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Anaesthesia and portosystemic shunt ligation in the feline patient

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ABSTRACT: Portosystemic shunts (PSSs) are abnormalities of the blood circulatory system, whereby blood from the heart bypasses the liver and enters the general circulation.[1,2]

The initial section of this article discusses the background and theory of PSSs.

The second part illustrates how this is applied when treating patients affected with PSSs in practice, with particular emphasis on anaesthetic and drug protocol using a case study.

Cabassu et al. (2011) explain that portosystemic shunts (PSSs) are anomalous vascular connections between the portal and systemic venous circulatory systems.[3] Padgett (2002) suggests that this, therefore, allows portal blood draining the stomach, intestines, pancreas and spleen to deviate from the liver, causing cirrhosis, hepatic atrophy and encephalopathy.[4]

Bennett (2010) and Carter (2010) add that because the liver is unable to filter toxins appropriately, they circulate throughout the body entering the central nervous system affecting neural function and transmission. [5&6]

The American College of Veterinary Surgeons (2009) and Padgett (2002) state that PSSs are classified as intrahepatic or extrahepatic based on their location relative to the liver.[1,4]

Fossum et al. (2007) discuss how intrahepatic shunts are located within the liver and are usually congenital, singular shunts which occur as a result of the ductus venosus remaining patent following birth.[7] However, extrahepatic shunts are vascular abnormalities outside of the hepatic parenchyma and are either congenital or acquired.[4]

Congenital extrahepatic shunts are commonly single, abnormal vessels that allow atypical blood flow from the portal vein to the systemic circulation.[7]

In contrast, acquired extrahepatic shunts are typically multiple; thought to occur as a result of increased resistance to portal blood flow and subsequent portal hypertension. This hypertension causes normal, non-functional microvascular connections – which are present at birth, between portal and systemic veins – to become functional.[4]

Multiple shunts are most commonly associated with chronic, severe hepatic disease, such as cirrhosis.[7]

Cabassu et al. (2011) established that single congenital extrahepatic portosystemic shunts (CEPSSs) are commonly diagnosed in cats; however according to Fossum et al., intrahepatic portosystemic shunts (IPSSs) have been reported and account for approximately 10 per cent of cases seen in felines. [3,7]

In addition, domestic short-haired cats are most commonly affected by PSSs.

Carter (2010) explains that animals presenting with PSSs can show signs of weakness, ataxia, stupor, head pressing, circling, pacing, blindness, dementia and seizures.[6] The American College of Veterinary Surgeons (2009) adds that the signs are often episodic and may be more noticeable following eating.[1]

In addition, Padgett (2002) has established that PSS patients often have a plethora of historical findings including vomiting, diarrhoea, intermittent anorexia, polyphagia, polyuria, polydipsia and prolonged recovery times following a previous anaesthetic experience such as neutering, secondary to the liver being unable to metabolise the drugs used.[4]

Greene and Marks (2007) add that patients affected with PSSs are usually under weight and small for their breed and age.[8] According to Padgett (2004) the commonest clinical abnormalities in cats with a PSS are ptyalism, copper coloured irises and heart murmurs **(Figure 1)**.[9]

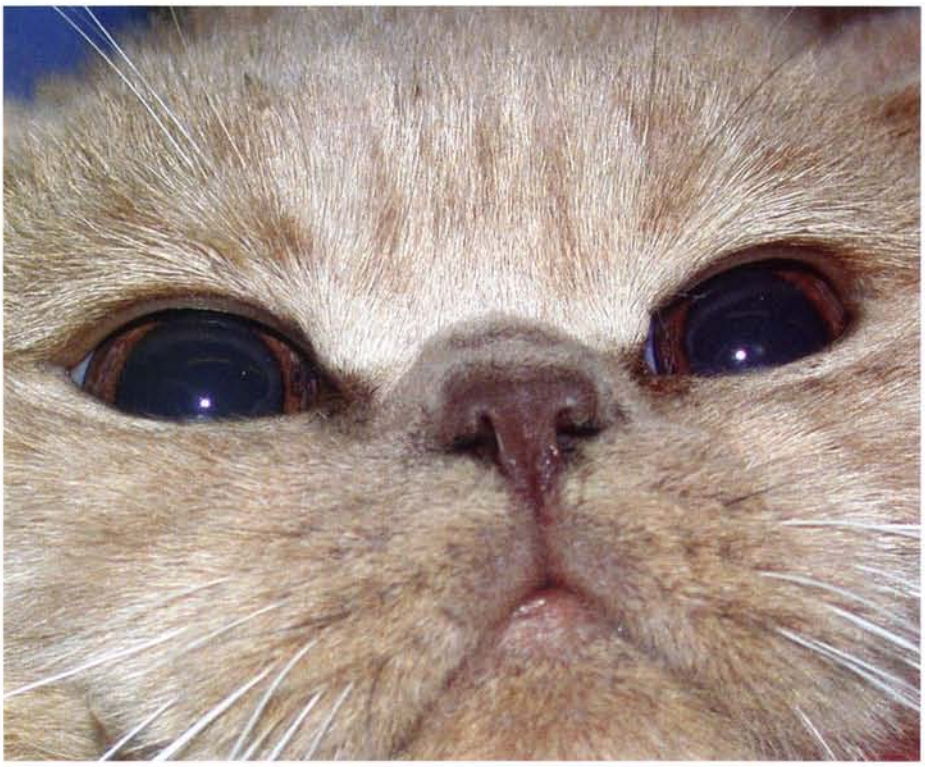

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Figure 1. A common clinical abnormality in cats with a PSS is copper coloured irises

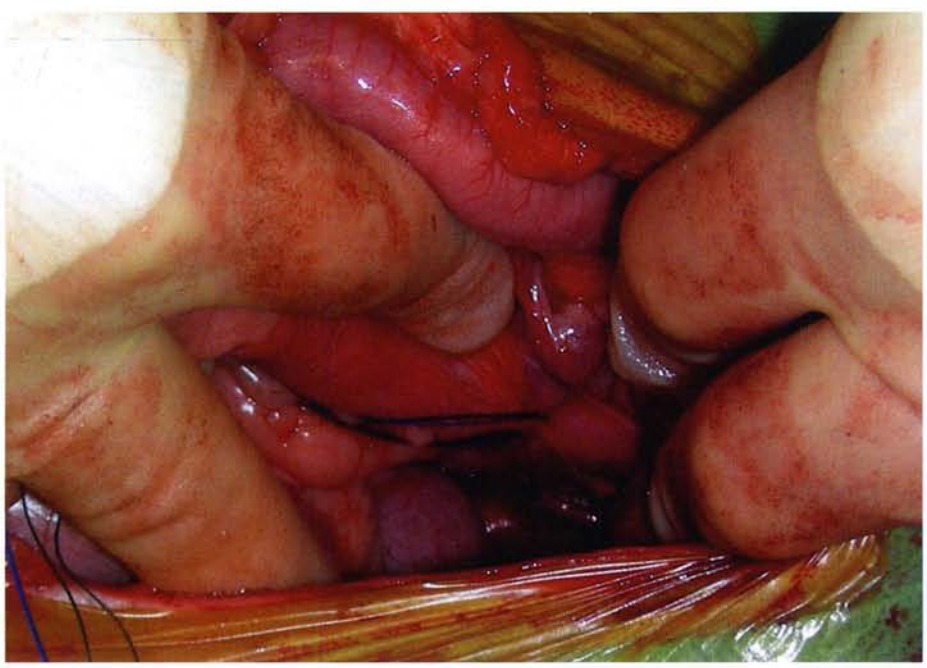
Diagnostic evaluation

The liver is important for drug metabolism and clearance. However diseases such as PSSs affect hepatic function.[5, 10] It is, therefore, vital to perform a thorough evaluation of the hepatic system in order to determine how well the patient is able to metabolise and excrete drugs plus anaesthesia agents.[11] This allows an appropriate drug and anaesthetic protocol to be determined in terms of drug choice and dose to help prevent anaesthetic complications.[6]

The American College of Veterinary Surgeons (2009) determined that patients with PSSs often have increases in total bilirubin, serum bile acids, white blood cell count and prolonged clotting times.[1] In addition, these patients usually have decreased blood urea, blood glucose (BG), albumin, total plasma protein concentrations and packed cell volume (PCV) tends to decrease, invariably with low albumin values.

Bennett (2010) adds that patients with PSSs generally have elevated fasting ammonia concentrations and hepatic enzymes.[5, 11] Plasma proteins, alkaline phosphatise (ALK Phos) and alanine aminotransferase (ALT) are diagnostic tests that help identify the severity of the disease. In addition, diagnostic imaging is often useful in identifying obvious abnormalities.[11]

Figure 2. A definitive diagnosis is made only by surgical identification of the shunt



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Although not fully understood, ammonia is the primary toxin associated with hepatic encephalopathy.[5] Ammonia is produced by a protein that is broken down by the intestinal tract. However as the result of a shunt, ammonia causes hepatic encephalopathy (inability of the liver to metabolise nutrients and toxins).[6]

As a consequence of the lack of nutrients from the pancreas and intestines, the liver can atrophy, potentially leading to hepatic failure.[7]

Medical management

The primary aim of medical management is to minimise clinical signs, thus maximising patient condition and reducing the likeliness of postoperative complications.[4] This should, therefore, be instigated prior to surgery.[5]

The main therapies used in medical management are lactulose, oral antibiotics and a protein-restricted diet.[1] Lactulose is hydrolysed within the colon to organic acids, creating osmotic diarrhoea and decreasing colonic pH, thereby reducing the amount of ammonia absorbed.[4]

Orally administered antibiotics are used to reduce the number of ammonia-producing bacteria in the colon, therefore decreasing the amount of toxins absorbed.[9] A low fat diet should be fed to minimise the amount of faecal material available in the colon for

potential breakdown.[4] Within this, diet protein should be restricted to reduce the substrate for ammonia production.[9]

Surgical therapy

The performance of an extensive abdominal ultrasound scan performed by a specialist in veterinary diagnostic imaging contributes in the diagnosis of a PSS.[6] However, a definitive diagnosis is made only by surgical identification of the shunt, supported with contrast angiography highlighting abnormal portal flow directed by intraoperative fluoroscopy **(Figure 2)**.[7]

Fossum et al. (2007) explain how attenuation is usually performed with the use of an ameroid constrictor placed around the abnormal vessel.[7] This is a cellophane band that slowly occludes the shunt. As the inner portion of the ameroid constrictor swells, the shunt is constricted.[9] Shunt occlusion continues as fibrosis develops around the abnormal vessel. [6,7]

Anaesthesia and perioperative complications usually do not arise until the shunt is ligated.[6] Portal hypertension is the concern, however, because the ameroid constrictor occludes the vessel slowly, this does not often occur.[6]

It should be noted that because there is an increase in clotting times associated with PSS patients, prothrombin time (PT) and activated partial thromboplastin time (PTT) should be measured prior to surgery.[11,8]

CASE REPORT

A six-month-old, 1.6 kg entire male, British short-haired cat was presented, following referral with a history of ataxia, behavioural changes, failure to grow, small body stature, heart murmur, ptyalism and copper colouring of the iris.

On physical examination the above historical findings were confirmed. The patient was also found to have pale mucous membranes and was identified as lethargic.

Plasma proteins, ALK (Phos), ALT, ammonia, PCV and BG were tested. In addition, an extensive abdominal ultrasound was performed which suggested that the patient had a PSS. As a result the following were prescribed: oral lactulose (Lactulose, Sandoz) at a dose of 2ml three times a day, oral amoxicillin/clavulanic acid (Synulox, Pfizer) at a dose of 25mg twice a day.

A hepatic support diet was also recommended and surgical intervention was scheduled for six weeks later.

In the meantime, the patient attended regular appointments with the referring veterinary surgeon, where repeat liver function tests were performed and drug doses were altered, as per effect with regards to the lactulose and according to the animal's body weight.

On the day of surgery, when a shunt was successfully identified and attenuated, pre-surgical physical examination revealed that the patient had increased in body weight to 1.8 kg and his overall body condition had improved. The animal's BG, ammonia and PCV parameters were obtained prior to premedication.

In addition his rectal temperature, pulse and respiratory rate (TPR) mucous membrane colour, and capillary refill time (CRT) were recorded prior to induction of anaesthesia **(Table 1)**.

The patient was premedicated using a dose rate of 0.2 mg/kg of methadone (Physeptone, Martindale Pharmaceuticals) administered intramuscularly (IM) to provide pre-emptive analgesia. (Note: methadone is not currently licensed for use in felines).

After 30 minutes the patient was transferred to the induction area where a 22G intravenous (IV) catheter was placed into the right cephalic vein following aseptic preparation. Pre-oxygenation was administered for five minutes prior to induction of anaesthesia achieved by intravenous administration of alfaxalone (Alfaxan, Vetoquinol) at a dose rate of 5mg/kg.

It is, however, the view of Bennett (2010) that propofol (Vetofol, Norbrook Laboratories) would be a more appropriate anaesthesia induction agent as it undergoes respiratory metabolism and, therefore, does not rely so greatly on the liver.[5]

Diazepam (Diazemuls, Actavis Nordic) was not administered at induction, owing to its metabolism by the liver.

Tracheal intubation was achieved using a laryngoscope and 4mm PCV low pressure, high volume, un-cuffed endotracheal tube (ETT). Cuffed ETTs should not be routinely used in cats because of the risk of damaging – and consequently causing narrowing of – the respiratory tract.[12]

The patient's eyes were lubricated using carbomer (Viscotears, BR Lewis Pharmaceuticals) to prevent drying and possible ulceration of the corneas.

General anaesthesia was maintained using the volatile anaesthetic agent sevoflurane (Sevoflo, Abbott Laboratories) carried in 100% oxygen and delivered via a T-piece anaesthetic

Table 1. Pre-operative parameters

		Reference range
Temperature	38.3°C	38.0 - 38.5°C*
Pulse	208 beats per minute	110 - 180 beats per minute*
Respiratory rate	60 breaths per minute	20 - 30 breaths per minute*
Mucous membrane colour	Salmon pink	Salmon pink*
Capillary refill time	1.5 seconds	< 2 seconds*
Packed cell volume	33 per cent	24 - 40 %*
Ammonia	44mcg/dl	30 - 65mcg/dl**
Blood glucose	4.6mmol/l	3 - 7mmol/l*

*(Lane & Cooper, 1999)[18]

**(Vetlab, 2011)[19]

circuit (Note: sevoflurane is currently not licensed for routine use in cats). A fresh gas flow was set at 600 ml/kg/per minute.

Bennett (2010) and Greene and Marks (2007) suggest that sevoflurane is suitable for maintaining anaesthesia in patients with PSSs as it undergoes little hepatic metabolism. In addition, the minimum alveolar concentration (MAC) (1.58 in cats) of sevoflurane is reduced owing to increased toxicity.[5, 8]

In order to achieve adequate intraoperative analgesia and decrease the concentration of inhalant anaesthetic required, thereby assisting the maintenance of cardiac output and arterial blood pressure, the patient received an IV bolus of fentanyl (Sublimaze, Martindale Pharmaceuticals) at 3mcg/kg followed by fentanyl 8mcg/kg/hour as a constant rate infusion (CRI) **(Table 2)**.[13]

Table 2. Fentanyl calculations

Owing to the small size of the patient and, therefore, small volume of drug required; 100mcg of fentanyl was diluted in 10ml of water for injection thus
100 ÷ 10 = 10 mcg per ml

Fentanyl bolus
3mcg/ kg/per hour
3mcg x 1.8 kg = 5.4
5.4 ÷ 10 = 0.54 ml

Fentanyl continued rate infusion
8mcg/kg/per hour
8mcg x 1.8 kg = 14.4
14.4 ÷ 10 = 1.4 ml/per hour

Rates for 7 to 1 mcg were also calculated so that the level of analgesia could be rapidly altered to the individual's requirements throughout the surgical procedure.

To facilitate a timely extubation, the fentanyl infusion was discontinued 30 minutes prior to the anticipated completion of surgery. Fentanyl was chosen as research has shown that liver disease is unaffected by the use of this drug.[5]

Non-steroidal anti-inflammatory drugs (NSAIDs) are contra-indicated in cases of hepatic disease and were, therefore, not administered. Nitrous Oxide (N_20) is not available within the practice and was consequently not used to augment analgesia.

Intravenous fluid therapy (IVFT) was administered using compound sodium lactate (Vetivex, Dechra) at a rate of 10 ml/kg/hour from the point of general anaesthesia induction. The plan was to continue with this rate of infusion throughout the duration of anaesthesia. However, owing to acute reflex hypotension (systolic <90 mmHg) caused by the ameroid constrictor being placed following the diagnosis of an extrahepatic shunt, this rate was incrementally increased, initially to 15 ml/kg/hour and then to 20ml/kg/hour over a 30-minute period.[14]

This did not increase the animal's blood pressure sufficiently, therefore a 5ml bolus of hydroxyethyl starch in isotonic sodium chloride solution (Voluven, Fresenius Kabi) was administered over 15 minutes. This was repeated following a 15-minute break. Whilst the Voluven boluses were being administered, the flow rate of compound sodium lactate was reduced to 10ml/kg/per hour, but was increased back to 20ml/kg/hour following the boluses. (Note: the Doppler technique measures only systolic pressure. In cats this method underestimates true systolic pressure and tends to be more closely related to the mean blood pressure).

Blood flow to the major organs is compromised when mean blood pressures fall below 60 - 120mmHg.[15]

Owing to the presence of anaerobic bacteria in the liver, antibiosis should be administered whilst performing hepatic surgery.[6] So 20mg/kg of amoxicillin clavulanate (Augmentin, Glaxo Smith Kline) was administered 20 minutes prior to the initial surgical incision and every 90 minutes thereafter, until a final dose had been administered following the closure of the surgical site.

Anaesthesia monitoring included electrocardiogram, pulse oximeter, capnogram, non-invasive (indirect) blood pressure monitoring using the Doppler method and oesophageal core temperature using a Mindray PM8000Vet anaesthetic monitor.

Anaesthesia depresses a patient's ability to thermoregulate.[14] In addition, patients are prone to quickly losing heat when the liver is not functioning correctly, as it is a major exothermic organ.[16] The monitoring of body temperature is therefore extremely important, especially in small animals owing to their large surface area to volume ratio, allowing rapid loss of core body heat.[5] This is particularly true during surgery to ligate PSSs because of the open abdominal cavity and surgical preparation solutions used.

As previously mentioned, patients with PSSs usually have decreased BG and albumin concentrations, often resulting in hypotension as a result of hypoalbuminia. Consequently, these animals are at risk of hypothermia owing to hypoglycaemia. It is, therefore, vital to monitor these parameters during anaesthesia.[9]

Whilst maintained under general anaesthesia, the animal was positioned in dorsal recumbency. To prevent hypothermia, the patient was placed on an under-patient Bair Hugger blanket and his upper body and extremities were wrapped in bubble wrap. A heat moisture exchanger was also used. A heat pad was avoided, however, owing to the risk of thermal burns.

The animal's BG was monitored approximately every 30 minutes throughout the duration of anaesthesia **(Table 3)**. Supplementary glucose was not considered necessary as the readings remained above 2.7 millimoles per litre (mmol/l). Neurological damage may occur when levels decrease below this.[16]

Table 3. Perioperative blood glucose sample results

1st sample	4.7mmol/l
2nd sample	3.7mmol/l
3rd sample	8.2mmol/l
4th sample	6.0mmol/l

During periods of reduced respiratory rate End Tidal Carbon Dioxide (ETC02) levels were correspondingly high, which resulted in the patient being ventilated intermittently in order to maintain normocapnia (35 - 45 mmHg).[14]

After the sevoflurane had been discontinued, the patient was

maintained on oxygen until signs of the swallowing reflex became apparent. It was, however, ensured that the ETT was removed prior to the animal swallowing, which is preferable in cats because of their predisposition to laryngeal spasm and oedema.[17]

Following surgery, the patient recovered in an incubator to compensate for a degree of hypothermia. Parameters recorded postoperatively are shown in **Table 4**. His TPR was recorded every 15 minutes during the recovery period until he was sitting in sternal recumbency and his temperature had returned to within the normal parameters of 38° - 38.5°C.[18]

A registered veterinary nurse was present continually throughout the recovery period.

In an attempt to prevent postoperative seizures, a single dose of phenobarbitone (Phenobarbital, Martindale Pharmaceuticals) 5mg/kg was administered slowly IV on recovery. Despite this, as a consequence of its high toxicity, the animal developed hepatic encephalopathy causing seizures which were managed medically.

IVFT was continued at a rate of 4ml/kg/day until the animal had fully regained consciousness and was consuming food and water voluntarily. This rate was then reduced to 2 ml/kg/day until the following morning, when IVFT was discontinued.

Methadone was administered at a rate of 0.2 mg/kg every four to six hours as required – IM until the following morning. The patient was then switched on to buprenorphine (Vetergesic, Alstoe Animal Health) at a rate of 0.005 mg/kg every eight hours IM/IV.

Antibiosis continued in the form of oral amoxicillin clavulanate. Lactulose and diet modification was continued as prior to surgery. Phenobarbital (Epiphen, TEVA) was introduced orally at a rate of 15mg (¼ of a tablet in the morning and ½ a tablet in the evening) following postoperative seizure activity.

The patient was discharged from the hospital four days after surgery having made an acceptable recovery.

Table 4. Postoperative parameters

Rectal temperature	33°C
Pulse rate	144 beats per minute
Respiratory rate	16 beats per minute
Mucous membranes	Pale pink and dry
Capillary refill time	1.5 seconds
Blood pressure	140mmHg
Blood glucose	6.8mmol / l

Discussion

Animals with PSSs have an underdeveloped liver with abnormal circulation, which may result in altered uptake, metabolism and elimination of drugs, with variable consequences.[5, 8] These patients, particularly the young, therefore, often have an exaggerated response to centrally acting drugs and are readily sedated with low opioid doses without the administration of a sedative drug.[5]

A low plasma protein concentration means that the volume distribution of drugs that bind to the albumin is reduced, leading to a relative drug overdose.[10] As a consequence of a reduction in liver function, drugs that are metabolised by the liver – or are highly protein bound – should be avoided, or reduced amounts should be used.

These drugs include phenothiazine tranquillisers such as acepromazine (Calmivet, Vetoquinol), with the consequence of a methadone-only premedication being administered and benzodiazepines such as diazepam, as previously discussed in the above case study.[8]

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