Neurological dysfunction in three dogs and one cat following attenuation of intrahepatic portosystemic shunts

Neurological dysfunction is an uncommon complication following extrahepatic portosystemic shunt ligation. Three dogs and one cat are described that developed neurological signs within 21 to 42 hours of attenuation of intrahepatic portosystemic shunts. None of these cases had biochemical evidence of hepatic encephalopathy postoperatively. Two dogs died during management of status epilepticus following aspiration of food. One dog died six months postoperatively. The cat had persistent neurological dysfunction at discharge, but was alive and had recovered most of its neurological function at the time of writing, 37 months after surgery. This report demonstrates the potential for animals with intrahepatic portosystemic shunts to develop postoperative neurological signs and highlights the difficulty of managing such cases. Two dogs had both intrahepatic and extrahepatic portosystemic shunts. Large intestinal malrotation (partial situs inversus) may have been linked to the development of a portosystemic shunt in the remaining dog.

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INTRODUCTION

Central nervous system dysfunction is a well recognised but uncommon postoperative complication following attenuation of extrahepatic portosystemic shunts in dogs (reviewed by Tisdall and others 2000) and cats (van Gundy and others 1990, Levy and others 1995), but often is not associated with biochemical evidence of metabolic encephalopathies. A range of signs from ataxia and behavioural changes to generalised motor seizures and status epilepticus has been reported (Hunt and Hughes 1999, Tisdall and others 2000). However, there are only sporadic reports of a similar syndrome following intrahepatic shunt ligation; for example, in a cat (White and others 1996), a dog (White and others 1998) and, possibly, a further two dogs (Komtebedde and others 1991). In fact, one recent large series identified this syndrome in 11 of 89 dogs following surgery for extrahepatic portosystemic shunts but in none of 32 dogs after surgery for intrahepatic portosystemic shunts (Tisdall and others 2000).

The present report describes four cases in which generalised motor seizures developed following surgical attenuation of intrahepatic portosystemic shunts. None of the cases had biochemical evidence of hepatic encephalopathy or hypoglycaemia, although one dog was mildly hypocalcaemic and hypokalaemic. In two dogs, both single intrahepatic and single extrahepatic portosystemic shunts were identified. The remaining dog had large intestinal malrotation and a single intrahepatic portosystemic shunt.

CASE HISTORIES

Clinical details at presentation and diagnostic findings are summarised in Tables 1 and 2, respectively. Table 3 describes shunt morphology, management and postoperative complications. Table 4 lists biochemical parameters during postoperative neurological complications. Venoportography was performed using sodium iothalamate (Conray 280; Mallinckrodt) at 420 mg iodine/kg.

Case 1
An eight-and-a-half-month-old female oriental cat was presented with a 10-week history of polydipsia, hypersalivation and episodes of ataxia. On examination, the animal had no neurological deficits. Results of serum biochemical analysis were suggestive of hepatic encephalopathy, which responded to medical therapy (Table 1). Serology for feline coronavirus (Feline Virus Unit, University of Glasgow) and *Toxoplasma gondii* (Veterinary Parasitology, Liverpool School of Tropical Medicine) were negative. At surgery, an anomalous branch of the right division of the hepatic portal vein was identified. Mesenteric venoportography confirmed that this was an intrahepatic portosystemic shunt (Table 2). Occlusion of the vessel led to portal hypertension measured intra-

Table 1. Results of serum biochemical analysis and medical therapy at presentation

Case	Breed	Gender	Age (weeks)	Presenting clinical signs	Bile acids (0–7 µmol/litre)*	Ammonia (0–95 µmol/litre)*	Preoperative therapy
1	Oriental cat	Female entire	34	Polydipsia and episodes of hypersalivation and ataxia	176·6	160	15 mg/kg amoxycillin twice daily 0·2 ml/kg lactulose three times daily Low protein diet
2	Irish wolfhound	Male entire	9	Two episodes of head pressing, ataxia and circling	Preprandial: 9·6 2 hours postprandial: 118·5	Not available	Ampicillin Lactulose
3	Hungarian vizsla	Male entire	28	Polydipsia and poor growth	84·7	Not available	Hills I/D diet
4	Shetland sheepdog	Male cryptorchid	28	Polydipsia, poor growth and episodes of ataxia, aggression and hypersalivation	239	406	25 mg/kg ampicillin twice daily 0·4 ml/kg lactlose three times daily Hepatic support diet

*Reference range

operatively, so the vessel was attenuated to 50 per cent of its diameter using 3 metric silk (Mersilk; Ethicon).

Approximately 42 hours postoperatively, the cat exhibited two generalised motor seizures 30 minutes apart. These were controlled with two 1·4 mg/kg intravenous boluses and one enema of diazepam, and 0·2 ml/kg lactulose enemas were administered every four hours (Table 3). The cat became ambulatory by 52 hours postoperatively, at which point vertical nystagmus, head pressing, wandering and lack of response to auditory and visual stimuli were apparent. The animal improved over the following 24 hours, but remained ataxic and blind.

At 79 hours postoperatively, the cat became unresponsive again but there was no biochemical evidence of a metabolic encephalopathy (Table 4). Following the administration of 0·3 to 0·6 mg/kg diazepam intravenously (IV) three times daily, the cat became responsive within 12 hours and continued to improve. The drug was discontinued after five days and the cat was discharged after eight days, when it could hear, but was blind and ataxic. By three weeks postoperatively, vision had returned.

Generalised motor seizures started again seven weeks postoperatively. Phenobarbitone (2·2 to 3·25 mg/kg orally twice daily) reduced the frequency of seizures to once every four to six months.

Mesenteric portography six months postoperatively showed good liver arborisation and no evidence of portosystemic shunting. No metabolic cause for the continued seizure activity could be found. Three years postoperatively, the cat was no longer ataxic, but had persistent dorsal visual field deficits and intermittent seizures.

Case 2

A nine-week-old male Irish wolfhound was presented following two episodes of neurological dysfunction (disorientation, ataxia, head pressing and circling) during the preceding four weeks. Results of biochemical analysis were suggestive of hepatic encephalopathy (Table 1), and ultrasonography identified changes consistent with an intrahepatic portosystemic shunt (Table 2). Operative mesenteric portography revealed a large single intrahepatic shunt arising from the left division of the portal vein (Table 2). The anomalous vessel was ligated using 3·5 metric silk (Mersilk; Ethicon) as it emptied into the left lateral hepatic vein. Situs inversus of the large intestine was noted, although small intestinal, gastric, splenic and hepatic positioning were normal. Postoperative mesenteric venoportography was equivocal for complete shunt ligation.

There was no improvement in the animal's demeanour or growth rate postoperatively. Four weeks following surgery, elevated fasting and postprandial bile acids were suggestive of continuing portosystemic shunting. At eight months of age,

Table 2. Investigation of suspected portosystemic shunt

Case		Ultrasonographic findings	Preoperative portographic findings	Liver biopsy	Shunt type
1		No abnormalities detected	Contrast entering caudal vena cava via right division of portal vein, no hepatic arborisation	Moderate fatty degeneration of hepatocytes	Intrahepatic from right division of portal vein
2		Single anomalous intrahepatic vessel	Contrast entering caudal vena cava via left division of portal vein, no hepatic arborisation	Arteriolar duplication and small portal veins in portal tracts	Intrahepatic from left division of portal vein to left middle and left lateral hepatic veins
3	First surgery	Turbulent flow in caudal vena cava and microhepatica	Non-diagnostic		Extrahepatic portocaval
	Second surgery	Single anomalous intrahepatic vessel	Contrast entering caudal vena cava via central division of portal vein. No contrast passing through extrahepatic shunt identified by ameroid constrictor	Small hepatic lobules, arteriolar duplication and small or absent portal veins in portal tracts	Intrahepatic from central division of portal vein
4		Microhepatica with small vessels, single anomalous vessel queried to be intrahepatic	Contrast arborisation of right lateral liver lobe before filling of the caudal vena cava via right division of the portal vein	Vacuolation and arteriolar duplication in portal tracts, sinusoidal dilation	Extrahepatic left gastric to cava Intrahepatic from right division of the portal vein

Table 3. Surgical treatment and neurological complications

Case	Shunt type	Method of attenuation	Time to first neurological complications	Time to seizures	Treatment	Other complications	Outcome
1	Intrahepatic from right division of portal vein	50 per cent occlusion with silk	42 hours	42 hours	1·4 mg/kg diazepam IV + PR boluses 0·2 ml/kg lactulose PR		Intermittent seizures and doral visual field deficits
2	Intrahepatic from left division of portal vein to left middle and left lateral hepatic veins	100 per cent occlusion with silk	21 hours	29 hours	0·1 mg/kg diazepam IV bolus 2·5 mg/kg phenobarbitone IM bolus 0·17 to 0·5 mg/kg/hour midazolam CRI	Aspiration pneumonia	Died 159 hours after surgery
3	First surgery Extrahepatic portocaval	6·5 mm ameroid constrictor				Hepatic encephalopathy continued	
	Second surgery Intrahepatic from central division of portal vein	50 per cent occlusion	24 hours	64 hours	0·1 to 0·2 mg/kg/minute propofol CRI 5 mg/kg propofol IV bolus	Aspiration pneumonia	Died 92 hours after surgery
4	Extrahepatic left gastric to cava Intrahepatic from right division of portal vein	100 per cent occlusion of both	42 hours	44 hours	8 mg/kg propofol IV bolus 0·1 to 0·2 mg/kg/minute propofol CRI	Aspiration pneumonia	Altered behaviour and central blindness Died six months after surgery following seizures and bleeding

CRI Constant rate infusion, PR Per rectum, IV Intravenous; IM Intramuscular

the dog still showed sporadic signs of hepatic encephalopathy and bile acids were still elevated. At this stage, medical therapy for hepatic encephalopathy was instituted.

A second exploratory surgery was performed at 10 months of age. Operative mesenteric portography showed that portal blood continued to bypass the liver via an intrahepatic portosystemic communication. A catheter was passed from the hepatic portal vein into the posthepatic caudal vena cava via the left middle hepatic vein. Further dissection revealed that the original intrahepatic shunt had emptied into both the left middle and left lateral hepatic veins. The left middle hepatic vein was ligated as it entered the posthepatic vena cava, using 3·5 metric silk (Mersilk; Ethicon).

By 21 hours postoperatively, the dog started to vocalise abnormally and by 29 hours it had developed generalised motor seizures (Table 3). These were partially controlled using 0·1 mg/kg IV boluses of diazepam. However, the dog remained unconscious, with muscle fasciculations and vertical nystagmus. A nasoesophageal tube was placed and medical therapy for hepatic encephalopathy was initiated, although there was no biochemical evidence of a metabolic encephalopathy (Table 4).

Phenobarbitone at 2·5 mg/kg, orally twice daily, was instituted in conjunction with a constant rate infusion (CRI) of 0·17 to 0·5 mg/kg/hour midazolam. Attempts to withdraw the midazolam infusion proved unsatisfactory, with generalised motor seizures starting within five hours. Three days after seizures had started, the dog developed aspiration pneumonia and deteriorated until it died 130 hours after the first seizure. Postmortem examination was not performed, at the owner's request.

Case 3

A seven-month-old male Hungarian vizsla was presented for investigation of poor growth and polydipsia/polyuria. Results of serum biochemical analysis were consistent with hepatic encephalopathy (Table 1). Ultrasonography did not identify anomalous intrahepatic vessels but, instead, a single extrahepatic portosystemic shunt. An extrahepatic portocaval shunt was identified at surgery and was attenuated using a 6·5 mm ameroid constrictor (Research Instruments NW; Corvallis). However, the dog remained polydipsic and there was evidence of continuing portosystemic shunting 11 weeks postoperatively, when postprandial bile acids were elevated. Ultrasonography and mesenteric portography performed at this time identified an intrahepatic portosystemic shunt (Table 2). The caudal vena cava was catheterised through the central division of the portal

Table 4. Serum biochemical parameters during period of postoperative neurological dysfunction

Case		Postoperative interval	Ammonia (0–95 µmol/litre)*	Glucose (4·28–6·94 mmol/litre)*	Total calcium (2·30–3·00 mmol/litre)*	Potassium (3·50–5·80 mmol/litre)*	Urea (1·7–7·4 mmol/litre)*
1		79 hours	53	6·92	3·00	4·07	3·9
2		29 hours	36	4·50	2·31	4·80	2·4
3		24 hours	6	6·38	Not available	4·86	Not available
4	Postoperative seizures	44 hours	33	4·20	2·14†	3·10	3·2
	Terminal seizures	6 months	165	1·50	Not available	3·80	Not available

*Reference range
†Corrected for total protein

vein and the shunting vessel was attenuated to 50 per cent of its diameter as it branched from the central division.

By 24 hours following surgery, the dog had become unresponsive, ataxic and was vocalising abnormally. There was no biochemical evidence of hypoglycaemia or hyperammonaemia (Table 4) and the dog recovered by 29 hours postoperatively. Generalised motor seizures began 64 hours postoperatively, again with no biochemical evidence of a metabolic encephalopathy. Seizures were controlled with a 0·1 mg/kg/minute CRI of propofol (Rapinovet; Schering-Plough Animal Health) (Table 3). A nasogastric tube was placed, but this was removed after the dog regurgitated and aspirated. Attempts to wean the animal off the infusion were unsuccessful and breakthrough seizure activity 80 hours after surgery necessitated a 5 mg/kg IV bolus of propofol and an increase of the CRI to 0·2 mg/kg/minute. Eighty-six hours postoperatively, the dog became anuric and 2 mg/kg frusemide IV was given followed by an infusion of dopamine (0·01 mg/kg/minute). After one hour, urine output had not increased and the propofol infusion was stopped while aggressive fluid therapy was given. Within two hours, seizure activity commenced. The CRI was restarted, but the dog regurgitated and aspirated 92 hours postoperatively. This was rapidly followed by respiratory arrest and death. Postmortem examination was not performed, at the owner's request.

Case 4

A seven-month-old male cryptorchid Shetland sheepdog was referred with a history of poor growth, polydipsia/polyuria and, from five months of age, episodes of depression, ataxia, aggression and hypersalivation. There was biochemical evidence of hepatic encephalopathy (Table 1). Mesenteric venoportography identified an intrahepatic portosystemic shunt from the right division of the hepatic portal vein to the caudal vena cava (Table 2). Surgical isolation of the shunt was abandoned

when the dog had a cardiac arrest, thought to be secondary to severe hypothermia. Resuscitation by open chest cardiac massage via a diaphragmatic myotomy was successful. A second attempt at surgery two days later was abandoned after hypotension developed following haemorrhage from an intra-arterial line concealed under the surgical drapes.

A third attempt at surgery was made at nine months of age. Two portosystemic shunting vessels were found: one intrahepatic shunt from the right division of the portal vein, which could not be isolated from the hepatic parenchyma; and one extrahepatic shunt from the left gastric vein to the prehepatic caudal vena cava. The extrahepatic shunt and the right division of the hepatic portal vein were ligated with 1·5 metric polydioxanone (PDS II; Ethicon). Venoportography following ligation showed good perfusion and arborisation of contrast within the liver.

Hindlimb paddling started 42 hours postoperatively and progressed to status epilepticus within two hours. The dog was both mildly hypokalaemic and hypocalcaemic (after correction of total serum calcium for total protein), although these were not considered to be significant and there was no other biochemical evidence of a metabolic encephalopathy (Table 4). Seizures were controlled with an 8 mg/kg IV bolus and 0·1 to 0·2 mg/kg/minute CRI of propofol (Table 3). These induced a light plane of anaesthesia, but seizure activity started when the CRI was reduced below 0·1 mg/kg/minute. Total parenteral nutrition was instituted. After a further 72 hours, 20 mg/kg phenobarbitone intramuscularly was administered and the propofol infusion was stopped. Phenobarbitone (2 mg/kg IV twice daily) was continued for a further eight days.

A nasogastric tube was placed after parenteral nutrition was stopped, but aspiration pneumonia developed and the tube was removed the following day. Initially, the dog was blind, deaf and could not stand. Ten days after the infusion had been stopped, photomotor responses returned,

although the dog remained blind. By 11 days, the dog could hear and by 12 days the dog was able to stand, although it was ataxic with a hypermetric gait. The animal was discharged from the hospital 23 days after surgery, although it was still blind and ataxic. The owner reported that the dog initially did not respond to commands and urinated and defecated in the house. However, by six months house-training had been successfully re-established and it was no longer ataxic, but remained blind.

Six months postoperatively, the dog was presented to the referring veterinary surgeon in status epilepticus. Seizures stopped after two boluses of 1 mg/kg diazepam IV, but the dog remained unresponsive and was referred to the Royal (Dick) School of Veterinary Studies (R[D]SVS). Frank blood was regurgitated after six hours and profuse nasal and rectal bleeding began 90 minutes later. Activated blood clotting time was increased (four minutes; reference range less than 2·5 minutes) and the dog was thrombocytopenic (manual count 45×10^9/litre). A whole blood transfusion was started. At this point, the dog had elevated serum bile acids (309·7 µmol/litre; reference range 0 to 15 µmol/litre) and ammonia (Table 4) and was hypoglycaemic (Table 4) and hypoalbuminaemic (15 g/litre; reference range 27 to 38 g/litre). The animal became increasingly dyspnoeic and radiography showed an alveolar lung pattern. The dog went into respiratory arrest followed rapidly by cardiac arrest 14 hours after the initial period of status epilepticus. Postmortem examination was not performed, at the owner's request.

DISCUSSION

Neurological dysfunction should be considered a potential early complication following attenuation of intrahepatic portosystemic shunts as well as following attenuation of extrahepatic portosystemic shunts in the dog and cat. In this report, seizures in the early postoperative period were not

associated with hyperammonaemia or hypoglycaemia, although hypoglycaemia and status epilepticus were documented in case 4 as terminal events six months postoperatively. These cases are comparable with those described by Hunt and Hughes (1999) and Tisdall and others (2000), suggesting that a mechanism other than hyperammonaemia-associated hepatic encephalopathy accounts for neurological dysfunction in some cases postoperatively. Some studies suggest that older dogs are predisposed to these neurological sequelae to shunt ligation (Matushek and others 1990, Tisdall and others 2000), but all animals in the present study were less than 12 months of age.

Other reports of cases which survive postoperative seizures describe persistent neurological defects in the majority, ranging from intermittent seizures or persistent visual deficits to blindness, ataxia and behavioural changes (Hardie and others 1990, Matushek and others 1990, Levy and others 1995, Heldmann and others 1999, Hunt and Hughes 1999, Tisdall and others 2000). The prognosis for any animal that experiences seizures following portosystemic shunt ligation must be guarded. Initial management is difficult, with a high mortality rate. Persistent neurological dysfunction of varying severity can be expected in survivors. However, animals with less severe neurological signs and many of those that do survive postoperative seizures, even with persistent neurological deficits, seem to function adequately as pets (Levy and others 1995, Heldmann and others 1999, Hunt and Hughes 1999, Tisdall and others 2000).

The cause of persistent neurological deficits in cases 1 and 4 is unknown, but previous studies have described brain pathology in dogs that have died or been euthansed during postoperative seizures (Hardie and others 1990, Matushek and others 1990). The changes in these previously described cases included selective 'ischaemic' neuronal necrosis with variable glial responses, changes that are consistent with ischaemia/hypoxia, hypoglycaemia or prolonged seizure activity. Previously, the changes identified in these three disease processes have been difficult to separate and have been considered to be part of the same syndrome, resulting from disruption of common neuronal metabolic pathways. However, it is now apparent that they are distinct pathological processes, resulting from separate cellular events (reviewed by Auer and Benviste 1997, Honavar and Meldrum 1997, Auer and Siesjö 1988).

As both ischaemia and hypoglycaemia can induce seizures, the changes described in the present two cases might have been the cause of, rather than the result of, seizure activity. However, hypoglycaemia does not cause cerebellar lesions, possibly because of more efficient energy metabolism in the cerebellar neurons, and there were no obvious ischaemic episodes in these cases.

The distribution of lesions caused by ischaemia and prolonged seizure activity are similar and reflect the sensitivity of neurons to these insults possibly mediated through glutamate receptor activity. Typically, hippocampal neurons are affected, with variable involvement of the neocortex and cerebellum. The persistent deficits in cases 1 and 4 were consistent with pathology in these regions and probably resulted from seizure-induced selective neuronal death.

The factors leading to the terminal events of case 4 are unclear. Results of serum biochemical analysis were consistent with hepatic dysfunction, and abdominocentesis recovered a small volume of ascitic fluid. Portosystemic shunting may have recurred through the original shunting vessels or through acquired extrahepatic shunts. Alternatively, hepatic dysfunction may have resulted from undiagnosed hepatic pathology. Frustratingly, this case had been scheduled for reassessment during the preceding week, but had not been presented at that time. Elevated ammonia may have been significant in the aetiology of seizures but equally may have been secondary to intestinal haemorrhage. Similarly, hypoglycaemia cannot be excluded as a cause of seizures, but may have been secondary to the prolonged period of status epilepticus. This dog was presented with a bleeding disorder and seizures may have occurred secondarily to a cerebral bleed.

Propofol CRIs have been described for the management of seizures following portosystemic shunt ligation at rates of 0·1 to 0·25 mg/kg/minute (Heldmann and others 1999). These are said to have advantages over other anticonvulsant therapies, including the ability to titrate to effect the infusion rate to reduce sedative effects, making patient care easier. However, in both cases receiving propofol CRIs in this study (cases 3 and 4), the animals remained unconscious, necessitating intensive nursing care. In fact, both of these cases and case 2, which received a CRI of midazolam, aspirated food, leading to deterioration of their condition and death of cases 2 and 3. Midazolam and propofol suppress swallowing and gag reflexes, while propofol also reduces gastro-oesophageal sphincter pressure (Waterman and Hashim 1992). These features may have contributed to regurgitation and aspiration following nasal tube feeding.

Given the profound sedative effects seen with both types of infusion in these cases, the authors cannot recommend nasoesophageal or nasogastric feeding. Jejunostomy enteral nutrition or total parenteral nutrition offer better alternatives for nutritional support in such cases.

The pre-emptive use of phenobarbitone has been advocated to reduce the incidence of neurological complications following portosystemic shunt ligation, but has not been fully evaluated (Tisdall and others 2000). Falling levels of circulating endogenous benzodiazepine ligands have been implicated in the pathogenesis of neurological complications. These metabolites are thought to originate in the intestinal tract, being metabolised by the liver before they can enter the systemic circulation. During portosystemic shunting, the metabolites may bypass the liver and enter the systemic circulation, where they could

contribute to hepatic encephalopathy by depressing central nervous system function. One hypothesis suggests that the brain becomes dependent on the endogenous ligands and neurological dysfunction occurs after systemic levels fall following shunt ligation (Aronson and others 1997). If this is correct, benzodiazepines may be preferable to phenobarbitone for prophylactic treatment to prevent postoperative neurological complications following shunt ligation.

Three of the cases in this series had atypical shunt morphologies. Cases 3 and 4 had a combination of both single congenital intrahepatic and single congenital extrahepatic portosystemic shunts. In case 3, ultrasonography identified an extrahepatic portosystemic shunt that was attenuated using an ameroid constrictor. However, continuance of clinical signs prompted repeat ultrasonographic investigation, identifying an additional intrahepatic shunt. This shunt was probably congenital and may have been missed during the original ultrasonographic examination because the extrahepatic shunt was easily identified before a survey of the liver had been performed.

There are several reports of dual congenital extrahepatic shunts (Johnson and others 1987, Lawrence and others 1992, Wilson and others 1997, Meyer and others 1999). There is also one report of patent ductus venosus associated with multiple small extrahepatic portosystemic shunts, which were thought to be congenital (Ewing and others 1974). However, there are no reports of single congenital extrahepatic shunts in combination with an intrahepatic shunt.

Case 2 was unusual because situs inversus of the large intestine was identified. Situs inversus totalis has been associated with multiple vascular anomalies, including malformation of the hepatic vena cava and portosystemic shunting in humans (Shiomi and others 1993) and in dogs (Sadanaga and Schulman 1987, Hunt and others 1998). The present case may represent a variation of this, with partial situs inversus associated with abnormal abdominal vasculature, providing an unusual cause of portosystemic shunting.

Conclusions
Postoperative neurological dysfunction must be considered following surgical correction of intrahepatic as well as extrahepatic portosystemic shunts in the dog and cat. This report supports the conclusions of Hardie and others (1990) and Tisdall and others (2000) that close postoperative monitoring of portosystemic shunt cases should extend for at least 72 hours. Different protocols for the medical management of post-ligation seizures have been reported. The ideal protocol and role of prophylactic anticonvulsant therapy await further study. The authors advise against nasoesophageal or nasogastric feeding in animals treated with CRI of propofol or midazolam due to the risk of aspiration. Aspiration was a complication in all three dogs in this study and was considered to be the cause of death in two of them. Animals that survive post-ligation seizures may have long-term neurological problems. Extrahepatic and intrahepatic portosystemic shunts may present together, highlighting the need for careful preoperative and intraoperative evaluation.

Acknowledgements

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