Surgical treatment of intrahepatic portosystemic shunts in 45 dogs

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The surgical attenuation of an intrahepatic portosystemic shunt in 45 dogs is described. Twenty-nine (64 per cent) had left divisional shunts consistent with a patent ductus venosus (PDV), 15 (33 per cent) had central divisional shunts and one had a right divisional shunt. In the dogs with a PDV, the shunt vessel could be most easily manipulated at a posthepatic site, whereas in those with central and right divisional shunts the manipulation could be more easily made intrahepatically but sometimes involved demanding intravascular surgical techniques. Eight dogs (18 per cent) died during the surgery or shortly afterwards. Of the 37 dogs surviving longer term, 28 (76 per cent) became clinically normal and required no medication or diet control. In a further three animals the shunt was ligated completely only during a second surgical procedure. The remaining six dogs were euthanased because of clinical signs of encephalopathy which were either surgically or medically uncontrollable.

CONGENITAL intrahepatic portosystemic shunts in dogs may be due either to the failure of the ductus venosus to close after birth, or to the presence of other intraparenchymal communications between the portal vein and the hepatic vein or the caudal vena cava (Payne and others 1990, Whiting and Peterson 1993). Intrahepatic shunts can be categorised according the hepatic division through which they pass before entering the caudal vena cava (Martin and Payne 1990, Payne and others 1990, White and others 1996a, Lamb and White 1998). Right divisional shunts pass through either the caudate process of the caudate lobe or the right lateral lobe of the liver before entering the vena cava; central divisional shunts pass through either the right medial or quadrate lobes before entering the vena cava, and left divisional shunts traverse either the papillary process of the caudate lobe, the left lateral lobe or the medial lobe before entering the vena cava (Sleight and Thomford 1970, Swalec Tobias and Rawlings 1996, White and others 1996a). The patent ductus venosus (PDV) has been defined as a left divisional shunt which drains into the left hepatic vein before entering the caudal vena cava (Payne and others 1990). A diagram of the normal portal supply to, and the venous drainage from, the liver of the dog is shown in Fig 1.

A small number of surgical procedures have been described for the management of intrahepatic shunts. Extravascular techniques include prehepatic dissection and attenuation of the portal tributory entering the shunt (Breznock and others 1983, Swalec and Smeak 1990), posthepatic attenuation of the left hepatic vein (Martin and others 1986, Martin and Payne 1990, Komtebedde and others 1991), posthepatic direct shunt ligation (Breznock and others 1983), intrahepatic direct shunt dissection and ligation with the aid of an ultrasonic aspirator (Swalec Tobias and others 1996), and the blind placement of the attenuation ligature around the shunt with ultrasonographic guidance (Wrigley and others 1983). Intravascular techniques include the placement of embolisation coils (Partington and others 1993), intraluminal closure of the shunt via the thoracic vena cava (Breznock and others 1983, Rawlings and Wilson 1983, White and others 1996b) and transportal closure of the shunt via portal venotomy (Hunt and others 1996).

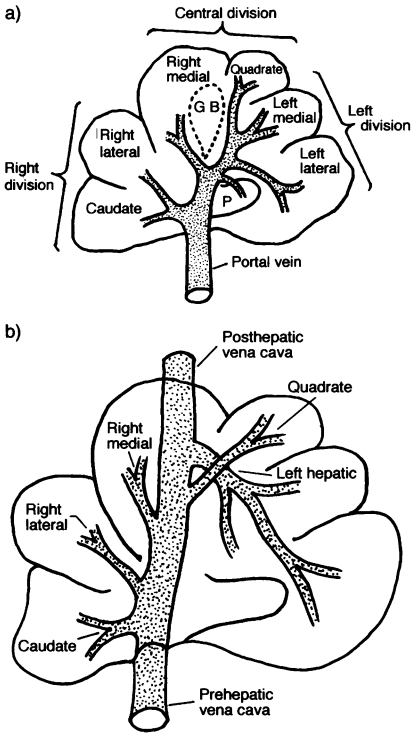

FIG 1: a) Diagram of the lobar and divisional anatomy of the liver of the dog. The normal pattern of branching of the portal vein is shown (adapted from Sleight and Thomford 1970). GB Gall bladder fossa, P Papillary process of the caudate lobe. b) Diagram of the normal pattern of venous drainage of the liver of the dog

This paper describes the surgical management of 45 dogs with intrahepatic portosystemic shunts, and discusses the shunt morphology, the surgical technique and perioperative complications, and the long-term results.

Materials and methods

The surgical records of 45 dogs treated consecutively for the attenuation of an intrahepatic portosystemic shunt between April 1991 and October 1996 were reviewed. Each dog was given a full clinical examination and blood samples were collected for routine haematological and biochemical evaluation. The diagnosis of portosystemic shunt was made on the basis of clinical signs, pre- and postprandial serum bile acid concentrations, abdominal ultrasonography and mesenteric portography. Portal scintigraphy studies were made of all the dogs examined from October 1995 onwards. The case details are given in Table 1.

Before the operation the dogs were treated with ampicillin (Amfipen; Mycofarm UK) at 20 mg/kg three times a day, oral lactulose (Duphalac; Duphar Laboratories) at 3 to 10 ml three times a day, and were fed a low protein diet.

The anaesthetic protocol used was similar to that described by White and others (1996a,b). Ampicillin (Penbritin Veterinary Injectable; SmithKline Beecham Animal Health) 20 mg/kg was administered perioperatively by slow intravenous injection. The mesenteric venous pressure (P1) of each dog was measured and mesenteric portography (PV1) was performed with a C-Arm fluoroscope (Phillips BV22; Phillips Medical Systems) as described by White and others (1996b). The angiographic findings were recorded on video tape. Portography identified an intrahepatic portosystemic shunt passing through either the left, right, or central division of the liver of each dog (Lamb and White 1998).

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TABLE 1: Breed, sex, age and weight of 45 dogs with intrahepatic portosystemic shunts and the hepatic division in which the shunt was sited

Dog	Breed	Sex	Age (months)	Weight (kg)	Hepatic division
1	Golden retriever	M	6	16·5	Left
2	Golden retriever	M	8	21·0	Left
3	Golden retriever	F	5	11·5	Left
4	Golden retriever	F	3	7·7	Left
5	Golden retriever	M	3	6·6	Central
6	Golden retriever	M	5	10·9	Central
7	Golden retriever	M	5	10·2	Central
8	Golden retriever	M	3	10·5	Central
9	Labrador retriever	M	6	16·0	Left
10	Labrador retriever	F	15	20·5	Left
11	Labrador retriever	M	4	13·8	Left
12	Labrador retriever	M	2	6·9	Central
13	Labrador retriever	M	3	8·0	Central
14	Labrador retriever	M	13	8·4	Right
15	Irish wolfhound	M	11 weeks	7·5	Left
16	Irish wolfhound	M	5	25·0	Left
17	Irish wolfhound	M	14 weeks	15·0	Left
18	Irish wolfhound	M	3	17·0	Left
19	Irish wolfhound	F	3	11·5	Left
20	Old English sheepdog	M	18	22·0	Central
21	Old English sheepdog	M	3	10·2	Central
22	Old English sheepdog	M	3	7·5	Central
23	Old English sheepdog	M	10	16·0	Central
24	Deerhound	F	10	22·0	Left
25	Deerhound	M	3	10·0	Left
26	Deerhound	F	3	8·0	Left
27	Border collie	F	6	8·2	Left
28	Border collie	F	5	7·7	Central
29	Borzoi	M	6	27·0	Left
30	English springer spaniel	F	14	16·0	Left
31	German shepherd dog	F	5	18·7	Left
32	German shepherd dog	M	18	25·0	Left
33	Australian cattle dog	M	4	14·4	Central
34	Basset hound	M	14	9·0	Left
35	Boxer	M	15 weeks	6·8	Left
36	Cairn terrier	M	24	7·5	Left
37	Flat coated retriever	F	24	28·5	Left
38	Irish setter	F	6	10·4	Left
39	Italian spinone	M	18	32·2	Left
40	Miniature poodle	F	10	3·5	Central
41	Pyrennean mountain dog	F	3	9·0	Left
42	Samoyed	M	7	20·0	Central
43	Siberian husky	M	3	8·2	Central
44	Staffordshire bull terrier	M	6	12·7	Left
45	Weimaraner	F	3	11·0	Left

Left divisional shunts (dogs 1-4, 9-11, 15-19, 24-27, 29-32, 34-39, 41, 44 and 45)

A diagram of the vascular anatomy of the left divisional shunts is shown in Fig 2. The left lateral and medial lobes of the liver were mobilised by making incisions in the falciform and left triangular ligaments, exposing the posthepatic caudal vena cava and a voluminous left hepatic vein. The exposure of the left hepatic vein was increased by gentle retraction of the left medial and lateral lobes of the liver towards the midline. In most dogs, an anomalous vessel was clearly visible passing between the left lateral lobe and the papillary process of the caudate liver lobe before entering the left hepatic vein. In some dogs, the vessel passed through the left lateral liver lobe before entering the left hepatic vein. Ligature-passing forceps (90° Waterstons; GU Manufacturing) were used to dissect bluntly around the shunt vessel close to its entrance into the left hepatic vein (Fig 3). On initial inspection, the correct tissue plane was not always clearly visible, but with patience and careful blunt dissection, the vessel was encircled without rupture in all of the dogs.

Central divisional shunts

A diagram of the most common vascular anatomy of the central divisional shunts is shown in Fig 4 (excluding dogs 23, 28, 40 and 42).

Dissection of the shunt within the liver (dogs 5, 20, 23, 42 and 43). – In five dogs attempts were made to dissect around the shunt

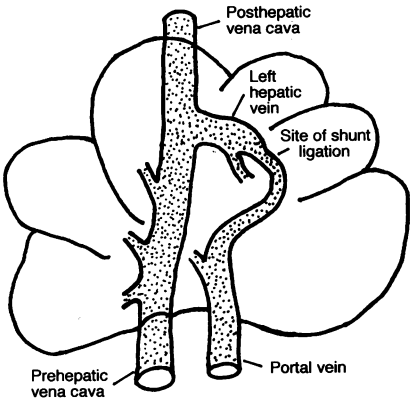

FIG 2: Diagram of a typical left divisional shunt entering the left hepatic vein. The site of shunt ligation is shown

at its junction with the hepatic vena cava. The right lateral and medial lobes of the liver were mobilised by making incisions in the falciform, the right triangular and the hepatorenal ligaments. These incisions exposed the lateral walls of the caudal vena cava within the liver (the hepatic vena cava) and a voluminous shunt vessel entering a dilated hepatic vena cava or central hepatic vein. The exposure of the shunt was increased by gentle retraction of the right medial and lateral lobes of the liver towards the midline. The 90° ligature-passing forceps were used to dissect bluntly around the shunt vessel close to its entrance into either the hepatic vena cava (dogs 5, 20 and 43) or the central hepatic vein (dogs 23 and 42). A diagram of the central divisional shunt entering the central hepatic vein is shown in Fig 5.

In two dogs, the shunt vessel passed between the right and left medial lobes before entering the hepatic vena cava within the right medial lobe of the liver (dog 40) or traversing the diaphragm before entering the intrathoracic vena cava (dog 28). In dog 40, the ligature-passing forceps were used to dissect the shunt vessel free from surrounding parenchyma and to encircle it between the right and left medial lobes of the liver. In dog 28, the shunt was encircled extrahepatically at a site between where it left the liver parenchyma and where it passed through the ventral diaphragm to join the vena cava.

Intravascular closure of the shunt via a posthepatic caval venotomy (dogs 6, 7, 8, 12, 13, 21, 22 and 33). – In these dogs the shunt was closed via a posthepatic caval venotomy during total hepatic vascular occlusion similar to that described by White and others (1996b). In dogs 6 and 7 the opening of the shunt into the hepatic vena cava was partially closed with a continuous 5-0 polypropylene (Prolene; Ethicon) suture.

In the remaining dogs, the shunt opening into the hepatic vena cava was completely closed. In these animals the caval venotomy

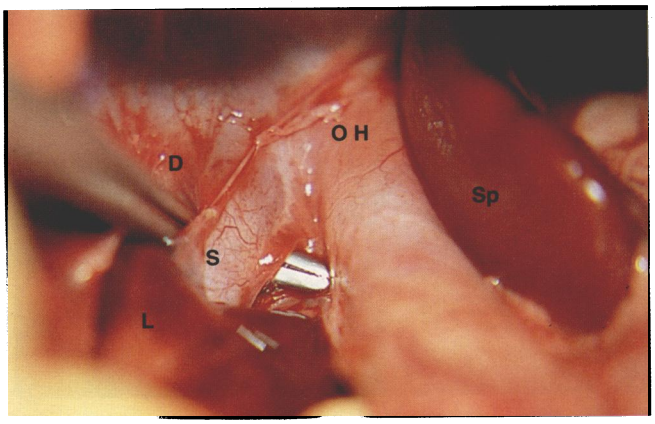

FIG 3: Intraoperative view of a dog with a patent ductus venosus showing the ligature-passing forceps passed around the shunt vessel close to its entrance into the left hepatic vein. D Diaphragm, L liver, OH Oesophageal hiatus, S Shunt, Sp Spleen

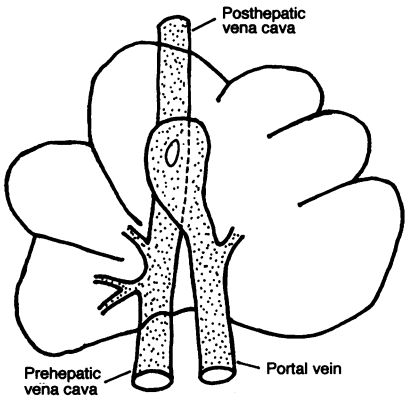

FIG 4: Diagram of the most typical central divisional shunt. The shunt takes a straight course into the right medial lobe and has a dilatation at its communication with the hepatic vena cava. The communication between the two vessels is via a foramen

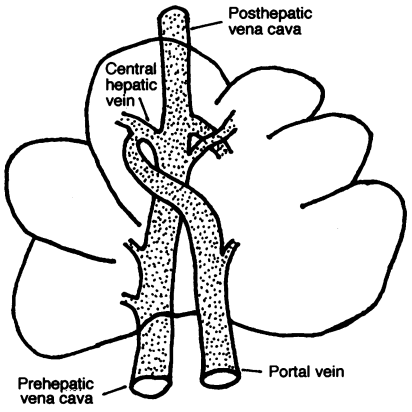

FIG 5: Diagram of an atypical central divisional shunt which enters a central hepatic vein before it enters the vena cava (dogs 23 and 42)

procedure was preceded by the creation of a vascular graft anastomosis between the portal vein and the prehepatic vena cava to prevent the development of life-threatening portal hypertension (Fig 6). In dog 8 the vascular graft was fashioned from polytetrafluoroethylene (GoreTex: W. L. Gore and Associates), but in the others, an autologous jugular vein graft was used in a similar manner to that described by White and others (1996b). Approximately 10 minutes after the re-establishment of hepatic blood flow, and with unobstructed flow in the vascular graft, the mesenteric venous pressure was measured (P3). The vascular graft was then manually occluded and the mesenteric venous pressure was re-measured (P2).

Right divisional shunt (dog 14)

A diagram of the vascular anatomy of the right divisional shunt is shown in Fig 7. The angiographic findings indicated that the shunt vessel passed as a broad loop through the right lateral lobe of the liver. The right medial and lateral lobes of the liver were mobilised by making incisions in the falciform and right triangular ligaments. None of the vessel was visible and it was completely surrounded by liver parenchyma. Palpation of this lobe revealed a softness over the site of the vessel. The shunt was dissected using ligature-passing forceps which were passed through the liver parenchyma and around the vessel.

In the dogs with left and right divisional shunts, and in the dogs with extravascularly dissected central divisional shunts in which the shunt was successfully encircled (dogs 23, 28, 40 and 42), a ligature of 2-0 polypropylene (Prolene; Ethicon) was passed around the shunt vessel after the dissection was completed (Fig 8). A snare was fashioned around the shunt as described by White and others (1996a) so that the vessel could be occluded atraumatically. The mesenteric venous pressure was measured (P2) and a further portogram (PV2) obtained. This was used to confirm that the shunt had been accurately located and that there were no other portosystemic communications. It was also used to assess the opacification of the hepatic parenchyma and the patency of the intrahepatic portal vessels. The splanchnic viscera were examined for venous stasis and congestion. The snare was released and the polypropylene ligature replaced with two or three 4 silk (Mersilk; Ethicon) ligatures. In all the dogs, the silk ligatures were tied to partially attenuate the shunt. The mesenteric venous pressure (P3) was again measured and the splanchnic viscera re-examined for venous stasis and congestion.

A hepatic biopsy was obtained from each dog. The surgical incisions were repaired routinely and intravenous fluid therapy was continued for 36 hours after surgery when all the dogs which were alive were eating and drinking. Oral lactulose (3 to 10 ml three times a day), oral ampicillin (20 mg/kg three times a day) and a low protein diet were continued for the first four weeks after the operation.

The dogs were re-examined one month after the operation, when blood haematological and biochemical measurements were made (including pre- and postprandial concentrations of serum bile acids) and abdominal ultrasonographic and, in individuals examined after October 1995, portal scintigraphic investigations were made. Depending on the clinical and laboratory findings, some of the dogs were re-evaluated later. The owners of the dogs were contacted independently for long-term assessment of the surgery. The mean follow-up period was 16 months (range, one to 66 months). In six of the dogs (7, 22, 23, 25, 27 and 28), the clinical and laboratory findings suggested that the shunt needed further attenuation, and a second portovenogram study and further attempts to attenuate the shunt vessel were made.

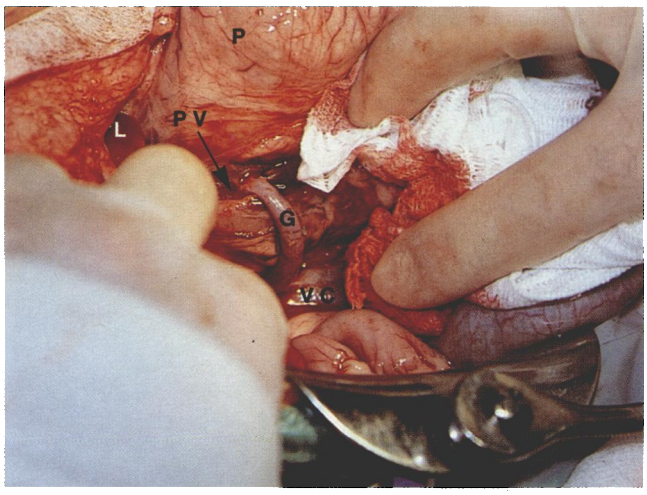

FIG 6: Intraoperative view of the right lateroventral cranial abdomen of dog 21 showing the completed jugular vein graft anastomosis between the portal vein (ventrally) and the caudal vena cava (dorsally). The cranial abdomen is to the left and the caudal abdomen to the right . G Graft, L Liver, P Pancreas, PV Portal vein, VC Vena cava

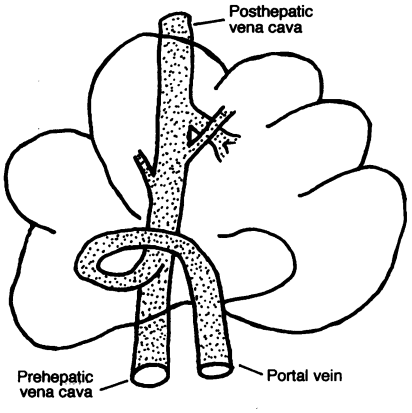

FIG 7: Diagram of a right divisional shunt. The shunt vessel passes as a broad loop through the right lateral lobe of the liver (dog 14)

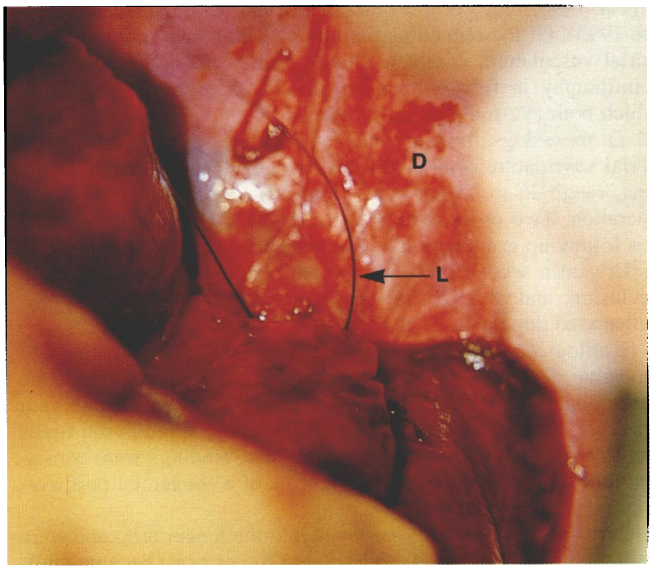

FIG 8: Intraoperative view of a dog with a patent ductus venosus showing the ligature of 2-0 polypropylene passed around the shunt vessel. D Diaphragm, L Ligature

The results of the blood analysis and the scintigraphic results were analysed and their significance assessed by the paired Student's *t* test. When several sets of results were available for a dog, the most recent results were used. In the six dogs in which a second attenuation procedure was attempted, the results obtained just before the second surgical procedure were used in the statistical analysis. The period over which laboratory measurements were assessed ranged from one month to 24 months (mean five months). A period of one month between the operation and an assessment was considered the minimum interval for the inclusion of the assessment in the statistical analysis.

Results

The breed distribution of the dogs is given in Table 1. The mean age of the dogs was seven months (range two months to two years) and their mean weight was 13·7 kg (range 3·5 to 32·2 kg). The sex distribution was 2:1 male:female. The results of the preoperative blood tests are shown in Table 2. The hepatic division through which each intrahepatic portosystemic shunt was located are listed in Table 1. Of the 45 dogs, 29 (64 per cent) had left divisional shunts consistent with a PDV, 15 (33 per cent) had central divisional shunts, and one had a right divisional shunt.

TABLE 2: Mean (se) concentrations of blood components in 45 dogs with intrahepatic portosystemic shunts before surgery

Parameter (units)	Normal range	Before surgery	% outside normal range
Resting bile acids (mmol/litre)	0-15	112 (18) (n = 40)	92·5% above
Postprandial bile acids (mmol/litre)	0-30	233 (25) (n = 22)	100% above
Resting ammonia (μmol/litre)	0-60	265 (22) (n = 31)	97% above
Postprandial ammonia (μmol/litre)	0-60	302 (49) (n = 10)	100% above
Alkaline phosphatase (U/litre)	10-300	671 (80) (n = 40)	72·5% above
Alanine aminotransferase (U/litre)	20-100	191 (37) (n = 40)	45% above
Albumin (g/litre)	24-43	24 (1) (n = 40)	30% below
Total protein (g/litre)	55-80	49 (1) (n = 40)	77·5% below
Urea (mmol/litre)	4·0-11·0	2·7 (0·2) (n = 40)	85% below
Mean cell volume (fl)	65-75	62 (1) (n = 36)	69% below
Red cell number (x 10^{12}/litre)	5·4-8·0	5·7 (0·2) (n = 36)	47% below

TABLE 3: Surgical time, pre- and postoperative shunt indices (SI) and portovenogram findings in 45 dogs with intrahepatic portosystemic shunts

Dog	Surgical time (minutes)	Pre-operative SI (%)	Post-operative SI (%)	PV1	PV2
1	50	NP	NP	V(–) S(+) O(–)	V(+) B(+) O(+)
2	45	71	–	V(–) S(–) O(–)	NP
3	60	67	21 (1 m)	V(+) B(+) O(+)	V(+) B(+) O(+)
4	45	67	25 (6 m)	V(–) S(+) O(–)	V(+) B(+) O(+)
5	45	NP	–	V(+) B(–) O(–)	NP
6	130	NP	NP	NA	NA
7	120	41	27 (3 m)	V(–) S(+) O(–)	NP
8	145	48	–	V(–) S(–) O(–)	NP
9	120	NP	NP	V(–) S(–) O(–)	V(+) B(+) O(+)
10	70	NP	NP	V(–) S(+) O(+)	V(+) B(+) O(+)
11	45	51	10 (3 m)	V(–) S(+) O(–)	V(+) B(–) O(+)
12	180	39	–	V(+) B(+) O(–)	NP
13	180	67	12 (3 m)	V(–) S(+) O(–)	NP
14	90	NP	8 (6 m)	V(–) S(+) O(–)	V(+) B(–) O(+)
15	50	NP	NP	V(–) S(–) O(–)	V(–) S(+) O(–)
16	60	76	40 (1 m)	V(–) S(–) O(–)	V(–) S(+) O(–)
17	40	49	23 (3 m)	V(–) S(+) O(–)	V(+) B(–) O(+)
18	50	NP	7 (1 m)	V(+) B(+) O(+)	V(+) B(+) O(+)
19	45	NP	6 (1 m)	V(+) B(+) O(+)	V(+) B(+) O(+)
20	90	NP	–	V(–) S(–) O(–)	NP
21	190	59	18 (1 m)	V(+) B(–) O(+)	V(+) B(–) O(+)
22	180	65	16 (1 m)	V(+) B(–) O(–)	NP
23	135	NP	NP	V(+) B(+) O(+)	NP
24	60	53	17 (6 m)	V(–) S(–) O(–)	V(+) B(–) O(+)
25	55	48	30 (3 m)	V(–) S(+) O(–)	V(–) S(+) O(+)
26	60	64	26 (3 m)	V(–) S(+) O(–)	V(–) S(+) O(+)
27	60	NP	31 (3 m)	NA	NA
28	40	NP	NP	V(–) S(–) O(–)	V(+) B(–) O(+)
29	90	NP	NP	NA	NA
30	90	NP	NP	NA	NA
31	90	NP	NP	V(–) S(+) O(–)	NA
32	80	NP	NP	NA	NA
33	120	64	–	V(–) S(–) O(–)	NP
34	65	NP	21 (3 m)	V(+) B(+) O(–)	V(+) B(+) O(+)
35	55	NP	NP	V(–) S(+) O(–)	V(+) B(–) O(+)
36	70	NP	NP	V(+) B(+) O(+)	V(+) B(+) O(+)
37	80	58	7 (6 m)	V(+) B(–) O(+)	V(+) B(+) O(+)
38	45	NP	NP	V(+) B(–) O(–)	V(+) B(+) O(+)
39	90	NP	–	V(–) S(+) O(–)	V(+) B(+) O(+)
40	45	NP	NP	NA	NA
41	55	53	14 (1 m)	V(+) B(+) O(+)	V(+) B(+) O(+)
42	45	NP	NP	NA	NA
43	90	61	–	NA	NP
44	50	NP	19 (6 m)	V(–) S(+) O(–)	V(+) B(+) O(+)
45	60	NP	45 (1 m)	V(–) S(+) O(–)	V(–) S(+) O(–)

B Vessel branching, m month, NA Not available, NP Not performed, – Not performed (dog dead), O Parenchymal opacification, PV1 Initial mesenteric portovenogram, PV2 Full shunt ligation mesenteric portovenogram, S Intrahepatic portal vessel stump, V Intrahepatic portal vessels, (+) present, (–) not present

The clinical outcomes for the 45 dogs are listed in Table 5. At the time of writing, 21 dogs were known to be alive, clinically normal and requiring no medication. The average follow-up period for these 21 dogs was 21 months (range three to 66 months). Eight other dogs were clinically normal and requiring no medication before being lost to follow-up after an average of 10 months (range two to 38 months). Sixteen of the dogs had died, eight of them within 48 hours of the surgical procedure. Among these eight was dog 2 which had a left divisional shunt and in which samples of urine and faeces obtained shortly before the operation had been shown to contain *Salmonella typhimurium*. As a result of this infection, the owners requested that the dog should be euthanased before it recovered from anaesthesia. Three dogs (7, 22 and 23) initially became clinically normal and required no medication, but all three developed medically unresponsive encephalopathic signs and were euthanased. Two dogs (21 and 32) died from unrelated causes approximately three to five months after the operation. Both dogs became clinically normal and required no medication after the surgery; dog 32 died as a result of gastric dilatation/torsion, and dog 21 was euthanased as a result of medically unresponsive renal failure.

The shunt vessel was successfully encircled and partially ligated in all of the dogs with a shunt passing through either the left or

TABLE 4: Mean (se) biochemical and haematological measurements made before and after surgery in dogs with an intrahepatic portosystemic shunt

Measurement	Mean before surgery	Mean after surgery	Significance
Resting bile acids (mmol/litre)	110 (21) (n = 25)	46 (12) (n = 25)	P<0·01
Postprandial bile acids (mmol/litre)	292 (47) (n = 11)	110 (30) (n = 11)	P<0·01
Resting ammonia (μmol/litre)	260 (43) (n = 11)	77 (18) (n = 11)	P<0·01
Alkaline phosphatase (U/litre)	674 (60) (n = 20)	313 (40) (n = 20)	P<0·01
Alanine aminotransferase (U/litre)	158 (51) (n = 20)	48 (6) (n = 20)	P<0·05
Albumin (g/litre)	24 (1) (n = 21)	27 (1) (n = 21)	NS
Total protein (g/litre)	49 (2) (n = 21)	57 (3) (n = 21)	P<0·05
Urea (mmol/litre)	3·0 (0·3) (n = 20)	3·6 (0·4) (n = 20)	NS
Mean cell volume (fl)	61 (1) (n = 15)	64 (1) (n = 15)	NS
Red cell number (x 10^{12}/litre)	5·5 (0·4) (n = 15)	6·7 (0·3) (n = 15)	P<0·01

NS Not significant

right hepatic division. In three of the dogs with a central divisional shunt (dogs 5, 20 and 43), the blind dissection of the medial wall of the shunt within the liver resulted in its rupture and the attenuation procedure failed. It was impossible to close the tear and all three dogs died.

The times taken to complete the surgical procedure are listed in Table 3. The average time taken to complete the surgery in dogs with left divisional shunts was 63 minutes (range 40 to 120 minutes), in dogs with central divisional shunts it was 116 minutes (range 40 to 190 minutes) and in dog 14, which had a right divisional shunt, the procedure took 90 minutes.

The mean initial mesenteric pressure measured before the shunt was manipulated (n = 45) was 1 cm H_2O (range 0 to 4 cm). The mean increase in mesenteric venous pressure after complete shunt occlusion (n = 38) was 41 cm H_2O (range 18 to 55 cm). The mean increase in pressure after partial shunt attenuation (n = 35) was 13 cm H_2O (range 6 to 25 cm).

The radiographic appearance of the hepatic portal vessels are summarised in Table 3. Of the 37 dogs in which an initial mesenteric portovenogram (PV1) was taken 13 (35 per cent) showed the presence of an intrahepatic portal vasculature and only nine of these (24 per cent) showed hepatic parenchymal opacification. Of the 24 dogs with no apparent intrahepatic portal vasculature, 15 (62·5 per cent) had an intrahepatic termination of the portal vessel which was described as looking like a 'stump'. Of the 26 dogs in which a fully ligated shunt portovenogram (PV2) was taken, 21 (81 per cent) revealed an intrahepatic portal vasculature and 23 (88 per cent) showed hepatic parenchymal opacification. Of the 17 dogs with no intrahepatic portal vasculature on PV1 in which the results of both PV1 and PV2 were available, 12 (71 per cent) revealed an intrahepatic portal vasculature on PV2. Similarly, of the 18 dogs with no hepatic parenchymal opacification on PV1, 15 (83 per cent) showed opacification on PV2.

The result of the scintigraphic studies are also shown in Table 3. The mean preoperative shunt index (SI) for the 19 dogs in which it was measured was 58 per cent (range 39 to 76 per cent). Previous studies have shown that normal values for SI on the same equipment are up to 15 per cent. The mean postoperative SI for the 21 dogs in which it was measured was 20 per cent (range 6 to 45 per cent). In the 14 dogs in which both preoperative and postoperative SI results were available, there was a significant (P<0·01) reduction in SI after the operation. The results of the postoperative blood test and their statistical significance are listed in Table 4.

There were 20 dogs in which both PV1 findings and postoperative SI values were recorded. Of the 12 dogs in this group which appeared to have no intrahepatic portal vasculature, complete shunt occlusion was demonstrated in three of them by virtue of the fact that they had an SI within the normal range (Swalec and Smeak 1990, Van Vechten and others 1994, Komtebedde and others 1995). Of the remaining eight dogs which had an intrahepatic portal vasculature, complete shunt closure was demonstrated by scintigraphy in four of them. Similarly, there were 17 dogs in which both PV2 findings and postoperative SI values were recorded. Of these dogs, the four which appeared to have no intrahepatic portal vasculature continued to have high SI values (mean 35 per cent, range 26 to 45 per cent) from one to six months after the operation. Two of these dogs (26 and 45) were clinically normal at a follow-up examination five months after the operation; dog 25 underwent a second procedure which resulted in complete shunt occlusion, and dog 16 showed persistent clinical signs and was euthanased three months after the operation.

Histological examination of the liver biopsies revealed mild to moderate degenerative changes (atrophy and vacuolation) of the centrilobular hepatocytes, inconspicuous or absent portal vein branches, arteriolar and bile duct proliferation, small portal triads and fibrosis around the central veins. These findings were consistent and supported the clinical diagnosis of a congenital portosystemic shunt in each of the dogs.

Thirty seven of the dogs survived into the longer term and were available for follow-up. Twenty eight (76 per cent) became clinically normal and required no medication or diet control. In seven of these dogs scintigraphic studies showed that the partial ligation of the shunt had induced full ligation of the vessel by one to six months after the operation. The other nine dogs showed either persistent or renewed encephalopathic signs, or their laboratory data were consistent with continued portal shunting. A second attenuation procedure was attempted in six of them. Three (dogs 7, 22 and 23) were euthanased during the operation at the request of their owners as a result of the failure to ligate the shunt, the presence of high mesenteric venous pressures and multiple extrahepatic shunts (Fig 9), and portovenogram findings consistent with the inadequate development of a hepatic portal vasculature. Hypovascularity of the hepatic parenchymal portal veins was confirmed histopathologically in all three dogs. In the other three dogs (25, 27 and 28) there was no increase in mesenteric venous pressure and angiographic studies revealed the persistence of blood flow through the original shunt with the partial development of a hepatoportal vasculature. In these three dogs the shunt vessel was ligated completely and they became clinically normal, with no haematological or blood biochemical abnormalities in the long term. One month after its second operation dog 25 had an SI of 7 per cent which was considered normal. Three dogs (13, 16 and 40) were euthanased without further investigation because they developed medically unresponsive signs of encephalopathy.

Discussion

This is the first long-term study of a large group of dogs with intrahepatic portosystemic shunts managed surgically. The age, breed distribution, clinical history and clinical signs of these 45 dogs were similar to those described in other dogs with intrahepatic portosystemic shunts (Breznock and others 1983, Rawlings and

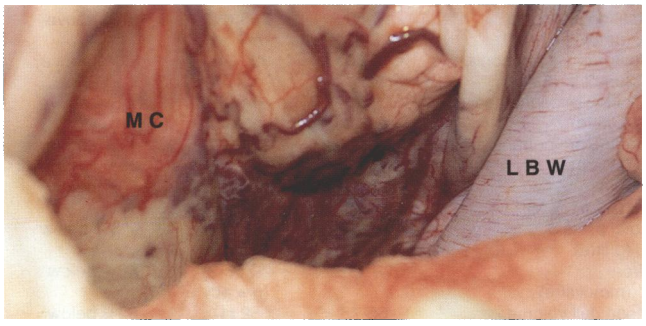

FIG 9: Intraoperative view of the left lateroventral cranial abdomen of dog 7 showing the multiple acquired extrahepatic shunts. LBW Left body wall, MC Mesocolon

TABLE 5: Results of the follow-up of 45 dogs with intrahepatic portosystemic shunts

Dog	Follow-up period (months)	Outcome
1	30	Clinically normal, no medication
2	–	Euthanased at surgery following positive *S typhimurium* urine culture
3	2	Clinically normal, no medication, lost to follow-up
4	7	Clinically normal, no medication
5	–	Ruptured shunt vessel during dissection
6	2	Clinically normal, no medication, lost to follow-up
7	16	Clinically normal, no medication for 12 months, then showed unresponsive encephalopathic signs; 2nd portovenogram showed portal atresia. Owners requested euthanasia
8	–	Died 12 hours postoperatively from shock
9	66	Clinically normal, no medication
10	46	Clinically normal, no medication
11	17	Clinically normal, no medication
12	–	Died 12 hours postoperatively from shock
13	3	Persistent encephalopathy, unresponsive to medication, euthanased
14	28	Clinically normal, no medication
15	24	Clinically normal, no medication, lost to follow-up
16	3	Persistent encephalopathy, unresponsive to medication, euthanased
17	11	Clinically normal, no medication
18	4	Clinically normal, no medication
19	3	Clinically normal, no medication
20	–	Ruptured shunt vessel during dissection
21	5	Clinically normal, no medication for five months, then euthanased for renal failure
22	10	Clinically normal, no medication for nine months, then showed unresponsive encephalopathic signs; 2nd portovenogram showed portal atresia. Owners requested euthanasia
23	15	Clinically normal, no medication for 12 months, then showed unresponsive encephalopathic signs; 2nd portovenogram showed portal atresia. Owners requested euthanasia
24	9	Clinically normal, no medication
25	5 (1)	Clinically normal, no medication (full ligation at 2nd procedure)
26	5	Clinically normal, no medication
27	60 (23)	Clinically normal, no medication (full ligation at 2nd procedure)
28	30 (25)	Clinically normal, no medication (full ligation at 2nd procedure)
29	12	Clinically normal, no medication
30	18	Clinically normal, no medication
31	2	Clinically normal, no medication, lost to follow-up
32	3	Clinically normal, no medication, died from GDV 4 months postoperatively
33	–	Died 18 hours postoperatively from shock
34	10	Clinically normal, no medication
35	6	Clinically normal, no medication, lost to follow-up
36	2	Clinically normal, no medication, lost to follow-up
37	3	Clinically normal, no medication, lost to follow-up
38	38	Clinically normal, no medication, lost to follow-up
39	–	Euthanased two days postoperatively for unmanageable seizures
40	1	Persistent encephalopathy, unresponsive to medication, euthanased
41	5	Clinically normal, no medication
42	63	Clinically normal, no medication
43	–	Ruptured shunt vessel during dissection
44	6	Clinically normal, no medication
45	5	Clinically normal, no medication

GDV Gastric dilatation torsion

Wilson 1983, Wrigley and others 1983, Martin and others 1986, Johnson and others 1987, Komtebedde and others 1991, Tisdall and others 1994, Bostwick and Twedt 1995, Smith and others 1995, Hunt and others 1996).

The results of preoperative blood tests were also similar to those reported in previous studies (Griffiths and others 1981, Center and Magne 1990). The majority of the dogs had high resting and postprandial concentrations of serum bile acids and ammonia and high activities of alkaline phosphatase and alanine aminotransferase. Low concentrations of serum albumin, total protein and urea were also a common finding. Most of the dogs showed evidence of a mild to moderate microcytic anaemia (Simpson and others 1997). Only the postprandial concentrations of serum bile acids and ammonia were outside the normal range in all the dogs measured, and these were the most sensitive tests for detecting the portosystemic shunts. Bostwick and Twedt (1995) have suggested that the extent of the increase in serum alkaline phosphatase activity may be used to predict the site of the portosystemic shunt. However, most of the dogs were under six months of age and it is known that serum alkaline phosphatase may be high in young normal dogs because of an increased production of the bone isoenzyme during growth (Bush 1991). As a result, the significance of high levels of this enzyme in a young dog suffering from a portosystemic shunt should be considered doubtful unless the level is very high.

The portovenogram and surgical findings showed that nearly all the intrahepatic shunts were associated with either the left or the central hepatic divisions. Twenty-nine of the dogs (65 per cent) had a shunt within the left hepatic division, where the shunt passed either through the left lateral lobe of the liver or between the left lateral and the papillary process of the caudate lobe, before draining into the caudal vena cava via the left hepatic vein. This arrangement is consistent with the normal morphology of the ductus venosus (Payne and others 1990) supporting the diagnosis of a PDV. Thirteen dogs (29 per cent) had a shunt within the central hepatic division, where the shunt took the form of a foramen between dilated portions of the intrahepatic portal vein and either the central hepatic vein or the caudal vena cava (Tisdall and others 1994, Hunt and others 1996, Lamb and White 1998). Rothuizen and others (1982), Vulgamott (1985) and Martin and Payne (1990) have classified similar 'right-sided' intrahepatic shunts as a right ductus venosus, a structure for which there appears to be no embryological evidence (Payne and others 1990). It is possible that this form of central divisional shunt represents an embryological persistence of the right omphalomesenteric vein, communication between the right umbilical vein and the cranial anastomosis of the vitelline veins, or the malformation of the hepatic sinusoids (Lamb and White 1998). This communication may develop in the absence of a normal ductus venosus, thereby providing hepatic venous bypass in the developing embryo, or it may develop in addition to the normal ductus venosus. This developmental issue may prove difficult to resolve without investigating neonatal dogs with central divisional shunts. Until it is resolved, the authors favour the hepatic divisional classification for intra-

hepatic shunts. The right divisional shunt observed in dog 14 may represent a malformation of the hepatic sinusoids during the development of the right lateral hepatic lobe.

Intrahepatic portosystemic shunts are shunts which are located within the liver parenchyma (Payne and others 1990). In most cases the shunt vessel originates from either the right or left intrahepatic branch of the portal vein, traverses the hepatic parenchyma and enters either the right, central or left hepatic vein, or the caudal vena cava (Hunt and others 1996, Swalec Tobias and Rawlings 1996, White and others 1996b, Lamb and White 1998). The classification of the shunt in dog 28 was difficult because although the shunt vessel originated intrahepatically from the right branch of the portal vein, its entrance into the caudal vena cava was within the pleural cavity. In one respect this morphology is consistent with an intrahepatic shunt (its origin from the right branch of the portal vein) and in another with an extrahepatic shunt (its entrance into the vena cava outside the hepatic parenchyma). There is no obvious embryological explanation for the development of such a shunt vessel.

Portography was used during surgery in the majority of the dogs. Komtebedde and others (1991) suggested that this procedure is unnecessary in the management of dogs with intrahepatic portosystemic shunts, but the authors consider that it provides the most accurate means of determining the position and morphology of the shunt. By applying the procedure in the theatre any problems associated with loss of sterility or hypothermia when moving an anaesthetised dog to a distant radiology suite were avoided. In most previous reports describing the use of portography, the hepatic portal vasculature has been assessed from a radiograph taken before any manipulation of the shunt vessel (Suter 1975, Rothuizen and others 1982, Martin and Payne 1990, Moon 1990). The dynamic angiographic studies improved the assessment of hepatic portal vasculature, and angiograms were recorded both before the shunt was manipulated and after it had been completely attenuated. The initial portovenogram provided information about the position and morphology of the shunt within the liver. As a result, the majority of the shunts could be accurately ascribed to a hepatic division and an informed decision could be made about the best surgical procedure to use to attenuate the shunt. The angiogram recorded after the shunt had been completely attenuated was used to confirm that the shunt had been identified accurately, to detect possible further shunting vessels (Tisdall and others 1994) and to assess the condition of the intrahepatic portal vasculature. The results of these two angiographic studies showed that the hepatic portal vasculature can be best assessed when the contrast is injected after the shunt has been completely attenuated. The increased portal pressure resulting from the complete shunt attenuation and the injection of a further volume of contrast into this vascular space, most effectively reveal the presence or absence of any intrahepatic portal vessels. The results of the portovenogram studies in conjunction with the postoperative scintigraphic findings suggest that the measurement of both PV1 and PV2 may have a predictive value for the long-term occlusion of the shunt. Swalec and Smeak (1990) suggested that an absence of aborising intrahepatic vasculature is correlated with more postoperative complications. In this study no attempt was made to correlate the portovenogram findings with postoperative complications in the short term, but there appeared to be no correlation between them in the long term.

The low mean mesenteric venous pressure (1 cm H_2O) recorded before the shunt was manipulated was more consistent with the findings of Breznock and others (1983) than with the more recent studies of Hunt and others (1996). In all the dogs in which the mesenteric venous pressure was measured with the shunt fully occluded, it was considered that a potentially fatal hypertension developed and the shunt vessel was therefore ligated only partially. The mean increase in pressure observed was 13 cm H_2O, close to what is thought to be the upper limit without increasing the risk of splanchnic venous hypertension and death (Martin and Freeman 1987, Swalec and Smeak 1990, Whiting and Peterson 1993, Bostwick and Twedt 1995). It is now widely accepted that several factors make the interpretation of pressure changes unreliable; they include variations in the manometric technique, the site of the cannula, the manipulation of the abdominal viscera, the effects of anaesthesia, hypothermia and systemic hypotension, and the venospasm that may be produced in the shunt vessel wall when it is being manipulated (Levy and others 1995, White and others 1996a). It is therefore recommended that other investigations, such as the examination of the splanchnic viscera for venous stasis and congestion and the measurement of systemic arterial and central venous blood pressures, should be used to help the degree to which a shunt may be safely ligated (Komtebedde and others 1991, Hunt and others 1996, Swalec Tobias and Rawlings 1996).

The goal of shunt attenuation surgery is to ligate the portosystemic shunt completely and induce the development of a 'normal' hepatoportal vasculature and function, without inducing a prolonged portal hypertension or the development of multiple portosystemic shunting vessels (Hottinger and others 1995). There is debate about how the hepatoportal vasculature may be induced by the attenuation procedure. In the 37 surviving dogs with a long-term follow-up, there appeared to be three outcomes associated with the partial ligation of the shunt vessel. First, in the majority of the dogs, the partial ligation procedure induced the shunt vessel to attenuate completely within one to six months. These dogs became clinically normal and required no medication or dietary management, and their haematological and biochemical characteristics, including postprandial concentrations of bile acids and ammonia, returned to within their normal ranges. Secondly, the partial ligation procedure failed to induce the shunt to attenuate completely, although the blood flow through both the partially developed hepatoportal vasculature and the shunt was sufficient to maintain a normal portal pressure. These dogs may become clinically normal in either the short or long term. They may not require medication or dietary changes to remain clinically normal, although in the longer term some may develop encephalopathic signs. The haematological and biochemical findings in these individuals rarely return to within their normal ranges. A second surgical procedure can often attenuate the shunt completely. Lastly, in a few of the dogs, the partial ligation procedure failed to induce hepatoportal vasculature development and the portal pressure remained high leading to the development of multiple portosystemic shunts. These individuals showed either persistent or renewed encephalopathic signs which may become unresponsive to medical management, and their haematological and biochemical characteristics commonly deteriorate. Attempts to further attenuate the shunting vessels in these dogs were not possible because of portal hypertension and the chronic hypovascularity of the hepatic parenchymal portal veins. The results of this study do not suggest an indicator which might reliably predict which of these three outcomes may occur in a particular individual.

The SI values measured after surgery were used to assess the persistence of shunting in the long term. Values above the normal range were considered to indicate the likelihood of either continued shunting of blood through the original shunt or the development of multiple portosystemic shunts. Of the 21 dogs in which these SI values were available, seven (33 per cent) had values which indicated that the shunt had been attenuated completely between one and six months after surgery. However, the scintigraphic results may not always predict the long-term outcome reliably. For example, dog 22 had a nearly normal SI of 16 per cent one month after surgery, but was euthanased because of uncontrollable encephalopathic signs nine months later. Pathological examination revealed hypovascularity of the hepatic parenchymal portal veins and the presence of multiple portosystemic shunts.

In all the dogs with a left divisional shunt, the shunt was attenuated successfully by direct posthepatic ligation (Breznock and others 1983). In the authors' opinion, the lack of complications during the surgery and the short average duration of the procedure (63 minutes) make this the best procedure for uncomplicated left divisional PDV shunts. The ligation of central divisional shunts proved less straightforward. Initially, attempts were made to ligate the vessel at a posthepatic site, as with the left divisional shunts. Unfortunately, the position of these shunts deep within the parenchyma of the right medial hepatic lobe (Lamb and White 1998) led to the rupture of the vessel wall during the dissection procedure in three consecutive cases. After these failures, attempts

were made to close this type of shunt by a posthepatic caval venotomy (Breznock and others 1983, Rawlings and Wilson 1983, White and others 1996b). This procedure was technically demanding and time consuming, and although the shunt was ligated successfully in a number of cases, it resulted in the death of three dogs (8, 12 and 33) shortly after surgery. The authors therefore suggest that the techniques of intrahepatic direct shunt dissection with the aid of an ultrasonic aspirator (Swalec Tobias and others 1996) or the transportal closure of the shunt via portal venotomy (Hunt and others 1996) may be more suitable for the attenuation of shunts within the central hepatic division.

On the basis of this series of 45 consecutive cases, it is concluded that the majority of intrahepatic shunts in the dog take one of two distinct morphological forms. The commonest form is a vessel which passes through the left hepatic division before entering the left hepatic vein. This vessel is anatomically consistent with a PDV. In the second form the vessel is situated within the central hepatic division and consists of a foramen between a dilated portion of the right intrahepatic portal vein and either the intrahepatic caudal vena cava or the right hepatic vein. Surgical techniques are available for the attenuation of shunts within any of the hepatic divisions. In dogs with PDV, the shunt vessel may be manipulated most easily at a posthepatic site, whereas in dogs with central and right divisional shunts the vessel may be more easily manipulated intrahepatically, although the procedure may require demanding intravascular surgical techniques.

Addendum

The authors have examined a further 13 dogs with intrahepatic shunts. All of them underwent successful surgical procedures to attenuate the shunt vessel. The details of the cases are summarised in the following table.

Breed	Sex	Age (months)	Hepatic division	Surgical procedure	Ligation
Bernese mountain dog	F	3	Left	Posthepatic direct shunt ligation	Partial
Border collie	F	28	Right	Posthepatic direct shunt ligation	Partial
Border collie crossbred	F	11	Central	Transportal closure via portal venotomy	Partial
Clumber spaniel	M	7	Left (PDV)	Posthepatic direct shunt ligation	Partial
Golden retriever	F	11 weeks	Central	Transportal closure via portal venotomy	Partial
Golden retriever	M	3	Central	Transportal closure via portal venotomy	Partial
Golden retriever	F	10	Left (PDV)	Posthepatic direct shunt ligation	Partial
Irish wolfhound	F	3	Left (PDV)	Posthepatic direct shunt ligation	Partial
Irish wolfhound	M	2	Left (PDV)	Posthepatic direct shunt ligation	Partial
Irish wolfhound	F	2	Left (PDV)	Posthepatic direct shunt ligation	Partial
Irish wolfhound	F	3	Left (PDV)	Posthepatic direct shunt ligation	Partial
Labrador retriever	F	12	Left (PDV)	Posthepatic direct shunt ligation	Partial
Standard poodle	F	2	Central	Intrahepatic direct shunt ligation (similar to dog 14)	Full

PDV Patent ductus venosus

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Assessment of pain in dogs

FOUR veterinary surgeons used three methods to assess the pain being suffered by 50 dogs after they had undergone surgery. The three methods were a simple descriptive scale, a numerical rating scale and a visual analogue scale. With each scale there was significant variability between the observers which accounted for between 29 and 36 per cent of the total variability. It was concluded that the numerical rating scale was probably the most reliable method, because it provided greater sensitivity than the simple descriptive scale and avoided the tendency to over interpretation which was a problem with the visual analogue scale.

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