Outcomes of Cellophane Banding for Congenital Portosystemic Shunts in 106 Dogs and 5 Cats

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Objective—To report outcomes after cellophane banding of single congenital portosystemic shunts in dogs and cats.

Study Design—Retrospective study of sequential cases.

Animals—One hundred and six dogs and five cats.

Methods—Medical records were reviewed for breed, sex, age at surgery, shunt anatomy, results of pre- and postoperative biochemical analysis, development of postligation neurologic dysfunction, portal hypertension or other serious complications, and the owners’ perception of their animal’s response to surgery.

Results—Ninety-five dogs and all 5 cats had extrahepatic shunts. Eleven dogs had intrahepatic shunts. Six dogs (5.5%) died as a result of surgery from portal hypertension (2 dogs), postligation neurologic dysfunction (2), splenic hemorrhage (1) and suspected narcotic overdose (1). Serious complications were more common in dogs with intrahepatic shunts than those with extrahepatic shunts (P = .002). Postligation neurologic dysfunction necessitated treatment in 10 dogs and 1 cat; 8 dogs and the cat survived. Clinical signs attributed to portosystemic shunting resolved or were substantially attenuated in all survivors. Postoperative serum bile acid concentrations or results of ammonia tolerance testing were available for 88 animals; 74 (84%) were normal and 14 (16%) were abnormal. Multiple acquired shunts were documented in two animals.

Conclusions—Cellophane banding is a safe and effective alternative to other methods of attenuation.

Clinical Relevance—Slow occlusion of portosystemic shunts using a variety of methods is being evaluated world wide. Cellophane banding is a relatively simple procedure with comparable safety and efficacy to previously reported techniques.

INTRODUCTION

A GREAT DEAL of attention has been focussed on methods of slowly occluding congenital portosystemic shunts (CPS) in dogs and cats.1–12 The rationale for slow attenuation is manifold, including reduced risk of life-threatening portal hypertension, speculation that slow occlusion may reduce the risk of post-ligation neurologic dysfunction, reduced operating time, less extensive intraoperative monitoring, and the fact that animals undergoing complete shunt occlusion have a better long-term prognosis than those undergoing partial attenuation only.13,14 Two methods of slow occlusion using extravascular techniques have been reported: the
Cellophane banding of a congenital portosystemic shunt in a dog was first described in 1990.5 Rewarding results in a series of 11 dogs with congenital portosystemic shunts5 led to cellophane banding being adopted as the procedure of choice for single extrahepatic shunts in dogs in Sydney, Australia. In time, cellophane banding was also used for CPS in cats, and for attenuation of intrahepatic shunts where it was possible to dissect around the shunt, or the afferent branch of the portal vein.10,15 We report outcomes after cellophane banding of extrahepatic and intrahepatic shunts in 106 dogs and 5 cats.

**MATERIALS AND METHODS**

Medical records from 106 dogs and 5 cats that had surgical attenuation, by cellophane banding, of single, congenital portosystemic shunts between March, 1993 and August, 2002 were reviewed. Ninety-four cases had surgery at the University Veterinary Centre, Sydney and the remainder were treated at two private practices in Sydney. Recorded details were: breed, sex, age at surgery, anatomy of the portosystemic shunt, results of pre- and postoperative biochemical analysis including ammonia tolerance testing (ATT) and pre and postprandial serum bile acid concentration (SBA), development of postoperative complications such as neurologic dysfunction or portal hypertension, and the owners’ perception of the postoperative condition of their pet.

**Anesthesia**

Animals were anesthetized using a variety of techniques. Most were premedicated with phenobarbital (5–10 mg/kg subcutaneously) because of previous observations that perioperative phenobarbital administration may reduce the incidence and/or severity of postligation neurologic dysfunction.16 General anesthesia was then induced by administration of propofol (2–5 mg/kg intravenously [IV], dogs) or alphaxalone (1–2 mg/kg, IV) and maintained with inhaled isoflurane in a 1:2 combination of oxygen and nitrous oxide, supplemented by IV infusion or incremental doses of fentanyl, morphine, or methadone. Anesthetic monitoring included pulse oximetry, end-tidal isoflurane and carbon dioxide concentrations, non-invasive measurement of arterial blood pressure, electrocardiogram and esophageal temperature. In all animals with intrahepatic shunts, catheters were inserted percutaneously for measurement of direct arterial pressure and central venous pressure. Packed cell volume, total plasma protein and blood glucose concentrations were monitored at regular intervals.

**Surgical Procedure**

All animals had a ventral median celiotomy that was extended to a median sternotomy in those with intrahepatic shunts. Surgical times ranged from 30–150 minutes, depending on the site of the shunt. In animals with extrahepatic shunts, surgical times were typically <60 minutes. Portosystemic shunts were identified by abdominal exploration and classified as extrahepatic or intrahepatic. All extrahepatic shunts that entered the azygous vein were identified as portoazygous. Cellophane banding was performed in all animals with extrahepatic shunts, and in those with intrahepatic shunts where it was possible to dissect around either the shunt itself or a branch of the portal vein leading to the shunt. All animals with intrahepatic shunts and dogs weighing >10 kg had a jejunal vein catheterized for measurement of portal venous pressure because application of a cellophane band with a recommended diameter of ≤3 mm5,6 was considered likely to cause substantial and possibly dangerous shunt attenuation in these larger animals.

Cellophane bands were placed as reported previously.5 Briefly, a strip of cellophane (10 cm long, 1.2 cm wide), was folded longitudinally to form a 3-layered strip (10 cm long, approximately 4 mm wide). The strip was passed around the shunt and tightened around both the shunt and a stainless steel pin of pre-determined size, using one or more titanium clips. In dogs <10 kg, the size of the pin (and hence the diameter of the cellophane band) was determined by changes in heart rate, arterial pressure, intestinal color and motility, and pancreatic color when the shunt was totally occluded. Animals with minimal changes (elevation of heart rate <10 beats/minute and reduction in arterial systolic pressure <10 mm Hg) had a 2 mm diameter band applied, whereas those with moderate or severe changes had bands of 2.5 mm or 3 mm, respectively. In most cases, hemodynamic variables and intestinal color and motility were not substantially different to baseline once the band was applied. In dogs where portal pressure was monitored, a pin diameter was chosen that would constrict the shunt as much as possible without exceeding the maximum safe levels of attenuation described previously (a rise of portal pressure ≤10 cm H2O to a final portal pressure of ≤20 cm H2O).18

**Postoperative Care**

Animals were monitored intensively for 24 hours after surgery, then observed for another 48 hours for signs of postligation neurologic dysfunction or portal hypertension. Phenobarbital (2–5 mg/kg every 12 hours) was administered to most animals for 2 weeks to reduce the incidence on severity of postligation neurologic dysfunction.16 Many dogs became ataxic as a result of phenobarbital administration. Ataxia was not considered to be a manifestation of postligation neurologic dysfunction if the animal was otherwise normal and ataxia improved with reduction of the phenobarbital dose.

All animals requiring anticonvulsant therapy were administered IV phenobarbital (30 mg/kg during the first 24 hours, then 5 mg/kg every 12 hours), with or without incremental...
doses of midazolam and acepromazine.16 In 6 animals, IV propofol infusion was also used to control neurologic signs.18 Animals were encouraged to eat on the first postoperative day; usually a combination of chicken and rice or a commercial protein-restricted diet (Hills Canine L/D, Hills, Topeka, KS). Lactulose or antibiotics were administered at the discretion of the surgeon, however, in most animals, these medications were not continued after surgery. Animals were maintained on a restricted-protein diet for at least a month. Owners were then instructed to increase the protein content by adding variable proportions of meat or commercial food with a normal protein concentration. Most animals were receiving a normal diet by 8 weeks after surgery, however, some clients chose to continue protein restriction until liver function tests were performed.

**Evaluation of Hepatic Function After Surgery**

Owners were asked to return their animals for biochemical evaluation of hepatic function at least 8 weeks after cellophane banding wherever possible. Rectal ammonia tolerance testing (ATT) was performed by preference, especially in terrier-type dogs where serum bile acid (SBA) determination using a routine enzymatic test can be misleading.19 Portosystemic shunting was considered to have resolved if ATT was normal (<100 μmol/L after ammonia challenge) or postprandial SBA concentration was <40 μmol/L.19 If ammonia intolerance or elevated SBA concentrations were encountered, results of serum biochemistry (activity of alanine aminotransferase and alkaline phosphatase, concentrations of blood urea nitrogen, cholesterol, and albumin) were compared with preoperative values as an indirect means of assessing improvement in liver function. In Maltese dogs, whose owners were unwilling to return for ATT, the results of pre and postoperative biochemical panels were compared to provide an indication of changes in hepatic function.

Owners who were unable or unwilling to return their pet for re-evaluation were surveyed as to whether they considered their pet’s clinical signs to have resolved, whether it was eating a normal diet and whether they considered it to be otherwise healthy.

**Statistical Analysis**

Fishers Exact test was used to compare mortality, perioperative complication rates, incidence of postligation neurologic dysfunction, and results of biochemical testing between different types of shunt, and incidence of postligation neurologic dysfunction in different breeds. Ages of animals that developed postligation neurologic dysfunction or failure to recover normal hepatic function were compared with the rest of the population using the Mann-Whitney U Test. P values <.05 were considered significant.

**RESULTS**

Cellophane banding was performed in 106 dogs and 5 cats. Breed distribution was Maltese (38; 36%), Cross bred (16; 15%), Jack Russell terriers (8; 7.6%), Miniature toy poodles (6; 5%), Silky terriers (5; 4%), Shihtzus (5; 4%), Bichon frise (4; 3%), Miniature Schnauzers (4, 3%), and other individual breeds of dogs and cats. Fifty-five dogs and 2 cats were female and 51 dogs and 3 cats were male. Reported clinical signs were consistent with those previously reported.1–16

There were 100 extrahepatic shunts and 11 intrahepatic shunts. All 5 cats had extrahepatic shunts. Fifteen dogs had portoazygous shunts (15% of extrahepatic shunts). Thirty animals had shunts from the left gastric vein to the caudal vena cava, and 12 had shunts from the gastroduodenal vein to the caudal vena cava. In 31 animals, shunts were portocaval. In 5 animals, shunts arose from the splenic or gastrosplenic vessels. The vessel of origin of extrahepatic shunts entering the portal vein was not specifically mentioned in 2 animals.

Four dogs had right-divisional intrahepatic shunts and 6 had left-divisional shunts. The exact site of the intrahepatic shunt was not recorded in one animal. One dog had a complex shunt with an abnormal vessel arising from the left gastric vein and joining a patent ductus venosus at the termination of the left branch of the portal vein.

Cellophane band diameter was recorded in 105 cases and ranged from 2–6 mm, with most (99, 94%) being ≤3 mm diameter. No major intraoperative complications were reported.

**Mortality Rate**

Six dogs (5.5 %) died between 4 hours and 4 weeks postoperatively as a result of complications arising from anesthesia or surgery (Table 1) Two dogs (1.8%) died from portal hypertension and 2 from postligation neurologic dysfunction. Only 3 of 100 dogs (3%) with extrahepatic shunts died compared to 3 of 11 (27%) with intrahepatic shunts (P = .01). None of the cats died.

**Postligation Neurologic Dysfunction**

Postligation neurologic dysfunction occurred in 11 animals (10%; 10 dogs and 1 cat; Table 2), some of which were previously reported.16 Initial signs were observed 4–72 hrs after surgery (mean, 36 hours). The incidence of postligation neurologic dysfunction was not significantly different for animals with extrahepatic shunts (10/100, 10%) versus intrahepatic shunts (1/11, 9%, P = 1). Age at surgery for the dogs and cat that had neurologic dysfunction ranged from 5–74 months (mean, 24.7 months), which was not significantly different to the rest of the population (mean, 22.1 months; range 2–96 months, P = .77).
No obvious predisposing factors for postligation neurologic dysfunction were identified, although 3 (38%) of the 8 Jack Russell terriers were affected. This breed was significantly over-represented for postligation neurologic dysfunction when compared to Maltese (2/36; 5.5%, \( P = 0.03 \)) and all other breeds (5/62; 8%, \( P = 0.04 \)). The cat that had postligation neurologic dysfunction was a Scottish Fold.

Nine of 11 animals (82%) that had postligation neurologic dysfunction survived, including all 5 animals administered phenobarbital, and 4 of 6 dogs administered phenobarbital and a propofol infusion. One dog had a cardiorespiratory arrest while being treated for status epilepticus and another dog was euthanatized 4 weeks after surgery because of severe, residual neurologic deficits. Eight of 9 survivors had normal liver function at least 2 months after surgery based on rectal ATT. Two dogs remain on oral phenobarbital to control signs associated with partial motor seizures.

**Postoperative Liver Function**

Postoperative evaluation of liver function was performed at least 8 weeks after surgery (range, 2–6 months; median, 2.25 months) using rectal ATT in 27 Maltese, 42 non-Maltese dogs and 3 cats, and by determination of postprandial SBA concentrations in 14 non-Maltese dogs and 2 cats. Results indicated normal liver function in 74 of the 88 animals (71/83 dogs, 85%; 3/5 cats, 60%). Results indicated residual abnormalities in 12 dogs (15%) and 2 cats (40%) ranging from mild (post challenge serum ammonia 105 \( \mu \text{mol/L} \)) to severe (serum ammonia 800 \( \mu \text{mol/L} \)). In all cases, however, the owners reported that clinical signs had resolved or been substantially attenuated. In three animals, the owners reported that the dog seemed livelier when dietary protein concentration was reduced, however, in none of these cases were clinical signs as severe as before surgery (Table 3).

The causes of persistent elevations in liver function tests were not determined in most animals, but may have been because of persistent shunting for reasons that were not characterized. One dog had an episode of ascites 10 days after surgery, presumably because of portal hypertension. Multiple acquired shunts were identified at repeat celiotomy in a cattle dog after application of a 4 mm cellophane band to an intrahepatic shunt. Repeat celiotomy was performed in both cats with ammonia intolerance after surgery; multiple acquired shunts were seen in one of these cats. In the other, the original shunt had failed to close. Follow up ATT and postprandial SBA determination 2 months after surgery indicated persistent hepatic dysfunction.

Six dogs where results of ATT or SBA were not available had broad-based biochemical panels performed. Five had complete resolution of preoperative abnormalities such as elevation of liver enzymes, or low concentrations of urea, cholesterol, and albumin.

### Table 1. Cause of Death in 6 Dogs after Cellophane Banding of Portocaval Shunts

<table>
<thead>
<tr>
<th>Breed</th>
<th>Age (months)</th>
<th>Sex</th>
<th>Shunt Type</th>
<th>Cause of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Border collie</td>
<td>18</td>
<td>F</td>
<td>LDIH</td>
<td>Portal hypertension 3 days after banding left branch of portal vein</td>
</tr>
<tr>
<td>Bichon frise</td>
<td>5</td>
<td>F</td>
<td>L Gastric</td>
<td>Portal hypertension 2 days after surgery</td>
</tr>
<tr>
<td>Miniature poodle</td>
<td>28</td>
<td>F</td>
<td>Portoazygous</td>
<td>Cardiorespiratory arrest during treatment of seizures</td>
</tr>
<tr>
<td>Maltese</td>
<td>33</td>
<td>M</td>
<td>L Gastric</td>
<td>Euthanized 4 weeks after surgery due to severe residual neurological deficits</td>
</tr>
<tr>
<td>Old English sheepdog</td>
<td>4</td>
<td>M</td>
<td>LDIH</td>
<td>Hemorrhage secondary to splenic rupture 2 days after surgery</td>
</tr>
<tr>
<td>Miniature poodle</td>
<td>84</td>
<td>M</td>
<td>RDIH</td>
<td>Hypothermia and respiratory arrest 4 h after surgery. Suspected narcotic overdose.</td>
</tr>
</tbody>
</table>

Abbreviations: L, left; LDIH, left-divisional intrahepatic; RDIH, right-divisional intrahepatic

### Table 2. Details of 10 Dogs and 1 Cat Requiring Treatment for Postligation Neurologic Dysfunction

<table>
<thead>
<tr>
<th>Breed</th>
<th>Age (months)</th>
<th>Sex</th>
<th>Hours Postoperative</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jack Russell terrier</td>
<td>12</td>
<td>M</td>
<td>60</td>
<td>Recovered fully</td>
</tr>
<tr>
<td>Jack Russell terrier</td>
<td>16</td>
<td>F</td>
<td>30</td>
<td>Recovered fully</td>
</tr>
<tr>
<td>Jack Russell terrier</td>
<td>7</td>
<td>F</td>
<td>18</td>
<td>Recovered fully</td>
</tr>
<tr>
<td>Maltese</td>
<td>74</td>
<td>M</td>
<td>48</td>
<td>Recovered fully</td>
</tr>
<tr>
<td>Maltese</td>
<td>33</td>
<td>M</td>
<td>36</td>
<td>Euthanized 4 weeks after surgery due to severe residual neurological deficits</td>
</tr>
<tr>
<td>Australian silky terrier</td>
<td>10</td>
<td>M</td>
<td>72</td>
<td>Partial seizures</td>
</tr>
<tr>
<td>Terrier X</td>
<td>6</td>
<td>F</td>
<td>24</td>
<td>Recovered fully</td>
</tr>
<tr>
<td>Pug</td>
<td>40</td>
<td>F</td>
<td>40</td>
<td>Minor deficits, partial seizures</td>
</tr>
<tr>
<td>Miniature poodle</td>
<td>28</td>
<td>F</td>
<td>24</td>
<td>Cardiorespiratory arrest during treatment</td>
</tr>
<tr>
<td>Rhodesian ridgeback</td>
<td>8</td>
<td>M</td>
<td>40</td>
<td>Recovered fully</td>
</tr>
<tr>
<td>Scottish fold cat</td>
<td>9</td>
<td>M</td>
<td>4</td>
<td>Recovered fully</td>
</tr>
</tbody>
</table>
In the remaining 11 dogs, no objective assessment of liver function was performed. However, follow up reports from owners between 2 months and 6 years after surgery indicated resolution of clinical signs.

Results of Cellophane Banding for Extrahepatic versus Intrahepatic Shunts

The perioperative and early postoperative complication rate for cellophane banding of extrahepatic shunts was relatively low (13/100, 13%). Only 3 (3%) animals died. In contrast, the perioperative and early postoperative complication rate for intrahepatic shunts was significantly higher (6/11, 55%, \(P = .002\)), with 3 animals dying (27%, \(P = .008\)). One additional dog had postligation neurologic dysfunction and 2 had symptomatic but non life-threatening portal hypertension within 10 days of cellophane banding of intrahepatic shunts.

Postoperative evaluation of hepatic function was performed in 7 of 8 dogs that survived cellophane banding of intrahepatic shunts. Hepatic function was normal in 5/7 dogs (71%). By comparison, hepatic function tests were normal in 66/76 (87%) dogs with extrahepatic shunts. Hence, survival with resolution of biochemical abnormalities occurred in only 5 (50%) of 10 dogs with intrahepatic shunts compared to 66 (84%) of 79 dogs with extrahepatic shunts (\(P = .03\)).

Results of Cellophane Banding in Cats

All 5 cats survived cellophane banding. One cat developed mild neurologic dysfunction (twitching) that resolved within 7 days after surgery. Liver function normalized after surgery in three cats, whereas ammonia intolerance persisted in the other two cats. At repeat celiotomy, multiple acquired shunts were observed in one cat and failure of the cellophane band to produce fibrosis was observed in the other cat. Interestingly, the cat in which cellophane failed to promote shunt closure developed multiple acquired shunts after further attenuation using a silk ligature. The clinical condition of both cats improved substantially as a result of cellophane banding, even though portosystemic shunting persisted. Hence, although the clinical result was good to excellent in all cats, the rate of resolution of hepatic dysfunction was only 66%.

DISCUSSION

Our results indicate that cellophane banding is an effective method of alleviating hepatic dysfunction resulting from a congenital portosystemic shunt. Mortality and morbidity rates compare favorably with reports where attenuation was achieved by use of silk ligatures or ameroid constrictors.1-4,7-14 In particular, the incidence of life-threatening portal hypertension was very low, despite the fact that placement of cellophane bands produced substantial shunt occlusion. Three large breed dogs had signs compatible with portal hypertension within 10 days of surgery, however all responded well to symptomatic and supportive therapy. In an experimental study in dogs, cellophane banding produced up to 3 mm of occlusion in the first 6 weeks after placement around femoral veins.6 For this reason, bands of 3 mm diameter or less were applied where possible in our study. However, wider bands can cause eventual shunt occlusion as reported in the present series and a previous

Table 3. Details of 12 Dogs and 2 Cats with Evidence of Persistent Hepatic Dysfunction after Cellophane Banding

<table>
<thead>
<tr>
<th>Breed</th>
<th>Age (months)</th>
<th>Sex</th>
<th>Shunt Type</th>
<th>Band (mm)</th>
<th>ATT ((\mu)mol/L)</th>
<th>SBA ((\mu)mol/L)</th>
<th>Further information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bulldog</td>
<td>12</td>
<td>F</td>
<td>RDIH</td>
<td>6</td>
<td>500</td>
<td>NA</td>
<td>Ascites 10 d after surgery–suspected PH</td>
</tr>
<tr>
<td>ACD</td>
<td>24</td>
<td>F</td>
<td>LDIH</td>
<td>4</td>
<td>180</td>
<td>105</td>
<td>MAS at follow-up surgery</td>
</tr>
<tr>
<td>Pyrrenean mountain dog</td>
<td>30</td>
<td>M</td>
<td>LDIH</td>
<td>6</td>
<td>800</td>
<td>506</td>
<td>NA</td>
</tr>
<tr>
<td>Siberian husky</td>
<td>7</td>
<td>M</td>
<td>IH</td>
<td>NR</td>
<td>105</td>
<td>97</td>
<td>NA</td>
</tr>
<tr>
<td>Jack Russell terrier</td>
<td>11</td>
<td>M</td>
<td>Ileocolic</td>
<td>3</td>
<td>143</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Jack Russell terrier</td>
<td>12</td>
<td>M</td>
<td>Gastroduodenal</td>
<td>3</td>
<td>143</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Maltese</td>
<td>17</td>
<td>M</td>
<td>L gastric</td>
<td>2.5</td>
<td>337</td>
<td>280</td>
<td>Failure of shunt occlusion</td>
</tr>
<tr>
<td>Maltese</td>
<td>36</td>
<td>F</td>
<td>L gastric</td>
<td>2</td>
<td>299</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Maltese X</td>
<td>12</td>
<td>F</td>
<td>Gastroduodenal</td>
<td>2.5</td>
<td>299</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Maltese</td>
<td>12</td>
<td>M</td>
<td>L Gastric</td>
<td>2.5</td>
<td>105</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Bichon frise</td>
<td>84</td>
<td>F</td>
<td>Gastroduodenal</td>
<td>2.5</td>
<td>555</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Pekingese</td>
<td>34</td>
<td>M</td>
<td>Portaazygous</td>
<td>3</td>
<td>123</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Himalayan</td>
<td>6</td>
<td>F</td>
<td>Portocaval</td>
<td>3</td>
<td>337</td>
<td>280</td>
<td>MAS at follow up surgery</td>
</tr>
<tr>
<td>DSH</td>
<td>6</td>
<td>M</td>
<td>Portocaval</td>
<td>2.5</td>
<td>280</td>
<td>MA</td>
<td>NA</td>
</tr>
</tbody>
</table>

Abbreviations: ACD, Australian cattle dog; ATT, ammonia tolerance test; IH, intrahepatic; LDIH, left-divisional intrahepatic shunt; MAS, multiple acquired shunts; NA, not available; PH, portal hypertension; RDIH, right-divisional intrahepatic shunt; SBA, postprandial serum bile acids.
OUTCOMES OF CELLOPHANE BANDING

The incidence of postligation neurologic dysfunction we report was identical to that reported after portosystemic shunt ligation using silk,\textsuperscript{14} suggesting that rapidity of attenuation is not a major risk factor. Postligation neurologic dysfunction was no more common in animals with extrahepatic than those with intrahepatic shunts which also corroborates previous reports.\textsuperscript{10,20} Although administration of phenobarbital does not reduce the incidence of postligation neurologic dysfunction, it does seem to reduce the severity of signs,\textsuperscript{16} and this may explain why the survival rate in our study was higher than in some previous reports.\textsuperscript{18,20} The apparent predisposition of Jack Russell terriers to postligation neurologic dysfunction is interesting, however, the sample size was small and this observation should not be over-interpreted.

The number of cats in our study was also small, making it difficult to draw firm conclusions about the success of cellophane banding in cats, although their behavior after shunt attenuation was consistent with other reports.\textsuperscript{11,12,21} Two recent studies of ameroid constrictor placement in cats reported scintigraphic evidence of persistent shunting in 8/14 (57\%) and 3/7 (43\%) cases, respectively.\textsuperscript{11,12} Failure of the cellophane band to promote any shunt attenuation in one of our cats was disturbing and may indicate a species difference in response to cellophane banding. Further evaluation of cats and dogs with postoperative shunting is required to determine how many have failure of occlusion of the original vessel, development of multiple acquired shunts, or both conditions.\textsuperscript{23}

Biochemical analysis was chosen as the follow-up procedure of choice in our study for various reasons. Previously, we reported that persistent biochemical evidence of liver dysfunction was a sensitive predictor of clinical relapse in dogs undergoing partial shunt attenuation using silk.\textsuperscript{14} Portal scintigraphy was not available at our clinic. Unfortunately, the predisposition of Maltese to congenital portosystemic shunts, the difficulty of interpreting results of bile acid determination in this breed\textsuperscript{19} and the restricted availability of facilities for determining blood ammonia levels in regional centers impaired our ability to adequately evaluate some cases postoperatively. Nevertheless, readers should note that liver function tests such as ATT and SBA determination are only reliable when they are performed correctly and these tests may not detect small amounts of portosystemic shunting. Finally, animals with portosystemic shunts do not always have elevated fasting and/or postprandial ammonia levels.\textsuperscript{24}

The reason why some animals develop multiple acquired shunts after total shunt occlusion remains unclear. No obvious risk factors have been identified. It is tempting to implicate pre-existing portal hypoplasia, however, in our experience many animals with macroscopically narrow portal veins make complete recoveries. Presumably, in animals that develop multiple acquired shunts, the primary shunt closes too rapidly for the developing hepatic vasculature to decompress the portal system. The liver’s response to altered blood flow is thus the limiting factor.

Based on our observations, animals with portosystemic shunting through acquired vessels seem less likely to display severe signs of hepatic encephalopathy than those with a single, congenital vessel. The majority of animals in our study did not require medical management of hepatic encephalopathy after surgery, despite persistent shunting or biochemical abnormalities suggestive of shunting in some. Although these tests are not specific and can be affected by diet, increases in the products of hepatic metabolism such as urea and cholesterol support the contention that hepatic portal blood flow increases...
enough to improve hepatic function significantly, even when persistently elevated hepatic vascular resistance results in chronic portal hypertension.

Why the biological response, such as the rate and adequacy of hepatic vascular regeneration, differs so much between individual dogs is the subject of ongoing debate and is worthy of further investigation. Ultimately, it is likely that adjunctive treatments to encourage hepatic regeneration and development of the hepatic microvasculature will be shown to maximize the efficacy of surgical attenuation of portosystemic shunts.

In conclusion, cellophane banding is a relatively safe, effective technique that results in resolution of biochemical abnormalities resulting from portosystemic shunting in most cases. Results of cellophane banding compare favorably with those of other techniques and it should be added to the repertoire of surgeons treating this common condition.

ACKNOWLEDGMENTS

The authors thank Dr Richard Malik, Dr Jody Braddock and other clinicians at the University Veterinary Centre, Sydney, for assistance with case management, the private practitioners who referred cases for treatment, and Dr David Snow (Mayne Vetnostics) for donation of diagnostic services.

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