example septic shock and renal failure (Ammann *et al.* 2004). Cardiac troponin I levels in 23 mature Thoroughbreds in training were all <0.15 µg/l (Begg *et al.* 2006) and in 52 healthy foals 12–48 h old had a median of 0.14 µg/l with all foals having values <0.51 µg/l (Slack *et al.* 2005). There was reasonable evidence of cardiac injury in the present case, including multifocal ventricular tachycardia and ultrasonographic abnormalities. Increased troponin I values recorded in this case appear to support myocardial damage and the levels dropped with improvements in clinical signs. Ventricular arrhythmias have been reported in other species with splenic lesions, torsions and haematomas (Knapp *et al.* 1993; Marino *et al.* 1994). Treatment of ventricular arrhythmias usually involves the use of anti-inflammatory drugs, lidocaine, procainamide and more recently phenytoin (Wijnberg and Ververs 2004).

Splenic haematoma and rupture is a possible cause of haemoperitoneum in horses and may have fatal consequences. This case suggests that splenectomy should be considered in cases of splenic rupture with severe ongoing blood loss.

Manufacturers' addresses

¹Mila International, Erlanger, Kentucky, USA.

²Plasvacc, Kalbar, Queensland, Australia.

References

- Ammann, P., Pfisterer, M., Fehr, T. and Rickli, H. (2004) Raised cardiac troponins. *Br. med. J.* **328**, 1028-1029.
- Begg, L.M., Hoffmann, K.L. and Begg, A.P. (2006) Serum and plasma cardiac troponin I concentrations in clinically normal Thoroughbreds in training in Australia. *Aust. vet. J.* **84**, 336-337.
- Blikslager, A.T. and Wilson, D.A. (2006) Stomach and spleen. In: *Equine Surgery*, 3rd edn., Eds: J.A. Auer and J.A. Stick, Saunders Elsevier, St Louis. pp 374-386.
- Collinson, P.O., Boa, F.G. and Gaze, D.C. (2001) Measurement of cardiac troponins. *Ann. Clin. Biochem.* **38**, 423-449.
- Durham, A.E. (1996) Blood and plasma transfusion in the horse. *Equine vet. Educ.* **8**, 8-12.
- Finocchio, E.J. (1971) Splenic rupture in a horse. *Vet. Med. small anim. Clin.* **66**, 223.
- Jones, S.L. (2003) Right dorsal colitis. In: *Current Therapy in Equine Medicine*, 5th edn., Ed: N.E. Robinson, W.B. Saunders, Philadelphia. pp 141-143.
- Knapp, D.W., Aronsohn, M.G. and Harpster, N.K. (1993) Cardiac arrhythmias associated with mass lesions of the canine spleen. *J. Am. anim. hosp. Ass.* **29**, 122-128.
- Koterba, A.M., Drummond, W.H. and Kosch, P.C. (1990) *Equine Clinical Neonatology*, Williams and Wilkins, Baltimore.
- Marino, D.J., Matthiesen, D.T., Fox, P.R., Lesser, M.B. and Stamoulis, M.E. (1994) Ventricular arrhythmias in dogs undergoing splenectomy: a prospective study. *Vet. Surg.* **23**, 101-106.
- Mehl, M.L., Ragle, C.A., Mealey, R.H. and Whooten, T.L. (1998) Laparoscopic diagnosis of subcapsular splenic hematoma in a horse. *J. Am. vet. med. Ass.* **213**, 1171-1173, 1133.
- Pellegrini Masini, A., Baragli, P., Tedeschi, D., Lubas, G., Martelli, F., Gavazza, A. and Sighieri, C. (2000) Behaviour of mean erythrocyte volume during submaximal treadmill exercise in the horse. *Comp. Haematol. Int.* **10**, 38-42.
- Phillips, W., Giguere, S., Franklin, R.P., Hernandez, J., Adin, D. and Peloso, J.G. (2003) Cardiac troponin I in pastured and race-training Thoroughbred horses. *J. vet. int. Med.* **17**, 597-599.
- Pusterla, N., Fecteau, M.E., Madigan, J.E., Wilson, W.D. and Magdesian, K.G. (2005) Acute hemoperitoneum in horses: a review of 19 cases (1992-2003). *J. vet. int. Med.* **19**, 344-347.
- Reef, V.B. (1998) *Equine Diagnostic Ultrasound*, W.B. Saunders, Philadelphia.
- Rigg, D.L., Reinertson, E.L. and Buttrick, M.L. (1987) A technique for elective splenectomy of equidae using a transthoracic approach. *Vet. Surg.* **16**, 389-391.
- Roberts, M.C. and Groenendyk, S. (1978) Splenectomy in the horse. *Aust. vet. J.* **54**, 196-197.
- Rocha-e-Silva, M. and Poli de Figueiredo, L.F. (2005) Small volume hypertonic resuscitation of circulatory shock. *Clinics* **60**, 159-172.
- Roy, M.F., Lavoie, J.P., Deschamps, I. and Laverty, S. (2000) Splenic infarction and splenectomy in a jumping horse. *Equine vet. J.* **32**, 174-176.
- Saxon, W.D. (1994) The acute abdomen. *Vet. Clin. N. Am.: Small Anim. Pract.* **24**, 1207-1224.
- Schmall, L.M., Muir, W.W. and Robertson, J.T. (1990) Haemodynamic effects of small volume hypertonic saline in experimentally induced haemorrhagic shock. *Equine vet. J.* **22**, 273-277.
- Slack, J.A., McGuirk, S.M., Erb, H.N., Lien, L., Coombs, D., Semrad, S.D., Riseberg, A., Marques, F., Darien, B., Fallon, L., Burns, P., Murakami, M.A., Apple, F.S. and Peek, S.F. (2005) Biochemical markers of cardiac injury in normal, surviving septic, or nonsurviving septic neonatal foals. *J. vet. int. Med.* **19**, 577-580.
- Steiner, J.V. (1981) Splenic rupture in the horse. *Equine Pract.* **3**, 37-38.
- Walmsley, J.P. (1999) Review of equine laparoscopy and an analysis of 158 laparoscopies in the horse. *Equine vet. J.* **31**, 456-464.
- Westerduin, F.E., Lankveld, D.P., van der Velden, M.A., Back, W. and Sloet van Oldruitenborgh-Oosterbaan, M.M. (2003) Splenectomy in a dressage pony: End of sports career? *Tijdschr Diergeneeskd* **128**, 406-411.
- Wijnberg, I.D. and Ververs, F.F.T. (2004) Phenytoin sodium as a treatment for ventricular dysrhythmia in horses. *J. vet. int. Med.* **18**, 350-353.