

# Long-Term Outcome After Surgical Ligation for Treatment of Congenital Portosystemic Shunts in Dogs.

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## Abstract

**Objective:** To report long-term clinical outcome and dissolution of ammonium urate bladder uroliths and crystalluria in dogs treated for congenital portosystemic shunting (CPSS) with complete or partial ligation and to develop a scoring system to determine persistent or recurrent shunting.

**Study Design:** Retrospective.

**Animals:** Dogs (n=167) with surgically attenuated CPSS, intra- or extrahepatic.

**Methods:** Review of medical records of dogs with CPSS treated by ligation. Long-term information was obtained by telephone interviews with veterinarians and questionnaires sent to owners. A randomized group of 21 dogs came back for check-up to determine persistent or recurrent shunting.

**Results:** Preoperative results showed that ammonia metabolism was abnormal in all dogs in which fasting ammonia was determined or an ammonia tolerance test was performed. In most dogs the shunt was partially ligated. Mortality postoperatively (< 1 month) was 11%. At routine check-ups all dogs improved, only 8% still showed signs of HE. After surgery in 26% dogs with PC, 30% in PAZ and 66% dogs with intrahepatic CPSS, there was evidence of persistent shunting by abnormal ammonia metabolism. Urine samples were only tested when urological signs were seen, in 68% of the dogs bladder uroliths dissolved. A concept of a PSS scoring system was developed. Preoperative a PSS score of 43 (SD 27) and postoperative a PSS score of 11 (SD 13) was calculated. The improvement was significant ( $P < 0.001$ ). Long-term check-up showed in 58% of the dogs no shunting, in 16% asymptomatic shunting and in 26% symptomatic shunting.

**Conclusion:** Shunt attenuation by ligation is an effective technique to improve the quality of life of dogs with CPSS. Bladder uroliths dissolved in most patients. The concept of the PSS scoring system developed shows a promising additional method for diagnosing clinical PSS.

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## Introduction

A portosystemic shunt (PSS) is an anomalous vein that allows portal blood to drain into the systemic circulation. In case of PSS blood does not flow through the hepatic sinusoids. A PSS can be congenital or acquired. Acquired shunting develops because of portal hypertension so collaterals are formed to maintain normal portal pressure. A congenital portosystemic shunt (CPSS) is an anomalous vessel that is present without portal hypertension. (Szatmari, et al., 2004) CPSS can be classified in intra- and extrahepatic shunts. Extrahepatic CPSS are more common than intrahepatic CPSS. Small-breed dogs are predisposed for extrahepatic CPSS and large-breed dogs are predisposed for intrahepatic CPSS. An intrahepatic shunt is a direct continuation of a trunk of the portal vein, it can be a left, right or central intrahepatic shunt.

The ductus venosus, a normal embryologic shunt, is necessary for bypassing the umbilical blood along the liver, directly to the heart of the fetus. After birth the ductus venosus closes, so portal blood from the gastrointestinal tract and splanchnic drainage flows through the intrahepatic portal capillary system and liver parenchyma. In case of a left intrahepatic CPSS, the ductus venosus does not close after birth, so the blood still bypasses the liver, and the hepatic metabolism is decreased. In an extrahepatic shunt a connection between the embryonic vitelline veins and the cardinal veins is made by the shunt. This is not a 'normal' functional shunt in the embryo and therefore it must be a developmental anomaly. (Berent & Tobias, 2009; van den Bossche et al., 2012; van Steenbeek et al., 2013) Extrahepatic shunts can be classified in the more common portocaval (PC) and less

common portoazygos (PAZ) shunts. Blood is drained from the portal system to the vena cava or the azygos vein, in which there are many anatomical variations. When a CPSS enters the thorax, it is usually assumed that it terminates in the azygos vein, however with ultrasonography it is not possible to visualize. (Szatmári, et al., 2004)

The difference between the resistance of flow through the shunt and through the normal hepatic circulation results in a variable percentage of portal venous blood bypassing the normal intrahepatic circulation. The liver normally receives 25% of the total cardiac output, with 75% coming from the portal vein and 25% from the hepatic artery. Portosystemic shunting results in less nutrients and portal hepatotrophic factors in the liver. These factors are necessary for liver growth, structure maintenance and glycogen storage. CPSS in dogs lead to hepatic atresia, hypoplasia and dysfunction. (Holt, 1994; McQuaid, 2005) Most of the dogs with CPSS have a small liver, sometimes parts of the stomach are in direct contact with the diaphragm. The average liver size of a PSS dog is approximately 47% of the normal hepatic size. (Szatmári et al., 2004; Washizu et al., 2004)

### ***Clinical Signs***

Clinical signs that are often reported are associated with hepatic encephalopathy (HE) and decreased liver function. This includes seizures, vomiting, anorexia, ascites and dysuria. Signs are mostly episodic, and often related to a meal. Also drug intolerance, for example to sedatives or anesthetics, can be noted. Poor growth and weight loss, anorexia, ascites and polyuria and polydipsia are other common signs.

The accumulation of ammonia, certain amino acids and other protein catabolism products play an important role in the development of clinical signs. Ammonia is generated by deamination of proteins in the systemic circulation, and by bacteria in the gastrointestinal tract. Usually the liver converts ammonia to glutamine or urea. In animals with CPSS the liver cannot metabolize the ammonia directly in the portal blood, so ammonia concentrations increase in the systemic circulation, crossing the blood brain barrier and causing HE signs. (McQuaid, 2005)

Polyuria and polydipsia are mostly caused by deranged neuroendocrine functions. (van den Ingh, T. S., Rothuizen, & Meyer, 1995)

Biochemical findings can be hyperammonaemia, increased serum bile acids, hypoalbuminemia, anemia, prolonged coagulation, decreased gluconeogenesis and ammonium biurate crystalluria. (Berent & Tobias, 2009; Lee et al., 2011; McQuaid, 2005)

Most dogs with PAZ CPSS usually have milder clinical signs and larger liver volumes than dogs with portocaval shunts, probably because of intermittent occlusion of the shunt by diaphragm compression during respiration or gastric distention after eating. (Sura, et al., 2007)

### ***Urolithiasis***

Ingested proteins and endogen protein turnover are sources of purines, which are metabolized to uric acid. In most mammals uric acid is converted to allantoin, which is more soluble. Excreted uric acid may complex with various cations, forming urate salts. There is a high incidence of ammonium urate -a salt of uric acid- uroliths in dogs with portal vascular anomalies like CPSS. Because the blood bypasses the liver and hepatic function is reduced, there is less conversion of uric acid to allantoin and reduced conversion of ammonia into urea. Ammonium urate is the most common form of naturally occurring purine uroliths observed in dogs. They are typically multiple, small, smooth, hard, round or ovoid stones with a brown-green color. In case of uroliths caused by CPSS there is little or no difference in occurrence between male and female dogs. (Bartges et al., 1999; Kruger & Osborne, 1986; McCue, et al., 2009)

Concentration of calculogenic substances is dependent on urine volume. Because commercial dry foods are associated with lower urine production, it is a risk factor for urate urolith formation. Also high protein containing foods result in an increased risk, because they usually contain a high purine concentration. In dogs with PSS, the degree of urine saturation with purines is related to the degree of vascular shunting. Urine acidity is also a risk factor for urate crystals and uroliths, because the solubility of most purines is pH dependent. However in contrast to uric acid, ammonium urate solubility is less affected by

pH. (Bartges et al., 1999; Kruger & Osborne, 1986)

Urate uroliths can lead to pollakisuria or hematuria, but dogs can also be asymptomatic. If the uroliths are small enough, they pass through the urethra. In male dogs they often become lodged behind the os penis. When urethral obstruction is present, signs of obstructive uropathy can be seen, like dysuria, anorexia, vomiting, depression or even collapse and shock. (Bartges et al., 1999) Also hydronephros and hydroureter have been seen as a result of a ureter obstruction. (Silva, et al., 1990)

Not all dogs with CPSS develop ammonium urate urolithiasis. Other factors responsible for urate urolithiasis formation are not clear yet. (Bartges et al., 1999)

Recurrence of urate uroliths may be influenced by different factors, the persistence of underlying causes (like PSS), incomplete removal of stones at the time of surgery or persistence of bacterial infection with urease-producing bacteria. (Bartges et al., 1999)

### **Diagnosis**

The diagnosis of CPSS is made on clinical signs of HE, biochemical profiles, and medical imaging. PC CPSS is mostly diagnosed at a younger age than PAZ CPSS, because most dogs with PAZ CPSS have less clinical signs. (van den Bossche et al., 2012; Washizu et al., 2004). Detection of high blood ammonia concentration can help to confirm that the signs are related to hyperammonaemia. Also high bile acids concentrations are suggestive for PSS. (Szatmári et al., 2004)

Fasting ammonia level measurements require one blood sample. Because of the instability of ammonia in vitro, the test has to be run immediately or the sample should be kept on ice. (Winkler, et al., 2003) Basal ammonia concentrations may be low or normal because of prolonged fasting, low-protein diet and use of lactulose. (Berent & Tobias, 2009)

A liver function test such as an ammonia tolerance test (ATT) can be done when basal ammonia in fasting animals is not conclusive whether there is shunting or not. Ammonium chloride is administered rectally, and dogs with PSS show an abnormal ATT, which means that ammonia levels will be more than twice as high at 20 and/or 40 minutes. (Berent & Tobias, 2009)

Even a small liver in case of CPSS has enough capacity, to produce sufficient bile acids. After a meal the bile acids are released in the intestine and via the portal system reabsorbed in the liver. In case of CPSS not all bile acids will be extracted from the portal blood at once, so systemic bile acid concentrations will be elevated. Paired bile acid measurements are performed as a screening for PSS. First a fasted blood sample is drawn. Two hours after providing a small meal, another blood sample is taken. An increase of bile acids over 40  $\mu\text{mol/L}$  is suggestive for CPSS. (Berent & Tobias, 2009; Winkler et al., 2003)

In medical imaging the most commonly used techniques are doppler ultrasonography and computed tomography for detailed characterization of vascular anatomy.

Ultrasonography is a non-invasive technique, which can detect shunting, and the direction (hepatopetal or hepatofugal) and velocity of the blood flow. It can also identify urinary calculi. The sensitivity of abdominal ultrasonography for detecting CPSS was found to range between 47-95%, and specificity was ranging from 67-100% by Kim et al. (2013). This wide variation depends for example on movement of the patient, full gastrointestinal tract, technology and experience of the radiologist. Abdominal ultrasonography may detect the existence of an anomalous vessel, the origin and insertion however are difficult to detect. Kim et al. (2013) found an 80% match in origin and 60% match in insertion between ultrasonography and surgery.

Computed tomography (CT) allows exact delineation of shunting vessels, Kim et al. (2013) found a 94% match between CT and surgical findings. Another benefit is that it is possible to determine vasculature outside of the abdomen, even with the insertion of a PAZ CPSS in the systemic vasculature in the thorax. However additional or prolonged episode of general anesthesia is necessary, and it is quite expensive. (Kim et al., 2013; Szatmari et al., 2004)

Mesenteric portography has been used in the past to determinate between congenital or acquired shunting and to evaluate intrahepatic portal branching. A jejunal or mesenteric vein is catheterized, so the route of toxin-rich blood can be followed. However this requires general anaesthesia and a laparotomy. Both procedures have an increased risk in dogs with hepatic insufficiency. Scintigraphy can also detect

PSS, however this technique does not distinguish between congenital and acquired shunting. (Szatmari et al., 2004) Another disadvantage is that it requires sedation and increased hospitalization because of radioactive waste excretion. (Winkler et al., 2003)

### ***Medical Management***

Medical management is necessary to reduce the HE signs caused by hyperammonaemia in dogs with CPSS.

Diet modification is most important in medical management. There must be adequate caloric intake, to keep the glucose level high enough, because dogs with CPSS have less glycogen storage. This intake is also necessary to prevent body own protein breakdown, so ammonia concentrations are kept low. It is necessary to have some high value protein in the diet, because most CPSS patients are already hypo-proteinemic. (McQuaid, 2005) Because long-term ultralow-protein diets have been associated with dilated cardiomyopathy in English bulldogs and Dalmatians, there has to be adequate protein in the diet. (McCue et al., 2009)

Lactulose can be used as an additional therapy. It is a non-absorbable disaccharide that is hydrolyzed by colonic bacteria to lactic acid and acetic acid. It lowers the colonic pH, which makes survival of bacteria harder, so less ammonia is produced. It also ionizes neutral ammonia to ammonium, which blocks its absorption. (Holt, 1994; McQuaid, 2005)

Oral antibiotics, such as neomycine, metronidazole and amoxicilline, were given in the past to alter the gastrointestinal bacterial flora, which decreases the amount of ammonia production. (McQuaid, 2005; Phongsamran, et al., 2010)

Medical dissolution of ammonium urate uroliths includes a combination of calculolytic diets, administration of xanthine oxidase inhibitors, urine alkalization, control of urinary tract infections and increasing urine formation. A purine-restricted alkalizing diet that does not contain supplemental sodium, results in reduction in urinary uric acid and ammonia excretion. Xanthine oxidase inhibitors decrease the production of uric acid, so less uroliths are formed. However this should not be used in dogs with CPSS, because the decreased hepatic metabolism may result in prolonged half-life and side effects. Alkalizing

the urine prevents formation of acid metabolites. Oral sodium bicarbonate or potassium citrate administration can be of value in preventing acid metabolites from increasing renal tubular production of ammonia. However alkalizing the urine reduces the solubility of already insoluble ammonium urate crystals.

Bacterial urinary tract infections should be prevented, eradicated or controlled because they may increase the problem of urolith formation. Urease producing microbes may produce ammonium ions, which can lead to formation of insoluble ammonium urate uroliths or struvite.

Increasing urine volume lead to decreasing uric acid and ammonium concentrations, which lead to less crystal formation. However the best treatment for dogs with CPSS is a low purine diet, resulting in decreased production of ammonium from dietary protein derived urea and reduced formation of dietary purine-derived uric acid. Problems of low protein diets may be expected in immature dogs with urate uroliths. These dogs require greater quantities of dietary protein and purines. (Bartges et al., 1999; Kruger & Osborne, 1986)

Medical treatments are used to manage clinical signs of CPSS. However they do not resolve the cause of the disease. Many patients, mostly with intrahepatic CPSS, have persistent or recurrent signs on medical management alone. To cure CPSS, surgery is necessary to redirect the blood through the liver by attenuating the shunt. (Berent & Tobias, 2009; Lee et al., 2011) Surgical treatment of intrahepatic CPSS involves attenuating the portal branch supplying the CPSS, the CPSS itself or the draining hepatic vein. (Mehl et al., 2007)

### ***Surgery***

Patients with CPSS who are cachectic or encephalopathic have to be managed with former therapies, until they can tolerate stress of anesthesia and surgery. (Berent & Tobias, 2009) Managing dogs with CPSS also includes correction of fluid deficits and electrolyte and acid-base; blood glucose concentration normalization and treating continuing seizures. (Holt, 1994)

The goal of surgery is to attenuate the shunt to improve portal perfusion and maximize hepatic function. When surgery is possible, CPSS can be completely or partially ligated with nonabsorbable ligatures or gradually

attenuated with cellophane band, an ameroid constrictor, hydraulic occluder or transvenous coil embolisation. (Berent & Tobias, 2009)

In only a minority of dogs complete ligation of the CPSS is possible. The choice of complete or partial closure is made during the surgical procedure. The rise of portal tension during closure defines if complete closure is possible or not. Visual inspection for cyanosis or pallor of the intestines and the pancreas, increased pulse and decreased mean arterial pressure are important indicators for portal hypertension. It is even possible to measure and evaluate changes in portal and central venous pressures during attenuation.

Even when total occlusion of the shunt is not possible, clinical signs can cease. If the flow through the shunt was hepatofugal before surgery, partial occlusion can tend to change the flow cranial to the origin of the shunt in hepatopetal. This means that blood from all portal tributaries flows via the portal branches to the hepatic sinusoids. This improvement of hepatic circulation results in enlargement of lobes of the liver and blood flow in right and left portal branches. When this happens further attenuation is not necessary, because it does not reduce the shunting of blood. However it can increase the level of portal hypertension because a larger proportion of blood is forced through a smaller vein. (Swalec & Smeak, 1990; Szatmari et al., 2004) Because of the hepatopetal flow and liver growth, the liver is able to clear the blood from toxins, so there is less ammonia in the blood, and no or less clinical signs will appear. In this case there is a functional closure of the shunt.

In most dogs clinical signs disappear after attenuation, even despite persistent shunting. However clinical signs do not disappear in all dogs, which may depend on the type of shunt and surgery technique. (Berent & Tobias, 2009)

Hyperammonaemia can still persist in dogs with persistent shunting through the original, partly closed shunt or in dogs with acquired collaterals. (Szatmari et al., 2004)

When only partial ligation is possible during the first attempt to close a shunt, it is possible to perform a second surgery for complete ligation. In most cases however complete closure is probably not necessary to obtain a good long-term outcome. As mentioned before, functional closure often has been seen

after partial ligation and local inflammation around the ligature may even further reduce the diameter of the shunt after the first surgery. A second surgery is only an option when there is still flow through the original congenital shunt and there are no acquired collaterals. Ligation forces blood flow through the portal branches, which are mostly hypoplastic in CPSS dogs, resulting in portal hypertension. Partial ligation at the first procedure allows the portal system to adapt, so it might be possible to ligate the shunt more or even completely in a second surgery. (Berent & Tobias, 2009; Szatmari et al., 2004)

Hottinger et al. (1995) showed that a partial ligated CPSS is easily ligated completely in a second surgery, with minimal increases in portal pressure. Considering the more frequent recurrence of clinical signs, and patient morbidity after partial ligation Hottinger et al. (1995) recommended complete ligation in a second surgery for all dogs with partial ligation after the first surgery.

At Utrecht University a second surgery is only recommended when there are still considerable clinical signs caused by persistent portosystemic shunting through a single CPSS, and no collaterals were formed.

For gradual attenuation cellophane bands can be used. A strip of cellophane is folded during surgery to form a 3-layered, flexible band. The band is held into place with surgical clips. This band causes a fibrous tissue reaction so the shunt gradually closes. (Berent & Tobias, 2009)

A different method for gradual attenuation is an ameroid constrictor, which has a casein ring with stainless steel surrounding it. Casein swells when absorbing fluids, which reduces the diameter. It also stimulates tissue reaction which results in gradual shunt occlusion. Early rapid expansion occurs during the first 14 days, in the next two months slow expansion occurs. In some cases extrahepatic CPSS attenuation with ameroid constrictors led to multiple acquired extrahepatic PSS as a severe complication. In these cases the occlusion happens within 7 days after implantation, or theoretically it could be because of acute vessel kinking after the dog changes position. (Berent & Tobias, 2009; Hurn & Edwards, 2003; Winkler et al., 2003)

Hydraulic occluders can also be used to gradually attenuate CPSS. It consists of a silicone and polyester cuff, connected by tubing to a vascular access port. Every two weeks a small amount of sterile saline is injected into the port, which inflates the cuff. The shunt closes in six to eight weeks. (Berent & Tobias, 2009)

Also transvenous coil embolisation is a possible surgical technique to attenuate shunts. Through a venous catheter an elastic embolization coil is placed into the shunt, which gradually narrows the lumen of the shunt. Weisse et al. (2014) showed that it is an effective treatment for intrahepatic CPSS with a lower morbidity and mortality rate and equal long-term outcome compared with traditional open surgery techniques. (Bussadori et al., 2008; Partington, Partington, Biller, & Toshach, 1993; Weisse, et al., 2014)

All different shunt attenuation techniques have been described for extrahepatic and intrahepatic CPSS, however most techniques are more difficult and less safe to use in intrahepatic CPSS.

Attenuation of CPSS may reduce the presence of uroliths. Bartges et al. (1999) observed substantial reduction of uric acid concentration in urine in a 3-month old Miniature Schnauzer. If diet or shunt attenuation do not dissolve the uroliths, or when CPSS cannot be attenuated and there are signs of urinary tract disease, surgical removal of the uroliths should be advised. (Bartges et al., 1999)

### ***Prognosis and Complications***

Anatomy of CPSS is important for the long-term outcome. Dogs with portoazygos CPSS are more likely to tolerate total ligation than portocaval shunts. This improves their long-term prognosis. (Sura et al., 2007)

An intrahepatic shunt is more difficult to isolate and ligate than an extrahepatic shunt, this leads to a more guarded prognosis. (Winkler et al., 2003)

For dogs with extrahepatic CPSS, portal development is important for short-term clinical outcome, however it is not related with long-term outcome, because the portal system and liver can adapt. (Kummeling, Van Sluijs, & Rothuizen, 2004) Also direction of flow in the portal vein cranial to the origin of CPSS is important. The prognosis is excellent for dogs when the flow after surgery is in hepatopetal

direction, compared to dogs with hepatofugal flow. (Szatmári et al., 2004)

The extrahepatic portal development is not associated with short-term outcome for dogs with intrahepatic CPSS, because the cranial part of the portal vein and the shunt are the same vessel. (Kummeling et al., 2004)

Causes of death during and after surgery of dogs with CPSS are portal hypertension, haemorrhage, postoperative seizures, euthanasia because of portal hypoplasia or atresia, or the inability to ligate the shunt. (Lipscomb, Jones, & Brockman, 2007)

Primary hypoplasia of the portal vein is a disorder characterized by hypoplasia of the intrahepatic portal branches and sometimes even the extrahepatic portal vein. Secondary portal vein hypoplasia can be found when the blood supply to the liver from the portal vein is reduced, for example with CPSS. Most CPSS have a diameter equal to, or larger than the diameter of the portal vein segment caudal to the shunt. This offers a lower resistance route of flow though the shunt, resulting in hypoperfusion of the portal vein cranial to the shunt origin and intrahepatic portal branches. This is mostly seen in dogs with intrahepatic CPSS and in dogs with extrahepatic CPSS in which the portal vein cranial to the shunt origin is much thinner. (Szatmári et al., 2004) After attenuation of CPSS, portal hypertension may develop. The degree of portal hypertension depends on the severity of hypoplasia of the portal vein and degree of attenuation of the shunt. It can lead to pain, ileus, ascites, decreased central venous pressure and gastrointestinal hemorrhage. In case of severe complications the ligature should be removed. Persistent portal hypertension might lead to acquired collaterals, depending on the degree of hypoplasia of the portal circulation and severity of hypertension after shunt attenuation. That is why in these cases gradual or partial attenuation is preferred. Severe portal hypertension can result in hypovolemia, hypoproteinemia, decreased tissue perfusion, sepsis or shock and death. (Berent & Tobias, 2009; Holt, 1994; Szatmári et al., 2004)

Another complication associated with surgery is hemorrhage because of coagulopathy, which can be fatal despite early treatment. Clotting times are prolonged in many dogs with CPSS, and have to be checked before surgery. In case

of CPSS, APTT is often prolonged and PT is usually normal. In dogs with CPSS, abnormalities are found in the plasma concentrations of multiple clotting factors and a surgical procedure will decrease the concentrations even more. (Kummeling, et al., 2006; McQuaid, 2005)

Central nervous system dysfunction (post ligation seizure complex) is a complication following attenuation of CPSS, mostly 12 hours to 3 days after surgery with a poor prognosis. Most often it is not associated with metabolic encephalopathy. Signs reported are progressive ataxia, behavioral changes, generalized motor seizures or even status epilepticus. Brain pathology in dogs that died or were euthanized during postoperative seizures showed ischaemic and hypoxic neuronal necrosis. (Yool & Kirby, 2002)

***Aims of this study***

At this moment a randomized, multicenter study is conducted to prospectively compare partial attenuation of extrahepatic CPSS in dogs by ligation and by cellophane banding, with or without a second surgical intervention to perform complete closure by ligation. The clinical performance of dogs is evaluated by quality of life questionnaires that were developed by the RVC, London. Studies that evaluate outcome in animals after treatment need a practical, reliable and simple system to evaluate clinical performance and to establish the diagnosis of clinical portosystemic shunting. This retrospective study was performed to evaluate clinical signs in relation with the evidence of residual portosystemic shunting in dogs with shunts after ligation. The recovery of the CPSS dogs was described and classified. Afterwards a scoring system was developed for evaluating clinical performance in future study populations.

Dogs with CPSS are predisposed to the formation of ammonium urate stones in the urinary tract and sometimes calculi are noticed in the urinary bladder during preoperative ultrasonography. Normally these calculi are not removed during PSS surgery, because of the prolonged surgery time, extra trauma caused by a longer abdominal incision and a cystotomy, and the risk of perioperative complications in these patients. We assume that these calculi, if they are ammonium urate, will dissolve spontaneously after plasma ammonia decreases postoperatively and will

not lead to clinical signs after successful surgery, but it is not clear if this really happens. In this retrospective study, our second purpose is to screen ultrasonography reports before and after surgery and to score the presence of bladder stones. The results of the screening of the ultrasonographic reports will be compared with the clinical performance after surgery.

The ultrasonography after surgery was also performed to determine if there was still shunting. After surgery persistent shunting was also determined based on ammonia metabolism. These results were used to calculate the sensitivity and specificity of diagnosing persistent or recurrent shunting by ultrasonography.

**Material and methods**

***Study Population***

Dogs surgically treated for a CPSS by ligation at Utrecht University between 2003 and November 2013 were included. All different anatomical CPSS, intrahepatic and extrahepatic, were included.

Only dogs were studied when shunt attenuation was possible during surgery, if ligation was not possible the dog was not a study case.

***Medical Records Review***

Data extracted from the medical records from the UKG clinical database (Vetware) included breed, date of birth, age at surgery, sex and management before and after surgery.

Clinical signs of HE were classified before and after surgery following the system of Rothuizen (1993), see table 1.

*Table 1: HE classification*

<b>Class HE</b>	<b>Symptoms</b>
0	No clinical signs
1	Sopor (lazy, indolent), personality changes, polyuria
2	Ataxia, desorientation, compulsive movements, apparent blindness, ptyalism
3	Stupor, excessive ptyalism, incidental seizures
4	Coma, non-responsiveness

*HE signs classification system by Rothuizen (1993).*

Preoperative bile acid and ammonia concentrations, and if performed, ammonia tolerance test results were recorded. If urine had been analyzed, pre- and postoperative,

presence of ammonium urate crystals was noted. When there was an ultrasonography report, presence of uroliths were reported, pre- and postoperative.

Intraoperative information extracted from the medical records included anatomical location of the shunt (intra- or extrahepatic, and portocaval or portoazygos), and the degree of shunt attenuation.

Postoperative data recorded from the medical records included short-term postoperative deaths (<1 week) as well as 4-40 week postoperative ammonia tolerance testing, fasting bile acid concentration and ultrasonography results.

### **Follow Up**

Outcome information was obtained by telephone interview with the referring veterinarian whether the dog was still alive, dead or euthanized. When the dog died or was euthanized, the owner was asked when the event occurred and if circumstances were known.

Owners of dogs still alive, or dogs that died or were euthanized at least one year after surgery were asked to participate in this study.

The questionnaire was developed by the RVC, London, translated into Dutch and consisted questions with regard to the preoperative and the postoperative period. Both parts of the questionnaire included questions about presence of clinical signs related to hepatic encephalopathy, gastrointestinal signs and signs concerning the urinary tract, frequency of these clinical signs and how severely these impacted the life of their dog. The questionnaires also evaluated medical management, activity level, body condition, possible relapse, how willing their dog was to play and interact with people and other dogs, and their quality of life. (Quality of Life Questionnaire is found in appendix 2a-c).

A randomized group of dogs came back to Utrecht University for an extra check-up. Anamnesis and physical examination were performed, after which ammonia and bile acid concentration were determined in a fasting blood sample. When ammonia levels were below 100 µmol/L, an ammonia tolerance test was performed. Forty minutes (t=40) after rectal administration of ammonia chloride (2ml/kg bodyweight) another blood sample was drawn to measure ammonia concentration. (Master thesis B.G.J. Bolscher) The test was performed to determine if there was persistent

or recurrent shunting. Owners collected an urine sample at home just before their visit, which was used to analyse presence of ammonium urate crystals.

### **Scoring system**

A concept of the scoring system for the clinical performance of the dogs, was developed based on the questionnaires, assigning each dog a PSS score. Owners staged the symptoms, ranging from 0 to 4 (0 being never, 1 being less than once a month, 2 being monthly, 3 being weekly and 4 being daily). The PSS score was calculated by the sum of scores of 19 different clinical signs. Seventeen signs were staged and multiplied with factor 1, 2 or 3 based on the clinical significance of the signs, see table 2.

*Table 2: Scoring of symptoms from questionnaire*

<b>Class</b>	<b>Clinical signs</b>	<b>Factor</b>
Class 1	Seizures	3
Class 2	Head pressing, ataxia, disorientation, aggression, salivation, collapse, unresponsive, apparent blindness, fatigue/ weakness, circling	2
Class 3	Vomiting, diarrhea, polyuria, decreased appetite, hematuria, dysuria	1

*Seventeen signs are staged and multiplied by factor 1-3, based on the clinical significance.*

Presence of urolithiasis or urethral obstruction was awarded with 2 additional points. Dogs with retarded growth were given 4 additional points, when this was doubtful 2 points were added.

The owners also scored the impact of the specific signs on their dog's quality of life.

### **Statistical Analyses**

Sensitivity and specificity for identifying persistence or recurrent shunting with abdominal ultrasonography after surgery were calculated. To include cases for these calculations, postoperative ultrasonography had to be performed and determined if the shunt is open or closed and persistence of shunting had to be known based on ammonia metabolism.

The Wilcoxon Signed Rank test was performed to test the significance of the PSS score before and after surgery.



## Results

### Medical records review (see appendix 1a-d)

#### Study Population

167 dogs were included in this study. These dogs underwent surgery between 2003 and November 2013. 129 (77%) with extrahepatic CPSS and 38 (23%) with intrahepatic CPSS. Study population is described in table 3.

Table 3: Study population

	Number of patients	Median age (range)	Sex % M / F
PC	92 (55%)	10.5 (4- 67)	41 / 59
PAZ	37 (22%)	20 (3-78)	30 / 70
IH	38 (23%)	8.5 (4-54)	53 / 47

The study population was described in 3 anatomical shunt variations. The number of patients was also given in percentage of the 167 patients. The median age at surgery was given in months, with the range also in months. The sex distribution was only given in percentage.

Breed distribution in dogs with extrahepatic CPSS included 26 different breeds, which included 19 (15%) Maltese dogs (17 PC, 2 PAZ), 17 (13%) Jack Russell terriers (13 PC, 4 PAZ), 13 (10%) Yorkshire terriers (9 PC, 4 PAZ), 11 (9%) Cairn terriers (10 PC, 1 PAZ), 9 (7%) Dachshunds (6 PC, 3 PAZ), 6 (4.6%) Miniature schnauzers (4 PC, 2 PAZ), 6 (4.6%) Chihuahuas (3 PC, 3 PAZ), 5 (3.9%) Pugs (4 PC, 1 PAZ), 4 (3.1%) West Highland White terrier (3 PC, 2 PAZ), 3 (2.3%) Norfolk terriers (3 PAZ), 3 (2.3%) Shih Tzus (2 PC, 1 PAZ), 19 (15%) dogs from other breeds (10 PC, 9 PAZ) and 14 (11%) dogs were crossbreeds (11 PC, 3 PAZ). Breed distribution in dogs with intrahepatic CPSS counted 18 different breeds, including 7 (18%) Golden retrievers, 5 (13%) Bernese Mountain dogs, 3 (7.9%) Labrador retrievers, 3 (7.9%) Cane Corsos, 2 (5.3%) Irish Wolfhounds, 2 (5.3%) Bearded collies, 2 (5.3%) Shih Tzus, 11 (29%) dogs from other breeds and 3 (7.9%) dogs were crossbreeds.

#### Preoperative Data

Many dogs received medical management before they came to Utrecht University. In total 75 dogs received medical management, 38 (41%) dogs with PC, 14 (38%) dogs with PAZ and 23 (61%) with IH CPSS. Treatment included a low protein, kidney or liver diet, lactulose, antibiotics or a combination. Details are found in table 4.

Table 4: Number of dogs on medical management before surgery

	Diet	Lact.	Ab.	Diet/lact	Diet/ab	Diet/ab/lact
PC	17	7	1	13	-	-
PAZ	5	1	-	6	1	1
IH	10	1	-	10	-	2

The diet used for management was low protein, kidney or liver diet. Also lactulose (lact) was used in many cases. Some dogs were treated with antibiotics (ab). A lot of dogs were treated with a combination, mostly a diet with lactulose.

Clinical signs of HE in dogs were classified by table 1. 83 (90%) dogs with PC, 31 (84%) dogs with PAZ and 36 (95%) dogs with IH CPSS showed signs of HE. The number of dogs showing these clinical signs are detailed in table 5.

Table 5: HE classification before surgery

	0	1	2	3	4
PC	9 (10%)	8 (8.7%)	62 (67%)	12 (13%)	1 (1.1%)
PAZ	6 (16%)	14 (38%)	13 (35%)	4 (11%)	-
IH	2 (5.3%)	12 (32%)	22 (58%)	2 (5.3%)	-

The clinical signs of HE were classified by table 1 in number of dogs and percentage. In which class 0 means no HE signs, class 1 moderate signs going up in class 2, 3 and 4 to more severe signs.

Clinical signs of pollakisuria and hematuria, which are the most obvious signs of crystalluria, were present in 17 dogs (19%) with PC, 11 dogs (30%) with PAZ and 6 dogs (16%) with intrahepatic CPSS. In 54 dogs bladder uroliths were found by ultrasonography. (See table 6a)

Table 6a: Urological signs and uroliths before surgery

	Hematuria	Pollakisuria	Both	Uroliths
PC	7	5	5	28 (30%)
PAZ	4	3	4	15 (41%)
IH	1	5	-	11 (29%)

The number of dogs showing urological signs, hematuria, pollakisuria or both. Also the number and percentage (related to the anatomical subgroup) of dogs found positive for bladder uroliths by ultrasonography.

Of 75 dogs urine was analyzed for ammonium urate crystals. In 44 urine samples ammonium urate crystals were found. Twenty dogs with crystalluria showed urological signs. Signs taken into account were hematuria and pollakisuria (See table 6b)

Table 6b: Crystalluria before surgery

	Crystalluria/ samples	Clinical signs
<b>PC</b>	25 / 42 (60%)	11 (44%)
<b>PAZ</b>	15 / 21 (71%)	8 (53%)
<b>IH</b>	4 / 12 (33%)	1 (25%)

Urine samples were not tested for ammonium urate crystals in all dogs, the number and percentage of positive samples were given. Also the number and percentage of dogs showing urological signs (hematuria and pollakisuria) were reported.

Seven dogs (7.6%) with PC CPSS came to Utrecht University for shunt screening, so these dogs had no clinical signs. In case of PAZ CPSS 2 dogs (5.4%) and also for intrahepatic CPSS 2 dogs (5.3%) were diagnosed by shunt screening.

Fasting ammonia concentrations (reference interval, 24-45  $\mu\text{mol/L}$ ) were available for 156 dogs (93%). In total 143 dogs (92%) had a concentration above reference. For the different groups the median value and range are found in table 7. In total 15 dogs had a value over 286  $\mu\text{mol/L}$ , in these cases 286  $\mu\text{mol/L}$  was entered for calculations of the median value, because the true value is not known.

Table 7: Fasting ammonia concentration before surgery

	Above reference/ samples	Median value $\mu\text{mol/L}$	Range $\mu\text{mol/L}$	Above 286 $\mu\text{mol/L}$
<b>PC</b>	79 / 86 (92%)	154	22 - >286	10 dogs
<b>PAZ</b>	31 / 36 (84%)	97	27 - >286	1 dog
<b>IH</b>	33 / 34 (97%)	163	44 - > 286	4 dogs

The number and percentage of dogs with fasting ammonia concentrations above reference. Reference interval: 24-45  $\mu\text{mol/L}$ . The median value and the range were given in  $\mu\text{mol/L}$ . When the concentration was >286  $\mu\text{mol/L}$ , 286 was entered for the median value.

In 11 dogs (12%) with PC CPSS an ammonia tolerance test was performed. All the tests were positive, which had more than threefold increase in ammonia concentration. Ten dogs (27%) with PAZ CPSS underwent an ammonia tolerance test, which were all positive. In dogs with intrahepatic shunting 3 dogs (7.9%) underwent an ammonia tolerance test, which were all positive.

Fasting bile acid concentrations (reference level, <10  $\mu\text{mol/L}$ ) were available for 108 dogs (65%). In 102 dogs (94%) the concentration was above reference. The median values and ranges for the different anatomical CPSS are found in table 8.

Table 8: Fasting bile acid concentration before surgery

	Above reference/ samples	Median value $\mu\text{mol/L}$	Range $\mu\text{mol/L}$
<b>PC</b>	55 / 57 (97%)	105	3 - 615
<b>PAZ</b>	29 / 30 (87%)	95	2- 465
<b>IH</b>	18 / 21 (86%)	35	2 - 380

The number of dogs with fasting bile acid concentrations above reference, also given in percentage. Reference level: <10  $\mu\text{mol/L}$ . The median value and the range were given in  $\mu\text{mol/L}$ .

#### Intraoperative Data

Of all 92 dogs with PC CPSS 70 dogs (76%) were partially ligated, 22 (24%) were completely ligated during surgery. One (4.5%) of the 22 dogs that were completely ligated was partially ligated during a first surgery. Of all 37 dogs with PAZ CPSS, 23 dogs (62%) were partially ligated, 13 (35%) completely and 1 (2.7%) is unknown. Of the 13 dogs of which the shunt was completely ligated, 1 dog (7.7%) was only partially ligated during the first surgery. Of all 38 dogs with intrahepatic CPSS 36 dogs (95%) were partially ligated and 2 (5.3%) were completely ligated. Of the 2 dogs that underwent complete ligation 1 was partially ligated during a first surgery.

#### Postoperative Data

Nineteen of 167 dogs (11%) died in the short-term (<1month) postoperative period (12 dogs (13%) with PC CPSS, 3 dogs (8.1%) with PAZ CPSS and 4 dogs (11%) with intrahepatic CPSS). Of 80 dogs with PC CPSS that survived over 1month, 75 (94%) came back for

check-up between 4 and 40 weeks postoperatively. Of 34 dogs with PAZ CPSS that survived over 1 month, 30 (88%) came back for check-up between 4 and 28 weeks postoperatively. Of 34 dogs with intrahepatic CPSS that survived over 1 month, 29 (85%) came back for check-up between 4 and 24 weeks after surgery.

After surgery 46 dogs (34%) were still receiving medical management at the time the dogs came back for check-up. These included 22 dogs with PC, 10 dogs with PAZ and 14 dogs with IH CPSS. The dogs received a low protein, kidney or liver diet or lactulose, or a combination of both.

Table 9: Medical management after surgery.

	Diet	Lactulose	Diet/ lact
PC	19	1	2
PAZ	9	-	1
IH	11	-	3

After surgery 34% of the dogs still received medical management. Only a low protein, kidney or liver diet, or lactulose or a combination (diet/lact) was given.

Clinical signs of hepatic encephalopathy were again classified following the system of Rothuizen (1993), see table 1. In total 13 dogs still showed signs of HE, only class 1 and 2 of signs were still seen. Three dogs (4%) with PC, 3 dogs (10%) with PAZ and 7 dogs (24%) with IH CPSS had remaining signs of HE. Ten of the dogs with remaining signs underwent partial ligation. Classification after surgery is detailed in table 10.

Table 10: HE classification after surgery

	0 c / p	1 c / p	2 c / p
PC	72 (96%) 14 / 58	2 (2.7%) 1 / 1	1 (1.3%) 0 / 1*
PAZ	27 (90%) 16 / 11	2 (6.7%) 1 / 1	1 (3.3%) 0 / 1
IH	22 (76%) 1 / 21	4 (14%) 1 / 3	3 (10%) 0 / 3

The clinical signs of HE were classified by table 1 in number of dogs and percentage. In which class 0 means no HE signs, class 1 moderate signs going up in class 2. No class 3 and 4 were seen after surgery. For all 3 groups of shunt variations in all classes was reported how many dogs underwent complete (c) or partial (p) ligation.

\* This dog was euthanized 3 months after surgery because of remaining signs.

Clinical signs of hematuria or pollakisuria after surgery were not reported with dogs with PC CPSS and intrahepatic CPSS. Two dogs (6.7%) with PAZ CPSS showed pollakisuria.

Urine analyses were also not routinely performed after surgery. Urine of 25 dogs was analyzed post operatively. Eight dogs were found positive on ammonium urate crystals (3 female and 5 male). Sixteen of these dogs had urine tested before and after surgery. Four dogs were positive on ammonium urate crystals pre- and postoperative, 2 dogs were found positive postoperative and were negative preoperative, and 2 dogs were found positive postoperative, their result preoperative was unknown. Details of urine sample results between the different anatomical shunts are found in table 11.

Table 11: Urine samples after surgery

	Crystals/ Samples	Pre/ Post	M/F	Results
PC	3 / 14 21%	7	2 / 1	2 + pre/post 1 - pre/+ post
PAZ	2 / 6 33%	6	1 / 1	2 + pre/post
IH	3 / 5 60%	3	2 / 1	1 - pre/+ post 2 ? pre/+ post

Urine samples were tested for ammonium urate crystals after surgery. The number found positive and total number of samples and the percentage are given. Only a couple of dogs were tested before and after surgery (pre/post). Reported are the results, with comparison between pre- and postoperative results; positive pre and postoperative (+ pre/post); negative pre and positive postoperative (- pre/+ post); not tested pre and positive postoperative (? pre/+ post).

Ultrasonography reported 14 dogs with urine bladder uroliths. Five dogs were not reported with uroliths before surgery. Of these 14 dogs only 9 dogs had uroliths before and after surgery. Preoperative 54 dogs were found positive with uroliths, so in 45 dogs the uroliths disappeared. However of these 45 dogs, 8 dogs died short-term postoperatively and 18 dogs underwent cystotomy. So in 28 dogs the uroliths could dissolve. In 19 dogs (68%) no uroliths were found post surgery. Details are found in table 12.

Table 12: Uroliths and dissolution after surgery

	Pre	Post	Died	Cystot.	Dissol.
PC	28	6	5	7	10
PAZ	15	1	2	8	4
IH	11	2	1	3	5

The number of dogs are given found positive on uroliths before, pre (table 6a) and after surgery, post (the dogs that were found positive after surgery but had no uroliths before surgery were left out). The number of dogs that died short-term postoperatively and the number of dogs that underwent cystotomy, are given. These numbers result in the number of dogs in which the uroliths dissolved.

Fasting ammonia concentrations (reference interval, 24-45  $\mu\text{mol/L}$ ) were available for 135 dogs. In 28 dogs (21%) the concentration was above reference. The median value and range for the three different shunt groups are found in table 13. Ten dogs were found with a concentration under 7  $\mu\text{mol/L}$ , in this case 7  $\mu\text{mol/L}$  was entered to calculate the median value. One dog was found with a concentration over 286  $\mu\text{mol/L}$ , 286  $\mu\text{mol/L}$  was entered for the median value.

Table 13: Fasting ammonia concentration after surgery

	Above reference/samples	Median value $\mu\text{mol/L}$	Range $\mu\text{mol/L}$	< 7 $\mu\text{mol/L}$ > 286 $\mu\text{mol/L}$
PC	10 / 77* (13%)	15	<7 - 282	7 dogs <7 $\mu\text{mol/L}$
PAZ	0 / 29 (0%)	15	<7 - 33	3 dog <7 $\mu\text{mol/L}$
IH	18 / 29 (62%)	63	12 – > 286	1 dog >286 $\mu\text{mol/L}$

The number and percentage of dogs with fasting ammonia concentrations above reference. Reference interval: 24-45  $\mu\text{mol/L}$ . The median value and the range were given in  $\mu\text{mol/L}$ . When the concentration was <7  $\mu\text{mol/L}$ , 7 was entered; when concentration was >286  $\mu\text{mol/L}$ , 286 was entered for the median value.

\* 75 dogs came back for check-up, 2 dogs were tested at home by a Master student of Veterinary medicine.

In 68 of 77 dogs (88%) with PC CPSS of which fasting ammonia concentration was determined, an ammonia tolerance test was performed. Of the 68 tests 19 (28%) were positive, which had more than twofold increase in ammonia concentration and a concentration over 45  $\mu\text{mol/L}$ . Twenty four of 30 dogs (80%) with PAZ CPSS that came back underwent an ammonia tolerance test, of which

9 (38%) were positive. In dogs with intrahepatic shunting 23 of 29 dogs (79%) that came back underwent an ammonia tolerance test, of which 14 (61%) were positive.

Fasting bile acid concentrations (reference level, <10  $\mu\text{mol/L}$ ) were available for 108 dogs. In 51 (47%) dogs the fasting bile acid concentration was found above reference. The median value and range are given per shunt group in table 14.

Table 14: Fasting bile acid concentration after surgery

	Above reference/samples	Median value $\mu\text{mol/L}$	Range $\mu\text{mol/L}$
PC	26 / 61 (43%)	8	0 – 440
PAZ	9 / 25 (36%)	7	1 – 51
IH	16 / 22 (73%)	55	1 - 340

The number and percentage of dogs with fasting bile acid concentrations above reference. Reference level: <10  $\mu\text{mol/L}$ . The median value and the range were given in  $\mu\text{mol/L}$ .

### Functional closure

In 59 dogs with PC CPSS the shunt was partially ligated, 20 dogs (34%) had persistent shunting based on ammonia metabolism, and in 39 dogs (66%) the shunt was functionally closed. In PAZ CPSS, 15 dogs had partial ligation of which 6 dogs (40%) had persistent shunting and 9 dogs (60%) had a functional closed shunt. In intrahepatic shunting 27 dogs had partial ligation, 20 dogs (65%) had persistent shunting and 7 dogs (35%) had no functional shunting after surgery.

In the other dogs with attenuated shunts the ammonia metabolism was unknown or the dog died, so it was not possible to determine persistent shunting or functional closure for these dogs.

### Persistent or recurrent shunting

The presence of portosystemic shunting after surgery was first determined using the ammonia metabolism: in PC CPSS 11 dogs had complete ligation and in these dogs, only one dog showed portosystemic shunting during check-up. In PAZ CPSS 9 dogs had complete ligation, of which 3 dogs (33%) showed shunting after surgery. In intrahepatic shunting only 2 dogs underwent complete ligation, and both had no shunting after surgery.

Sensitivity and specificity for persistence or recurrence of shunting determined with ultrasonography were calculated. Dogs were called positive for shunting when fasting ammonia concentration was over 45  $\mu\text{mol/L}$  or when ammonia tolerance test was more than twofold the fasting ammonia concentration and at least over 45  $\mu\text{mol/L}$ . In table 15a the numbers and percentage of persistent and recurrent shunting based on ultrasonography and ammonia metabolism are given. These numbers were used to calculate the sensitivity and specificity for ultrasonographic determination of persistent or recurrent shunting (table 15b).

*Table 15a: Shunting determined by ultrasonography and ammonia metabolism.*

	<b>Cases</b>	<b>+US +NH3</b>	<b>+NH3 -US</b>	<b>+US -NH3</b>	<b>-US -NH3</b>
<b>PC</b>	55	15 (27%)	3 (5.5%)	18 (33%)	19 (35%)
<b>PAZ</b>	19	4 (21%)	4 (21%)	4 (21%)	7 (37%)
<b>IH</b>	22	14 (64%)	3 (14%)	2 (9%)	3 (14%)

*Dogs were only included when ammonia metabolism and ultrasonography after surgery were known. Reported were the number and percentage of dogs in which shunting was found after surgery: positive by ammonia metabolism and ultrasonography (+US +NH3); positive by ammonia metabolism, but negative on ultrasonography (+NH3 - US); positive on ultrasonography, but negative on ammonia metabolism (+US - NH3); and negative on ammonia metabolism and ultrasonography (- US - NH3).*

*Table 15b: Sensitivity and specificity*

	<b>Sensitivity</b>	<b>Specificity</b>
<b>PC</b>	83%	51%
<b>PAZ</b>	50%	64%
<b>IH</b>	82%	60%

*Sensitivity and specificity for determining persistent or recurrent shunting after surgery with ultrasonography.*

### **Long-term follow-up**

*Questionnaires (see appendix 2a-c and appendix 3a-b)*

Initially 80 dogs with PC CPSS, 34 dogs with PAZ CPSS and 34 dogs with intrahepatic CPSS survived more than one month after surgery.

In total referring veterinarians of 114 CPSS dogs were contacted (64 PC (56%), 30 PAZ (26%) and 20 intrahepatic (18%) CPSS) to

evaluate survival after the last control visit. One dog with an extrahepatic PC CPSS was euthanized 3 months after surgery because of remaining signs. Fourteen dogs died or were euthanized later in life because of other reasons (5 dogs (36%) with PC, 3 dogs (21%) with PAZ and 6 dogs (43%) with intrahepatic CPSS).

In 34 cases it was unknown if the dog was still alive due to change of veterinary clinic (16 dogs (47%) with PC, 4 dogs (12%) with PAZ and 14 dogs (41%) with intrahepatic CPSS).

Forty-seven owners of dogs expected to be alive with PC CPSS, 24 with PAZ CPSS and 11 with intrahepatic CPSS were contacted and send a quality of life questionnaire. Of the dogs that died or were euthanized later in life 5 owners of dogs with PC CPSS, 2 owners of dogs with PAZ CPSS and 2 owners of dogs with intrahepatic CPSS were contacted and also send a quality of life questionnaire. Nine owners of dogs with PC CPSS, 4 owners of dogs with PAZ CPSS and 8 owners of dogs with intrahepatic CPSS could not be contacted by telephone. Although it was unknown if the dog was still alive, they were also send a quality of life questionnaire. A total of 112 (61 PC, 30 PAZ and 21 intrahepatic CPSS) quality of life questionnaires were send to all different owners.

In total 64 (57%) questionnaires were send back (43 PC, 13 PAZ and 8 intrahepatic CPSS). Of the dogs that were known to be euthanized or died after the last visit, 3 questionnaires of dogs with PC CPSS, 1 with PAZ CPSS and 1 dog with intrahepatic CPSS were received.

Of all questionnaires that were collected, 23 (36%) were correctly filled out, 41 (64%) questionnaires were not completely filled out, or some answers could not unambiguously be interpreted.

For the results the PC and PAZ shunts were analyzed together. The median follow-up period was 1652 days (range, 52-3889) days (56 dogs with extrahepatic and 8 dogs with intrahepatic shunting). Five of the 64 dogs died or were euthanized.

The mean PSS score of these 64 dogs was 43 (SD 27) preoperative. Postoperative the mean PSS score was 11 (SD 13). To test the improvement in this PSS score a Wilcoxon Signed Rank test was performed. The difference pre- and postoperative showed a

significant improvement ( $P < 0.001$ ). When the difference was analyzed separately in the extrahepatic shunts and the intrahepatic shunts, the scores postoperatively in both groups had also significantly improved (EH CPSS  $P < 0.001$ , IH CPSS  $P = 0.018$ ).

Presence of signs, that are mentioned below (before or after surgery) were also qualified by the owners, for the impact in their dog's life. This impact was on a scale of 0 to 10, 0 being no impact, and 10 being severe impact. The results are shown in table 16.

*Table 16: Impact of clinical signs*

Clinical sign	Impact
Seizures	7.7
Head pressing	6.1
Circling	6.0
Disorientation	6.2
Aggression	4.9
Collapse	5.1
Ataxia (wobbling)	6.5
Unresponsive episodes	5.9
Blindness	7.0
Lethargy/weakness	5.4
Vomiting	5.4
Diarrhea	3.8
Decreased appetite	4.9
Difficulty urinating	6.9
Blood in urine	3.1
Excessive drooling	7.4
Polyuria/polydipsia	5.0

*The average of impact of clinical signs found with CPSS dogs, scored by the owners in the Quality of Life Questionnaire, on a scale of 0-10.*

Forty-one of 64 dogs (64%) were treated before surgery with diet, medication or both. According to the owners, the mean clinical improvement with this medical management was 5.3 (scale 0-10;  $n=40$ ), with no improvement in 3 dogs (score 0) and a maximum improvement in 3 dogs (score 10). Willingness to eat the diet was scored with a 6.7 on average. The medical management hardly showed any negative effects on the dog's quality of life, the mean score was 0.9. Postoperatively, at the moment of the questionnaire or death of the dog, 13 of 60 dogs (22%) still received medical management.

The mean improvement of the dogs after surgery was scored with an 8.3 ( $n=59$ ).

Body condition improved on average according to the owner with a 6.6, the

improvement in body condition score before and after surgery (scale 0-10) was 2.4.

Also quality of life was scored before diagnosis, with medical management and at this moment, after surgery.

The mean quality of life before diagnosis and therapy was 2.4 ( $n=62$ ).

The mean quality of life with medical management was 3.7 ( $n=41$ ).

The mean quality of life after surgery was scored 8.7 ( $n=61$ ).

Owners were asked if they still worry about their dog's health, which was scored with a mean of 2.9 (0 being 'not worried at all'; 10 being 'could not be more worried').

Owners were asked about how satisfied they were with the result of the surgery. The mean score found was 9.1 (0 being 'not at all satisfied'; 10 being 'could not be more satisfied').

#### *Long-term outcome (see appendix 4a-b)*

Of all 167 dogs that underwent shunt ligation between 2003 and November 2013, 46 dogs were lost to long-term follow-up (26 dogs with PC, 3 dogs with PAZ and 17 dogs with intrahepatic CPSS). In the rest of the dogs 35 dogs had died at the time this study was performed; 25 with extrahepatic CPSS (19 PC, 6 PAZ), and 10 with intrahepatic CPSS.

Dogs were randomly chosen for a long-term check-up out of 80 dogs surviving CPSS with a minimum follow-up period of 6 months after surgery. The owners of 21 dogs accepted the invitation and visited the clinic. The sample included 14 dogs with PC, 5 dogs with PAZ and 2 dogs with intrahepatic shunting.

Clinical signs of HE were present in 8 dogs (38%) (5 with PC, 2 with PAZ and 1 with intrahepatic CPSS). In 6 dogs (5 with PC and 1 with intrahepatic CPSS) HE was scored as a class 1 HE and in 2 dogs with PAZ CPSS as a class 2 according to table 1 (Rothuizen, 1993).

Urological signs (pollakisuria, incontinence and/or hematuria) had been preoperatively present in 9 of 14 dogs with PC CPSS, in 3 of 5 dogs with PAZ CPSS and in 1 of 2 dogs with intrahepatic CPSS. After surgery no persistent or recurrent urological signs were noted in these dogs.

Before surgery, 6 of 14 dogs with PC CPSS, 2 of 5 dogs with PAZ CPSS and 1 of 2 dogs with intrahepatic CPSS had bladder uroliths. Three

dogs with PC CPSS, 1 dog with PAZ CPSS and 1 dog with intrahepatic CPSS had crystalluria before surgery. After surgery only 2 dogs, both with PC CPSS underwent ultrasonography, and both did not show bladder uroliths. One dog with PC CPSS had ammonia urate crystalluria during the extra control visit. This dog showed asymptomatic shunting.

The median fasting ammonia concentrations (reference interval, 24-45  $\mu\text{mol/L}$ ) in the 21 dogs was 31  $\mu\text{mol/L}$  (range, 8 - 201  $\mu\text{mol/L}$ ) with 5 dogs (24%) above reference. In 18 dogs of which fasting ammonia concentrations was measured <100  $\mu\text{mol/L}$ , an ammonia tolerance test was performed. Of the 18 tests 7 (39%) were positive, indicating portosystemic shunting.

The median value of fasting bile acid concentrations (reference level, <10  $\mu\text{mol/L}$ ) was 15  $\mu\text{mol/L}$  (range, 1-285  $\mu\text{mol/L}$ ). 12 dogs (57%) showed values above reference.

Using the results from the questionnaires and the measurement of fasting ammonia and the ammonia tolerance test, it was determined when there was symptomatic (clinical signs combined with evidence of abnormal ammonia metabolism), asymptomatic (evidence of

abnormal ammonia metabolism without clinical signs) or no shunting (no clinical signs and a normal ammonia metabolism) after surgery in each dog (table 17). At the last routine check-up after surgery of the 21 dogs, 7 dogs (33%) had no shunting, 6 dogs (29%) had asymptomatic shunting and 6 dogs (29%) had symptomatic shunting. In 2 dogs the degree of shunting at that time is unknown because no ammonia tolerance test was performed.

At the long-term check-up in this study, of only 19 dogs the questionnaire was filled out. Of these 19 dogs, 11 dogs (58%) had no shunting, the mean PSS score found in this group postoperatively was 12.2. There were 3 dogs (16%) with asymptomatic shunting, this group showed a mean PSS score of 19.3. Symptomatic shunting was seen in 5 dogs (26%), this group had a mean PSS score of 21.4. The differences in mean values are not significant.

Of the 2 dogs of which no questionnaire was filled out, based on the anamnesis and ammonia metabolism, 1 dog had no shunting, and the other dog had symptomatic shunting.

Table 17: shunting classification

Clinical Signs <sup>1</sup>	Abnormal ammonia metabolism <sup>2</sup>	Classification
+	+	Symptomatic shunting
-	+	Asymptomatic shunting
-	-	No shunting
+	-	No shunting

<sup>1</sup> Positive (+): HE class 1-4, weekly vomiting, diarrhea and decreased appetite, periods of urologic signs

Negative (-): HE grade 0, no or less than weekly vomiting, diarrhea and decreased appetite, no urologic signs

<sup>2</sup> Positive (+): Fasting ammonia >100  $\mu\text{mol/L}$  or a positive ammonia tolerance test (more than a twofold increase in plasma ammonia concentration with reference to the fasting ammonia concentration, the value also needs to be above 45  $\mu\text{mol/L}$ )

Negative (-): Fasting ammonia <46  $\mu\text{mol/L}$  or a positive ammonia tolerance test (no increase or less than a twofold increase in plasma ammonia concentration with reference to the fasting ammonia concentration)

The recovery of the dogs was evaluated using table 18. At the long-term check-up out of 21 dogs 11 dogs (52%) were completely recovered, 4 dogs (19%) were partially recovered and 6 dogs (29%) were not recovered.

Comparing shunting at last check-up after surgery and shunting at long-term check-up shows 11 dogs (52%) with CPSS without shunting, 7 dogs (33%) with persistent shunting, 1 dog (4.8%) has recurrent shunting and 1 (4.8%) dog is unknown.

Table 18: recovery classification

Clinical Signs <sup>1</sup>	Abnormal ammonia metabolism	Classification
-	-	Complete recovery
-	+	Partial recovery
+	-	Partial recovery
+	+	No recovery

<sup>1</sup> Positive (+): HE class 1-4, weekly vomiting, diarrhea and decreased appetite, periods of urologic signs

Negative (-): HE grade 0, no or less than weekly vomiting, diarrhea and decreased appetite, no urologic signs

<sup>2</sup> Positive (+): Fasting ammonia > 100 µmol/L or a positive ammonia tolerance test (more than a twofold increase in plasma ammonia concentration with reference to the fasting ammonia concentration, the value also needs to be above 45 µmol/L)

Negative (-): Fasting ammonia < 46 µmol/L or a positive ammonia tolerance test (no increase or less than a twofold increase in plasma ammonia concentration with reference to the fasting ammonia concentration)

## Discussion

This study was designed to evaluate long-term outcome data for all 167 dogs with CPSS who were surgically treated by ligation between 2003 and November 2013 at Utrecht University. The study was designed to support a prospective study of comparing extrahepatic shunt attenuation by ligation and cellophane banding.

In our study extrahepatic CPSS (77%) was more common than intrahepatic CPSS (23%). In the extrahepatic CPSS, PC was more common (71%) than PAZ (29%) CPSS. The distribution of these anatomical variations, are similarly found in other studies.

The type of breed is of significant interest in the anatomy of the shunt. Also in this study was found that extrahepatic shunting is mostly seen in small dog breeds, and intrahepatic shunting is mostly seen in large dog breeds.

This distribution may show heritability for shunting. (Hunt et al., 2004; van Steenbeek et al., 2013)

In this study the median age of surgery for PC CPSS was 10.5 months and for PAZ CPSS 20 months, also signs of HE were less severe and present in less dogs with PAZ CPSS. Similar observations of difference in age and signs between PC and PAZ CPSS were found in other studies. (Sura et al., 2007; van den Bossche et al., 2012) This probably is because less blood bypasses the liver so less clinical signs are seen, which might also lead to later diagnosis of CPSS.

Hepatic dysfunction resulting from CPSS can be expected in most dogs, based on the results of clinical signs and biochemical profiles.

Clinical signs of hepatic encephalopathy were noted in 90% of dogs with PC CPSS; 84% of dogs with PAZ CPSS and 95% of dogs with intrahepatic CPSS at time of presentation, determined by the classification system of Rothuizen (1993). This percentage is very high compared to other studies. (Falls et al., 2013; Winkler et al., 2003; Worley & Holt, 2008) However, in our study more signs of HE were taken into account, for example polyuria and character changes.

Dogs diagnosed with CPSS at Utrecht University, receive medical management, to reduce clinical signs before they go into surgery. A lot of dogs (41% with PC, 38% with PAZ and 61% with intrahepatic CPSS) already received medical management before they first came to Utrecht University, which will have a hold on the percentage of clinical signs and especially on the severity of these signs.

Biochemical profiles show that fasting ammonia concentration is above reference in 83-97% dogs with CPSS and ammonia tolerance test is positive in 100% of all cases. Also fasting bile acid concentration was above reference in most dogs (86-97%).

Ammonia and bile acids are both elevated in most cases because the venous portal blood bypasses the liver, so hepatocytes cannot extract it from the blood.



Surgery, in which the shunt is ligated, is not without risk. Mostly seen are hemorrhage, portal hypertension and cerebral necrosis. Short-term mortality rates in this study, 8.1-13%, were within range compared with other studies. (Hottinger, Walshaw, & Hauptman, 1995; Hunt & Hughes, 1999; Winkler et al., 2003) Short-term mortality in this study was mostly caused by abdominal hemorrhage and cerebral necrosis. Most of these dogs underwent partial ligation (58% of dogs with PC and 100% of dogs with PAZ and intrahepatic CPSS). The reason for higher mortality in the group of dogs with partial ligation might be because these dogs develop portal hypertension faster and more severe than dogs that underwent complete ligation. (Kummeling et al., 2004; Wolschrijn, Mahapokai, Rothuizen, Meyer, & van Sluijs, 2000)

In most dogs the shunt could only be partially ligated, as was also shown in previous studies. (Swalec & Smeak, 1990) The degree of attenuation was decided during surgery. Of 1 dog (PAZ) it was uncertain if the shunt was completely or partially ligated. Of 166 dogs 129 (78%) were partially ligated and 37 (22%) were completely ligated. It was also seen that complete ligation is mostly achieved in dogs with PAZ CPSS, 36% compared to 24% in PC and 5% in intrahepatic CPSS dogs. Although these differences were not statistically analyzed yet, it was also recorded in previous studies (Sura et al., 2007; Winkler et al., 2003). The reason for the higher percentage of complete ligation in dogs with PAZ CPSS is probably because they have a better developed liver and portal system and less severe signs. (Sura et al., 2007)

Most dogs with persistent shunting were asymptomatic, only 8% of the dogs still showed signs of HE (4% of dogs with PC, 10% of dogs with PAZ and 24% of dogs with intrahepatic CPSS) and these signs were less severe compared to preoperative signs. After surgery only class 1 and 2 were seen, compared to all classes (1-4) before surgery. Dogs had probably less clinical signs because an important part of the blood is now flowing through the liver.

Seven dogs with HE signs (2 PC, 1 PAZ and 4 intrahepatic CPSS) were still on medical management. In total 46 dogs (22 PC, 10 PAZ and 13 intrahepatic CPSS) were on medical

management at the last routine check-up after surgery. Medical management reduces signs of CPSS, so it is possible that the number of dogs with signs of HE and severity are in fact higher.

There was a large improvement after surgery with regard to the fasting ammonia concentrations. Before surgery dogs with PC CPSS had a median concentration of 153  $\mu\text{mol/L}$ , and after surgery it was 15  $\mu\text{mol/L}$ . For dogs with PAZ CPSS before surgery the median concentration was 97  $\mu\text{mol/L}$ , which was reduced to 15  $\mu\text{mol/L}$ . For dogs with intrahepatic CPSS the median concentration was 162  $\mu\text{mol/L}$  reduced to 63  $\mu\text{mol/L}$ .

Although all performed ammonia tolerance tests were positive before surgery, after surgery an ammonia tolerance test was found positive in 28% of dogs with PC CPSS, in 38% of dogs with PAZ CPSS and in 61% of dogs with intrahepatic CPSS.

In 26% dogs with PC, 30% in PAZ and 66% dogs with intrahepatic CPSS, there was evidence of persistent shunting by abnormal ammonia metabolism despite attenuation.

In 43% dogs with PC, 36% in PAZ and 73% dogs with intrahepatic CPSS fasting bile acid concentrations are above reference after surgery. However a large improvement was found for the dogs with extrahepatic shunting, looking at the median concentrations. In dogs with PC CPSS the median concentration of fasting bile acid was 105  $\mu\text{mol/L}$ , and after surgery it was only 8  $\mu\text{mol/L}$ . For the dogs with PAZ CPSS the median was 95  $\mu\text{mol/L}$  and was reduced to 7  $\mu\text{mol/L}$ . The intrahepatic CPSS dogs however had a median concentration of 35  $\mu\text{mol/L}$  before surgery, which is very low compared to extrahepatic shunting, and after surgery the median concentration is 54  $\mu\text{mol/L}$ .

There seems to be a bigger biochemical improvement for dogs with extrahepatic shunting, and the median concentration after surgery for the extrahepatic CPSS dogs is less than the reference level for both ammonia concentration and bile acids. For dogs with intrahepatic shunting the median is above the reference, and for bile acids the concentration even appears to be risen, which might suggest that dogs with extrahepatic shunting have better results after shunt ligation. Difference in this concentration however was not statistically

tested, so a conclusion cannot be made. The reason why intrahepatic CPSS dogs have less well biochemical profiles after shunt ligation is not totally clear.

Part of the group which has high bile acids after surgery do not show persistent shunting looking at ammonia metabolism, so the high bile acids are probably not caused by the shunt. It might be that these dogs have more liver damage during surgery, or the liver adapted less and did not return to a completely normal function.

If there is still postoperative hepatic dysfunction present, in most cases this will come from persistent shunting through the original shunt. In other cases it is likely that persistent shunting results from development of acquired shunting, however this was only confirmed by ultrasonography in one dog with PC CPSS. Because the clinical condition improved after surgery it is not likely that the other dogs all developed acquired collaterals. It is possible that in some dogs, other liver disorders may contribute to apparent failure of surgery, however in this research this was not investigated.

In most dogs we saw that hepatic function recovered, even though most dogs underwent partial ligation. Next to complete ligation during surgery, called mechanical closure, the shunt can also be functionally closed. In this case the shunt is partially ligated during surgery, and after the surgery, because of tissue reaction or because the flow changed to hepatopetal, there is no functional shunt left, so the blood is flowing through the liver. In 66% of dogs with PC CPSS, in 60% of dogs with PAZ CPSS and in 35% of dogs with intrahepatic shunting there was functional closure of the shunt after surgery. Although it was not known in all dogs if there was still persistent shunting or not, in most cases of extrahepatic shunting even after partial ligation there is no persistent shunting left, and we can say there is functional closure. If functional closure gives the same clinical outcome as mechanical closure during surgery, a second surgery is probably not necessary, because this will not lead to less shunting.

At the routine check-ups after surgery an ultrasonography is performed to determine if there is still functional shunting. Previous

studies have determined the reliability of Doppler ultrasonography for diagnosing CPSS. Kim et al. (2013) reported a sensitivity varying between 47-95% and a specificity of 67-100%. In this study sensitivity varying from 50% (PAZ) to 83% (PC) and specificity of 51% (PC) to 64% (PAZ) were found for determining persistent shunting. Persistent shunting was also determined by ammonia metabolism, and this test was used as the gold standard. There is a big range in this study, in which specificity is found in low percentage compared to other studies. This might be because radiologists were not blinded for the degree of clinical recovery.

CPSS can lead to urological problems, caused by formation of ammonium urate bladder uroliths or crystalluria. Dogs that had surgery underwent ultrasonography, during which the bladder was checked for uroliths. In 30% of dogs with PC, 41% of dogs with PAZ and 29% of dogs with intrahepatic CPSS bladder uroliths were reported. In 68% of all dogs with CPSS and preoperative bladder uroliths, the uroliths dissolved spontaneously. Because many dogs underwent a cystotomy, it is possible that the found chance for dissolution because of shunt ligation could be different when none of the dogs underwent cystotomy. For better research it is necessary to have more prospective follow-up and in the future a risk analysis can be made for additional cystotomy and the chance of dissolution.

Ammonium urate crystalluria can develop in CPSS dogs and lead to signs like hematuria and pollakisuria which can be the first signs seen by an owner. In 77 dogs with CPSS urine was analyzed before shunt attenuation, in 44 cases (57%) ammonium urate crystals were found. There were 26 (59%) female and 18 (41%) male dogs with crystalluria, which does not suspect gender influences.

After attenuation urine was not checked again of all these dogs, only 16 urine samples of dogs that were checked before surgery were checked after surgery. Of these 16 dogs 4 dogs (25%) were found positive for ammonium urate crystals in both samples. Two dogs that were found negative before surgery were found positive after surgery for ammonium urate crystals. Both of these dogs still had shunting after surgery based on the ammonium tolerance test.

When dogs are suspected of CPSS, urine sample analyzing is not a standard procedure, so dogs could be easily missed for crystalluria when the owner does not mention urological problems or the dog is asymptomatic. It is also possible that dogs were found incorrectly negative. Ammonium urate crystals are not common and not as easy to find as some other crystals, so a less experienced veterinarian can miss these crystals.

Because not all dogs that were found positive for ammonium urate crystalluria were also tested after surgery, it is not possible to draw a definite conclusion about dissolution of crystals after shunt attenuation.

In the long-term follow-up with this study, questionnaires were sent to owners of the dogs that underwent shunt attenuation between 2003 and 2014 at Utrecht University. Only 57% of the questionnaires were received back. This could be because the questionnaire contained many questions, and took some time to fill out. Also the questionnaire was not sent by email but by mail, which made it a bigger effort for the owners to send back. Only 36% of the questionnaires that were received back were correctly filled out. Some of the questions could easily be misinterpreted. Also most of the questions contained two parts, the staging of the sign and the impact on the dog's life, which made it easy to forget the second part. So the questions need to be improved and in some cases have to be easier. Also digitalizing would probably lead to a higher and more correct response. A bigger response will also allow the variations in shunt anatomy to be analyzed separately.

Follow-up period of dogs of which the owner filled out the questionnaire had a large range. Some dogs had surgery in 2003, and others only one month before the questionnaire was sent. It is hard to draw conclusions with this difference, because it is not possible to see if dogs had recurrent clinical signs, when they had surgery right before the questionnaire. However, because the questionnaires are still tested, all patients were taken into account.

The impact of clinical signs on the quality of life was calculated for all dogs, taken pre- and postoperative together. This is also because not all signs are present in all dogs, and not all questions about the impact were answered

correctly, which provided insufficient results to see if there is an association between the frequency of signs (never to daily) and the impact on the dog's quality of life assessed by the owners.

Medical management resulted in a mean improvement on the dog's life of 5.3 (scale 0-10), with hardly any negative effects. It also improved the quality of life of the dogs. According to the owners the mean of the quality of life with medical management improved from 2.4 without medical management to 3.7 on a scale of 0 to 10.

But when we look at the quality of life after surgery, the improvement is a lot better, increasing to a mean of 8.7. Also owners were very satisfied with the result of the surgery, a mean of 9.1 on a scale of 0 to 10 was found.

After surgery not many owners are still worried about the health of their dog, the mean score was 2.9 on a scale of 0 to 10.

These results show that owners are very positive about the surgery in many ways.

Because of cooperation with the RVC in London the development of the scoring system needs to be considered together. That is why in this study only a concept has been developed. With this system a significant ( $P < 0.001$ ) improvement was found in PSS score, comparing pre- and postoperative scoring. Preoperative the PSS score was 43 and after surgery the PSS score was 11.

Also in the long-term outcome of the 21 patients that came back for check-up it appeared that the PSS score was associated with postoperative shunting classification. The PSS score was the highest in dogs with symptomatic shunting and the lowest in dogs with no shunting. However, these differences were not found significant, probably due to small sample sizes.

These results suggest that the scoring system actually can determine clinical improvement and differences in shunting pre- and postoperative. The impact on the dog's life according to the owner was not taken into account for the concept of the scoring system, mostly because there is not enough data to use these results.

To develop a more accurate scoring system, more questionnaires have to be filled out to get larger sample groups. When there are enough results, and the system is optimized the goal is to use this system to help diagnosing dogs with

a possible CPSS or recurrent and persistent PSS.

Of the dogs still alive, 21 dogs came back for check-up, these dogs had at least 6 months of follow-up time. Although the dogs were randomly chosen, many owners that were contacted rejected the offer of check-up. The reason not to come back mostly given was that taking blood samples was too invasive for the dog. Owners were more motivated to come back, when the results of the last routine check-up were not as good as hoped, or when the owner was worried. So, although the initial group of dogs was chosen randomly, the actual group of dogs that was checked does not represent a randomized sample of the whole population. This might be a reason why the number of dogs with symptomatic and asymptomatic shunting is quite high. It was found that 11 dogs (58%) had no shunting, compared to the results at the routine check-up in the whole population this is a low percentage.

Limitations of this study lies mostly on its retrospective nature. Looking at the medical records, there was no standard protocol followed for patients suspected or diagnosed with CPSS. Many different people have been taking the dog's histories during these 10 years, which leads to different questions and more or less depth in asking. This makes that not all data of interest for this study were reported. In these 10 years there have also been some updates in, for example, surgery or anesthesia protocols, which can also influence some of the results after surgery.

For the long-term follow-up, owners of dogs that had surgery between 2003 and November 2013 were contacted. Time since surgery had been 10 years before follow-up for some of the dogs. A lot of efforts were made to contact referring veterinarians and owners, but because of the extended time since surgery many

individuals were untraceable. A number of dogs were lost to follow-up, so from only 64 of the original 167 dogs, data was available for long-term evaluation through the questionnaires.

Next to this, it is hard to draw conclusions about the clinical performance of dogs when follow-up is based on subjective reports of owners. Because the reports of owners might be misleading, an attempt was made to reduce this bias by using a standardized questionnaire. Also it is necessary to take into account that the number of patients is low especially in the PAZ and intrahepatic CPSS groups.

## **Conclusion**

The results of our study indicate that shunt ligation is an effective method for surgery if dogs suffer from CPSS. Clinical signs decrease and biochemical profiles are less deviating after surgery.

A concept of a scoring system has been developed; this system has the goal to eventually help diagnosing dogs with PSS. This study suggests that the scoring system can differentiate between symptomatic, asymptomatic and no shunting, which is promising for the future use of this system in diagnosing CPSS or detecting recurrent and persistent PSS after shunt surgery. However, the system was not completed yet, and has to be improved and validated in the future.

The results of this study indicate that there is a big chance that ammonium urate bladder uroliths dissolve after shunt ligation. To get a better view on this, there should be more prospective follow-up. In the future a risk analysis can be made to compare the outcome of dissolution after shunt ligation with cystotomy.

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**Appendix:**

- 1:** Medical records of UKG database
  - a. Details of patients with PC CPSS used in this study pg 26-34
  - b. Details of patients with PAZ CPSS used in this study pg 35-37
  - c. Details of patients with intrahepatic CPSS used in this study pg 38-40
  - d. Caption for 1a-c pg 41
  
- 2:** Quality of Life Questionnaire as send to the owners (translated in Dutch)
  - a. General information pg 42
  - b. Questionnaire before surgery pg 43-48
  - c. Questionnaire after surgery pg 49-55
  
- 3:**
  - a. Results of Quality of Life Questionnaires pg 56-65
  - b. Caption for 3a pg 66
  
- 4:**
  - a. Long-term results of patients that came back for check-up pg 67

1a: Medical records of UKG database, details of patients with PC CPSS.

EH PC CPSS

Patient	Breed	Gender	Age at surgery (months)		Medical management		Shunt attenuation		General impression		Retarded growth	Neurological signs		HE classification		
			before	after	before	after	before	after	before	after		before	after			
1	Rottweiler	M	4			1	partial	3,12	5	yes		5,11,13,16	3,5,6,13,14,16,18,20,21,22,23	4	2	
2	Cairn Terrier	F	4				complete								0	0
3	Crossbreed	F	21				partial	5,7	4	yes		3,4,8,9,14		3	0	
4	Malteser	M	36				complete	1	1			4,7,8,13,20,22		3		
5	Cairn Terrier	F	4				complete	1	1					0	0	
6	Malteser	M	14				complete	5,6,11	4			3,8,13,14,15		3	0	
7	Yorkshire Terrier	M	5				complete	1,3,10	1			5,2		2	0	
8	Malteser	M	4				partial	5	1			1,4,5,6,9,10,14,20,21,22		2	0	
9	Jack Russell Terrier	M	37				partial	3,11	4	yes		1,3,5,11,13,14,21,22,23		2	0	
10	Crossbreed	M	4		1		complete	5				5,8,11,16		3		
11	Dachshund	F	5		1		partial	1	1	yes		20		1	0	
12	Dachshund	M	15		1,2	1,2	partial	3,5,11	1,4	yes		1,3,4,6,7,11		2	0	
13	Jack Russell Terrier	F	34				partial	1,11	1			1,5,6,15,20		2	0	
14	Jack Russell Terrier	M	10		1		complete	3		yes				0		
15	Malteser	F	12		2	1	partial	3	4			13,15,21,22		2	0	
16	Dachshund	M	6		1		partial	1,3,11				1,11,15		2		
17	Miniature Schnauzer	F	27			1	partial	5	1			1,5,14,24		2	0	
18	Yorkshire Terrier	M	6				partial	6	1	yes		2,8		3	0	
19	Malteser	F	5		1,2		partial	3,5	1	yes		4,5,11,14,16,20,21		2	0	
20	West Highland White Terrier	F	5		1		partial	3,6	4			1,5,11,14,21		2	0	
21	Jack Russell Terrier	M	14		1,2		partial	1,3,6	1,4			3,4,8,11,14		3	0	
22	Malteser	M	50		1		partial					5,20,21		2		
23	Cairn Terrier	F	6				partial							0	0	
24	Malteser	M	14		1		partial	3,5	1			1,8,11,14		3	0	
25	Crossbreed	M	4,5			2	partial	3,5,7	1	yes		13,15		2	0	
26	Dachshund	M	36				partial	3,5				4,9,11,13,20		2		
27	Malteser	F	67		1,2		partial	5,6	1,4	yes		1,6,15		2	0	
28	Yorkshire Terrier	F	7		1,2		partial	1	1			6,13,14,21		2	0	
29	Malteser	F	15		1,2		partial	3	1	yes		3,5,11,12,13,14		2	0	
30	Pug	F	12			1	partial	3,5	1,4	yes		5,11		2	0	
31	Dachshund	M	7				complete	3,5	1	yes		9,13,21		2	0	

EH PC CPSS

Patient	Gastro- intestinal signs		Micturition		Appetite		Activity level		Fasting Bile acids $\mu\text{mol/L}$		Fasting ammonia $\mu\text{mol/L}$		NH3 tolerance test		Albumine g/L		White blood cell count $\times 10^9/\text{L}$	
	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after
1	1		4,6	4,6	3,6	3,6	2,4,5	2,5,6			7	>286	201					
2					6	1		1		16						28		
3	2,4	2	4,6,7		6	1	2	1	179	8	134	10			19	33		12,6
4		1	4,6		2,7		5		3	1	58	13	+		11			
5					1	1	1	1		4		19			26			
6					3	1	4	3	40		208	10			24		21,5	
7	1		1,4,5,6		6	1		1		1	170	16			30	30		
8						8	2	1	33		135	26			25		12,8	
9		1	6		6	1	4	1		6	187	16			30			
10	1				4		2,4			4		19				29		
11	1	1,3	2,3,4,5,6		4,6	1	1	1			162	62			20			14,1
12	1				4	8	2,5	1	23	73	33	17	+		26	26		
13	1	1			4	1	5,6	1	78		204	14			25		8,2	
14	1		2,5,6		4		2,5		37		100				25		18,6	
15	1	1	4,6,7		4	1	5,6	1				9			24			
16	1				4		2,4		58		159				31		12,9	
17	1		2,4		4,7	1	2	1	49	3	>286	12			23		12,9	
18	1		2		4	1	2,4	1	275	12	79	8			25		19,2	
19	1		4,6	6 <	4,6	1	2,4,6	1	54		153	45			19			
20	1		4,6	6 <	4,7	1	2,4	3			178	12			24		22,1	
21	1				4	8	2	1	66		149	16			20			
22			4		3,6		2,5		195		114				26		20,7	
23				6					13	6	32	7	+		31	33	11	8,4
24	1		2,3,4,7		2,6	1	2,4	1	168	10	224	7			18	5	16,5	9,3
25			3,4,6		2,7	6	2	1	230	19	194	23			20			
26	1,2,3		4		2,6		2,4,6		405		171				26		40,5	
27	1	1 <	2		1,6	1	2,5,6	1	135	22	123	18			16		42,6	15,2
28	1,2	1 <			6	1	2	1	125	26	286	9			21			
29	1,5				6	8	5	1	28	29	25	26	+		24			
30	1	1 <	4		4,6	8	5	5 <	615	26	94	13			28			
31	1		8		2,3	1	4	1		30	120	10			31		25,1	

EH PC CPSS

Patient	Crystalluria		Bladder ultrasonography		Complications		Check-up number	period, months p.o.	Ultrasonography post-operative	Mortality	Notes
	before	after	before	after	i.o.	p.o.					
1				1,2			2	2	open	3 (4 months p.o.)	Hypoplasia v.portae
2				4			1	1	closed		
3				2		yes	1	1	open		
4				2			0				
5							1	1	closed		
6				4			1	1	unknown		
7				2			1	1	unknown		
8							1	1	open		
9				2,5			1	1	closed		
10							0				
11	yes			2			1	1	unknown		
12	yes			1		yes	1	1	open		
13				2	4	yes	1	1	closed	4 (8 years p.o.)	
14				1,2		yes				3 (2 days p.o.)	Cerebral necrosis
15							1	1	open	4 (8 years p.o.)	
16				2		yes				3 (0 days p.o.)	
17	yes			1	1 <	yes	1	1	open		
18	yes			1,2	4		1	2,5	open		
19						yes	1	1	open		
20				2	4		1	1	open		
21				2	4		2	3	open		
22				1		yes				3 (5 days p.o.)	Cerebral necrosis
23							3	6	unknown		
24	yes			4			2	6	unknown		
25				5			1	1	open		
26	yes			1		yes				3 (0 days p.o.)	Liver hemorrhage
27				1			2	3	unknown		
28	yes			1			1	1	unknown		
29		yes		2	1		2	3	open		Urine was negative on crystals before surgery
30				2	2	yes	1	1	closed		
31	yes			1	4		1	1	closed		

EH PC CPSS

Patient	Breed	Gender	Age at surgery (months)	Medical management		Shunt attenuation	General impression		Retarded growth	Neurological signs		HE classification
				before	after		before	after		before	after	
32	Jack Russell Terrier	F	10		1	partial	3,6,11	4	yes	4,5,6,11,13,20		2 0
33	Jack Russell Terrier	M	26	1		complete	5			7		1 0
34	Jack Russell Terrier	M	21			partial	1,3	4	yes			0 0
35	Chihuahua	M	6			complete				4,5,6,7,20		2
36	West Highland White Terrier	F	15	1		partial	1,3	3	yes			0 0
37	Cairn Terrier	F	12			complete	1	1				1 0
38	Shih Tzu	F	7	2		partial	1,6	1		3,5,11,14,21,22,23		2 0
39	Miniature Schnauzer	F	16			partial	3,6	1,4	yes	1,14		1 0
40	Shih Tzu	F	21	1,2		complete	1					0
41	Pug	F	36			complete	1,5			5,9,13,16,19,20,21		2
42	Crossbreed	F	6			partial	3,6,11	1,4		5,6,13,14,15,16,19,20,21,22,23		2 0
43	Malteser	M	16	1		partial	5,11			1,5,6,11,14,20		2 0
44	Cavalier King Charles Spaniel	F	5			partial	1	1		9,11,13,14,16,22		2 0
45	Cairn Terrier	M	4			partial	1	1				0 0
46	Jack Russell Terrier	F	6	1		partial	1	1	yes	5,6,9		2 0
47	Yorkshire Terrier	M	6			partial	6,11			3,5,6,10,11,13,14,22		2
48	Cairn Terrier	M	6			partial	1	1				1 0
49	Malteser	F	39	2	1	partial	1,2	1	yes	3,4,6,9,14,15		2 0
50	Yorkshire Terrier	M	11	2		partial	2	1,4		4,6,9,14,21		2 0
51	Bologneser	F	6	1,2		partial	6	1,4		2,3,4,6,9,11,16,21		2 0
52	Malteser	F	19	3	1	partial	3,5	1,4		4,8,9,13,14		3 0
53	Cairn Terrier	M	11	2		partial	1,3	1				2 0
54	Malteser	F	37	1,2	1	partial	3,5,11	1	yes	2,3,4,9,11,12,14,21		2 0
55	Yorkshire Terrier	F	28	1,2	1	partial	1	1		3,6,11,13,14,21,22		2 0
56	Jack Russell Terrier	M	17			partial	3,5	1	yes	5,8,11,14,21		3 0
57	Crossbreed	F	18			partial	1	1		9,13,17		2 0
58	Crossbreed	F	67	1		partial	1,3,5	1,3		2,3,5,6,13,14,17,20,22		2 0
59	Malteser	F	10			partial				7,8,15,20,21		3
60	Jack Russell Terrier	M	67			partial	1,3,10	1				0 0
61	Pug	F	12			partial	1,3,5,6	1	yes	3,5,16,20		2 0
62	Yorkshire Terrier	F	4			partial	1,3			3,4,6,9,13		2
63	Crossbreed	M	36			partial	1			21		1

EH PC CPSS

Patient	Gastro-intestinal signs		Micturition		Appetite		Activity level		Fasting Bile acids $\mu\text{mol/L}$		Fasting ammonia $\mu\text{mol/L}$		NH3 tolerance test		Albumine g/L		White blood cell count $\times 10^6/\text{L}$	
	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after
32	1,2	1	4,6		4,6	1	4	1	260	0	>286	14	-	25		18,1	9,5	
33	1,2		2,4,6		3	1	2,5	1	102	1	90	8	-	27				
34			2,3		2	1	2,4	1	100	1	93	12	+	25	32		9,7	
35			4,6		2		2				133			24				
36							2,3	1		1	3	25	+	29				
37			4		1,6	1	1	1	275	1	187	16	+	25	28	18,2	13,8	
38					3	1	1	1		5	>286	8	-	18				
39	1		2,4		1,6	1,8	4	1	29		178	32	-	28				
40			2,3		1		1		105		53			29		13,2		
41	1		4,6,7		4,6		4		114		>286			21		21,1		
42	1	1	4,6		2,3,6	1	2,5,6	1	24	27	116	45	+	21	17	21,8		
43	2						4,5,6		300	56	79	61	+	20		18,4		
44			4		1,6	1	2	1		13	124	69	+		26			
45					1	1	1	1			89	7	+	15				
46	1,2	1	4,6		1,6	1	5,6	1	22	4	57	10	-	24				
47	1		4		2,3,6				19		>286			22				
48			4,6		1,6	1	1	1			132	7	+	22		13,7		
49	2,3	2	4		3,6	3	4	5		13*	154	7	-	12		12,1		
50	1		4		2,3,6	1	2	1	106		169	8	-	17		18		
51			4,6		3,6	1	2,5,6	1			233	37	+	17		13,8		
52			1,4,6		3,6	1	2,5	1	69	8	67	13	+	21		9,7		
53			4,6		1,7	1	4,5,6	1		59	276	51	+	27		20,6		
54	1		4,6		3,6	1	2,4	1	136	1	32	15	-	21	26	29,1		
55			4,7		1,6	1	1	1	158	19	185	19	+	22	28			
56	1,2				2,3	1	4	1	11	4	43	7	-	16		21,2		
57			4,6		3,6	1,2,3	5	1,5		56	90	62	+			9		
58	1,2		4,6		3,6	1,2	6	2,6		440	161	160	+					
59	1,2		4		6		2		67		55			27				
60	1,2		2,3,6,7		1,7	1	2,5	1	72	3	149	28		27	28	9,3		
61	1				1	1	2,5	1		8		18	-	21	28			
62	1				1		4		103		90			24		11,7		
63	1		4,6		2,6		1		99		178			25		16,7		

Patient	Crystalluria		Bladder ultrasonography		Complications		Check-up		Ultrasoundography post-operative	Mortality	Notes
	before	after	before	after	i.o.	p.o.	number	months p.o.			
32							3	2	closed		
33	yes		1				1	1	closed	4 (5 years p.o.)	
34	yes		1,2,5				1	1	open		
35						yes				3 (2 days p.o.)	Shock
36			4				3	2	closed		
37							3	2	closed		
38			4				2	2	unknown		
39	yes		1,2	1,2			1	1,5	open	4 (5 years p.o.)	
40	yes		1			yes				3 (2 days p.o.)	Hemorrhage
41	yes		4		yes	yes				3 (1 day p.o.)	
42							1	1	open	4 (4 years p.o.)	
43	yes	yes		3			1	1,5	open		
44			4				1	1	open and acquired shunting		
45							1	1	unknown		
46							1	1	closed		
47	yes		2			yes				3 (0 days p.o.)	Cardiorespiratory arrest
48			4	4			1	1	closed		
49							4	2	open		
50	yes		4				1	1	closed		
51			4				1	1	closed		
52			1				2	4	open		
53							3	2	open		
54			4				1	1,5	open		
55			2	1			1	1	open		
56							1	1,5	open		
57							1	1,5	open	4 (unknown)	
58			1	1			1	1	open		
59						yes				3 (0 days p.o.)	Abdominal hemorrhage
60			1,3			yes	1	1	unknown		
61			2				1	1	closed		
62			4			yes	0				
63			1				0				

EH PC CPSS

Patient	Breed	Gender	Age at surgery (months)	Medical management		Shunt attenuation	General impression		Retarded growth	Neurological signs		HE classification	
				before	after		before	after		before	after	before	after
64	Crossbreed	F	12		1	partial	3,5,11	4		1,5,14,15,20,21		2	0
65	Pug	F	5		1	partial	3,5	1,4	yes	1,5,21		2	0
66	Dachshund	M	6			partial	1	1		9,10,14,16		2	0
67	Maltese	F	7	1		complete	1,5	1	yes	1,3,4,5,11,13,14,21		2	0
68	Yorkshire Terrier	M	6			partial	1,5			5,9,13,14		2	
69	Border Collie	M	5	1		partial	1,5,6	4		5,14,15,21		2	0
70	Chihuahua	F	18	1		partial	1,3	1	yes	5,7,15,20,21	12	2	1
71	Jack Russell Terrier	F	12			complete	1,3,5	1,4	yes	4,6		2	0
72	Calm Terrier	F	5			partial	1	1				1	0
73	Chihuahua	F	23			complete	1,3	1,4		3,7,8,13,14		3	0
74	Labrador Retriever	F	10	2		partial	3	4	yes	3		2	0
75	Border Collie	F	4,5	1		partial	1	4	yes	1,2,6,9,14,21		2	0
76	Miniature Schnauzer	F	7	1		partial	5	1	yes	5,6,7,13,14,20,21		2	0
77	Yorkshire Terrier	F	6,5	1		partial	1,5	1,5	yes	3,4,8,11,14		2	
78	Miniature Schnauzer	M	13	1,2	1	complete	3,5	1		5,9,20,21		2	0
79	West Highland White Terrier	F	7,5	1	1	partial		1,4	yes	3,4,6,7,11,13,21,22		2	0
80	Maltese	M	39	1		complete	1,3,5		yes	8,11		3	
81	Crossbreed	F	5		1,2	partial	6	1,4		1,4,6,9,11,14,21,22		2	0
82	Crossbreed	F	8	1,2	1	partial	3	4		21		1	0
83	Calm Terrier	F	5		1	partial		1	yes	4,5		2	0
84	Maltese	M	9			complete	3	1	yes	3,12	12	2	1
85	Jack Russell Terrier	M	47	1		partial	3,6			2,3,14,16,23		2	
86	Beagle	F	11	1,2		complete	7		yes	1,5,9,14,20,21,22,23,24		2	0
87	Crossbreed	F	44			complete	5	1		11		2	0
88	English Cocker Spaniel	F	7			partial	3,6	1,4	yes	1,3,5,11,13,14,16,21		2	0
89	Jack Russell Terrier	M	21			partial	3,6	1		9,14		2	0
90	Calm Terrier	M	7	2		partial	1,5,9	1		9,13,20,21		2	0
91	Pekingese	F	33			complete	5,6	1		5,9,11,13,14,20,21,22		2	0
92	Shetland Sheepdog	F	6			partial	1,3	1	yes	4,6,9,11,13,14		2	0



EH PC CPSS

Patient	Gastro- intestinal signs		Micturition		Appetite		Activity level		Fasting Bile acids $\mu\text{mol/L}$		Fasting ammonia $\mu\text{mol/L}$		NH3 tolerance test		Albumine g/L		White blood cell count $\times 10^9 / \text{L}$	
	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after
64	1,2	1	3,4,6		4,6	2	4	1	18	>286	10	-	23	23				
65				6	3	1	2,4,5	1	110	28	285	36	+	19	25	24,3		
66					1	1	1	1	40	1	>286	18	-	19				
67			3,4,6		4	8	2,4	1	13	141	22			25		18,8		
68	2						2,4,5		161	285				23		21,2		
69	1,2		4,6		3,6	6,8	4,5	1	205	35	285	<7	+	23	27	17		
70	1		4		4	1	2,5	1	355	65	230	139		25	29	21,9	13,3	
71	1		4		6	1	2,4,6	1	275	3	132	15	+	31	31	10,2	9,5	
72			4,6		6	1	5	1		1	221	14		21	28	19	13,7	
73	1		3		2	1	3,5	5	12	155	20							
74	1				3	8	2	1	15	103	25	+		18		21,9		
75	1		4,6		2,7	6	4	1	7	178	38	+		18		21		
76			3,4,5		4,6	3	2,4	1	265	>286	11			18		20,9		
77	1		4,6		2		2,4		72	116				18		19,8		
78	2		5		4	1	2,5,6	1		1	55	<7	+	24		14,6		
79			4		4,7	1	2,3,4,6	1		9	111	12		20	27	11,7	7,6	
80			4,6		6		2,4				61			25		8,7		
81	2		4		6	1	2,4	1	8	219	<7			19	28	25,8		
82	1	1	4,7		6		2,5				7			24		7,5		
83		1				1	3	1		2	166	282	+	22		18,4		
84	1,2		4,6		4,6	8	2,5	3		3	123	19		24		13,8		
85			4,6		7		2,6		135	229				21		21,2		
86			4,6		2,4,6	8	2,5,6	1		1	83	<7		23	29	18,3		
87	1	1	2,4		3,6	1	2,5	1	117	4	22	<7						
88	1,2				2,3	1	4	1	161	24	>286	179		23	25			
89	1		4,6,7		3,6	1	2	1	126	5	157	<7		31	28	11		
90			4,6		3,6	1	4	1		14	218	17			26	20,9	15,7	
91	1		4		3,6	8	2,5,6	1	154	10	126	<7		28	30	15,4		
92	4		4		2,6	1	2,5	1	178	1	190	30		21	26	21,7		

EH PC CPSS

Patient	Crystalluria		Bladder ultrasonography		Complications		Check-up		Ultrasoundography post-operative	Mortality	Notes
	before	after	before	after	i.o.	p.o.	number	months p.o.			
64	yes		1	1			2	2,5	open		
65			4				2	4	open		
66			2	2			1	1	open		
67			4	4			1	2	closed		
68			4	4		yes				3 (0 days p.o.)	
69			1	1			3	10	unknown		
70			4	4			2	3	open		
71			4	4			1	1	closed		
72			1				1	1	unknown		
73			2				1	1	closed		
74	yes		4	4			1	1	unknown		
75	yes		4	4			2	3	open		
76	yes		1	4	yes		1	1	closed		
77			1				0				
78	yes	yes	1,2	1		yes	2	3	closed		
79			1	4			3	5	open		
80			1	1		yes				3 (3 days p.o.)	Cerebral necrosis
81			1				2	3	open		
82							1	1	unknown		
83			4	4			1	1,5	open		
84							1	2,5	unknown		
85	yes		2				0			3 (2 days p.o.)	
86			1				1	1	open		
87			1	2			1	1,5	closed		
88	yes		2				2	3	deviating vessel pattern		
89			4	4			1	1	closed		
90			2	4			1	1	closed		
91			4	4			1	1	unknown		
92			4	4			1	1	closed		

**1b: Medical records of UKG database, details of patients with PAZ CPSS**

EH PAZ CPSS

Patient	Breed	Gender	Age at surgery (months)	Medical management		Shunt attenuation	General Impression		Retarded growth	Neurological signs		HE classification	
				before	after		before	after		before	after	before	after
93	Norfolk Terrier	F	10	1		partial	2	4	yes	5, 20		2	0
94	Yorkshire Terrier	F	33			complete	3,	4	yes			1	0
95	Maltese	M	46	1,2,3	1	complete	1,3	4	yes	2,5,13,16, 22, 23		2	1
96	Norfolk Terrier	F	3	1		partial	3,5,6	4		1,5,14,15, 20, 21		2	0
97	Crossbreed	F	42	1,2	1	partial	1,5					1	0
98	Miniature Poodle	M	37			partial	3	1,4	yes			1	0
99	Maltese	F	47			partial	7,8	8		5,6,12,17	9,17	2	2
100	Lhasa Apso	F	54	1		partial	1,5			2,9,11,14		2	
101	Cairn Terrier	M	5,5		1	complete	5	4				1	0
102	Dachshund	F	6			partial	1,3,8					0	0
103	Great Dane	M	5		1	complete	1		yes			0	0
104	Crossbreed	F	55			partial	5		yes	1,14, 19		1	0
105	Crossbreed	M	14			partial						1	0
106	Welsh Terrier	F	40			complete	5		yes			1	0
107	Yorkshire Terrier	F	6	1,2	1,2	partial		1	yes	4,8,9,11,13		3	0
108	Shih Tzu	F	47			partial	1,8		yes	21, 24		1	
109	Dachshund	F	66		1	partial	3,5,6,8	1,2	yes	11,14,16,18		2	0
110	Pug	F	5,5	2		partial	3		yes	3,4,9,14,16, 21		2	
111	Fox Terrier	M	42	1		partial	2	1		14		1	0
112	Pekingese	M	56			partial	5	1			1,6,14, 20	2	1
113	Jack Russell Terrier	M	20		1	partial	1	1				0	0
114	West Highland White Terrier	F	23	1,2		partial	1	1				0	0
115	Jack Russell Terrier	F	5			partial	2,3,5,10	4		15		1	0
116	Norfolk Terrier	F	4			complete	1	1				0	0
117	Dachshund	F	7,5			complete						1	0
118	Miniature Pinscher	F	10			complete	1		yes	6,7		2	
119	Jack Russell Terrier	F	19	1,3		complete	3,11			3,5,6,(8),11,14,15, 21, 22		3	
120	Yorkshire Terrier	F	20			partial	5	4			15	1	0
121	Dwergschnauzer	M	14			partial	1,2		yes	4,16		2	
122	Fox Terrier	F	18	1,2	1	partial	3,5	1	yes	14,(18)		1	0
123	Chihuahua	F	35			complete	1,3	1,4	yes	5,15, 20, 21		2	0
124	Poodle	F	18	1,2		partial	1,3,8		yes	11		2	
125	Yorkshire Terrier	F	56	1,2		complete	3	1	yes	3,5,6,8,12, 22		3	0
126	Chihuahua	F	50			partial	1,5	4		5,8,14, 20, 21		3	0
127	Chihuahua	F	4			complete	1	4				0	0
128	Miniature Schnauzer	M	44	1		complete		1		6,14,16,17, 21		2	0
129	Jack Russell Terrier	F	78			complete	4	3				1	0

EH PAZ CPSS

Patient	Gastro- intestinal signs		Micturition		Appetite		Activity level		Fasting Bile acids $\mu\text{mol/L}$		Fasting ammonia $\mu\text{mol/L}$		NH3 tolerance test		Albumine g/L		White blood cell count /L	
	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after
93	2		4		5,7	1	5		115	5	142	13			17			
94			4		7	1,6	2	1	123	51	148	15			29		30	8,6
95	1		4,6		6		2	2	87		>286	15			21			16,3
96	1,2				2,4,6	8	4,5	1			118	17			15			
97	1	1	4		2,4,6	8	2	1	22	8	129	7			14			
98	1	2	4,5,6,7		4		2	1	53	13	49	7			29			
99	1		4,6	6	7	1	3	5	41	11	96	19			29			
100	1		4		2		2		109		154				16			
101	1,3		4		3,6	1	4	1	90	2	48	24			26		26	10
102	1	1			3,9	1	2,4,5	1		2	74	7			27			8
103										2	54	8			18		29	12,8
104	1				1,6	1	2	1	300	12	95	7			26		26	13,7
105	1,2		1,2,4			1		1		6	81	7			27			19,1
106	1	1	4,6		3,7	1	4	1	190	1	88				23			15,7
107	1,2				1	1	5	1	3	146	18							22,9
108			4,6		4				71		125				23			12,6
109	1		6		1	1	2	1		37	23				19		12	19,3
110	1		4		4,7		5		465		286				24			
111			4		6	8	5	1	17	1	27	7			25		28	21,1
112	1,2		4,6		3,6	8	4	1	175	14	117	28			26		30	
113	1		1,2		1	1	1	1	138	7	41	17			29			
114									107	1	238	22			30		36	12
115	1,2				2,3,9				130	9	136	33			21		24	16,1
116						1	1	1	14	30	111	28			28		28	12,4
117	1		2,4		3,6	1			15		27	13			26			14,3
118	3		2,4,6		1,6		3		35		46				28			6,4
119			2,4,6		2,6		2,4				84				29			14,4
120	1,2				1		4,5,6	5	70		129	19			<9			26,5
121			2,4,6		6,8		5		59		212							
122	1,2		4		3,7,9	3	2	6	41	18		19			15		23	13,8
123	1		4,6,7		4,6	3	1	1	127	2	28	9			30		31	
124	1				2		2,6		53		98				27			
125			3,6	3	6	1	1	1	190	8	211	<7			24			13,3
126						8			157	1	217	18			22			15,1
127	1					1				1	62	<7			26		27	15,1
128					3	1	1	1	100	51	49	<7			26		29	13,3
129			1,2,3,5,6	3	6		4	1	200	13	51	12			28		32	

EH PAZ CPSS

Patient	Crystalluria		Bladder ultrasonography		Complications		Check-up number	period, months p.o.	Ultrasonography post-operative	Mortality	Notes
	before	after	before	after	i.o.	D.o.					
93			2				1	2	open		
94							1	1	closed		
95			1,3				1	1	closed		Complete ligation in two surgeries
96	yes		2				1	1	closed		
97	yes		1,2	3			1	1	closed		4 (8 years p.o.)
98	yes		1,2				1	7	unknown		
99	yes						2	3	open		4 (8 months p.o.)
100			2			yes			unknown		Abdominal hemorrhage
101							3	2	unknown		
102			4				1	1	closed		
103						yes	2	7	unknown		Ruptured spleen intra-operative
104			4			yes	1	1,5	closed		Portal hypertension and abdominal hemorrhage p.o.
105	yes		1,2,3	2			3	2	closed		
106	yes					yes	3	2	closed		
107			2			yes	2	3	open		
108	yes		1,2,3			yes			unknown		3 (0 days p.o.)
109							2	2,5	open		
110			1			yes			unknown		3 (1 days p.o.)
111	yes	yes (<)	1,2,3	2			1	1	open		
112			1,2	1			1	1	open		
113	yes		1,3	4			1	2	closed		
114	yes		1,2	4		yes	1	1,5	closed		
115			4				1	1	open		4 (1 year p.o.)
116						yes	1	1,5	unknown		
117	yes		1,2				1	1	closed		
118	yes		2						unknown		
119	yes	yes	2,3,5						unknown		
120							1	1,5	closed		
121			2,3,5						unknown		
122						yes	1	1	closed		
123	yes		1	1			1	1	open		
124			4						unknown		
125			1				1	1	closed		
126			1	4			3	5	unknown		
127			4				1	1	closed		
128			4	2			1	1	closed		
129	yes		1				1	1	closed		

1c: Medical records of UKG database, details of patients with intrahepatic CPSS.

IH CPSS

Patient	Breed	Gender	Age at surgery (months)	Shunt type	Medical management		Shunt attenuation	General impression		Retarded growth	Neurological signs		HE classification		
					before	after		before	after		before	after	before	after	
130	Golden Retriever	F	8	Left		1,2	partial		1,2,3,4			1,14	19,22	1	2
131	Hovawart	M	14	Left			partial	2,3,5,10	1,4			5,21,24		2	0
132	Crossbreed	M	6	Left	1		partial	1,5		yes		5,13,14,15,20		2	
133	Grand Basset Griffon Vendéen	F	14	Left	1		partial	1	1	yes		6,13		2	1
134	Golden Retriever	F	7,5	Left	1,2	1,2	partial	1	1,4	yes		3,4,5,11,16,20,21		2	0
135	Dobermann Pinscher	F	5,5	Left			partial	1,2,3,11		yes				1	
136	Golden Retriever	M	4,5	Left	1,2	1	partial	1,5,3	1,8,9			3,4,13,14		2	0
137	Labrador Retriever	M	9	Left			partial	2,3	1			1,9,11,13,14,21,22		2	0
138	Deerhound	M	5	Left			partial	1	1,3	yes				0	0
139	Irish Wolfhound	F	5	Left	1		partial							2	0
140	Labrador Retriever	M	5,5	Left	1,2	1	partial	1		yes				1	0
141	Bernese Mountain Dog	F	13,5	Left			partial	1,3,5,6		yes		11,17		2	
142	Cane Corso	M	4,5	Left	1		partial	1,2	1,4					1	0
143	Bernese Mountain Dog	F	9	Left			partial	1,5	1	yes				1	0
144	Irish Wolfhound	F	3,5	Left			partial	3,11	1,4	yes		5,6		2	0
145	Bernese Mountain Dog	F	4	Left			partial	1	1					0	0
146	Golden Retriever	M	12	Left	1		partial	1,2,5	1					1	0
147	Golden Retriever	F	9	Left			complete	1	1,4			19		1	1
148	Cane Corso	F	54	Left	1,2,3		complete	1,3,11	1,4			3,5,10,11,12,14		2	0
149	Petit Basset Griffon Vendéen	M	6,5	Left		1	partial	1,3,5	1,5	yes		1,5,6,8,11,21		3	2
150	English Cocker Spaniel	M	10	Left	1,2		partial	1,2,11				8,13,20		2	
151	Crossbreed	M	17	Left	1,2		partial	1,9		yes		12,20,22		1	
152	Vizsla	M	12	Right	1	1	partial	1,9,11	1,4			6		2	0
153	Bearded Collie	M	6	Right	1	1	partial	2,5	1,4	yes		1,11		2	0
154	Labrador Retriever	F	3,5	Right	1,2		partial	1	1,4	yes		1,2,3,5,13,22		2	0
155	Golden Retriever	F	46	Right	1,2		partial	3,6		yes		2,5		2	
156	Shih Tzu/M	M	11	Right	1		partial	1,5				8,2		3	
157	Nova Scotia Duck Tolling Retriever	M	4	Right			partial	2	2,9,11	yes		4,8,11,14,15	2,12,14	2	1
158	Weimaraner	F	6	Right			partial	2,3,5		yes		2,15		1	
159	Bernese Mountain Dog	F	15	Right	1,2	1,2	partial	3,5						1	0
160	Stabyhoun	F	10	Right	1,2		partial	3,6				5,6,9,11,13,14,20,21,22		2	
161	Bearded Collie	M	4	Right	1,2		partial	1,9	1,3	yes				2	0
162	Shih Tzu	M	10	Right	1	1	partial	5	6			1,3,5,11,13,20		2	2
163	Crossbreed	M	11	Right	1,2,3	1	partial	1,2	1	yes		2,3,11,15,21,22,24	6,13,15	1	1
164	Cane Corso	F	5	Right	1	1	partial	2,3,5	1	yes		1,6		1	0
165	Australian Shepherd	F	13	Central	1	1	partial	1,2,3,5,9	1	yes		2,6,11,13,14,15,16,22,23		2	0
166	Bernese Mountain Dog	M	6	Central			partial	1	1			2,3,5,6,11,13,14,16,21		2	0
167	Golden Retriever	M	9,5	Central	2	1	partial	5	1			4,22		2	0

IH CPSS

Patient	Gastro-intestinal signs		Micturition		Appetite		Activity level		Fasting bile acids $\mu\text{mol/L}$		Fasting ammonia $\mu\text{mol/L}$		NH3 tolerance test		Albumine g/L		White blood cell count $\times 10^9/\text{L}$	
	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after	before	after
130					1	1	5	1	380	237	166	84	+	25		14.1		
131	1,2	1			4	3	4,5	1	28	51	113	37		19		10.7		
132	1				2,3		4				171			8		34.2		
133			4,5,7	4,6	1,5	1	1	1	35	6	114	37		22				
134	1,2,3	1	4		3,6	2,8	2	1	156	21	286	249	+	16		17.7		
135			4		2,6		2				183							
136			4		2,3,6	1	2	1				76		16				
137	1		4		3,6	2,8	2	1	37	14	171	18	+	30		27		
138					1	1,8	1	1			154	173		19				
139									6	5	139	63	+	13		10.9	14.4	
140			3,4		5,6		5			14		44	+	12		28	17.5	
141	1		2,4		3,6		2		125		120			22		15.4		
142			4,6		2,3,6	8	2,5	1		10		24		23		15		
143	1,2		4,5,6		3,7	1,8	1	1	63	5	120	14		16		30	15.5	
144			3,6		2,3	1,8	5	1	93	170	286	> 286		25		18	11	
145					1	1	1	1	18		156	49						
146					1	1,2	5,6	1		3	54	12	+			30		
147			4,6	4,6	1,7	1	1	1	140		>286	46		24		13.1		
148			4		1	1	2	1			104	20						
149	1		4		3,6	2	4,5	5		147	269	113						
150	1				1		2				159			18		11.1		
151			4,7		3,6		3				>286			21				
152			4		1,7	1	2	1	14		44	20		27				
153	1		4		1,6		4	1		58	56	80	+	22				
154			4,6		1,6	1	1	1				22		16				
155	1		4,6,7		1,6		2		36		214			17				
156			3,4		2,3,6		6				>286							
157	1		3	4,6	2,3	2,6,9	2	2,3	180	250	270	90	+	21		14.8		
158	1				2,3		2				>286							
159	1				6		4		30	185	117	179		12		35.4	19.1	
160			4		3,6		4,5,6		17		140			20		13		
161	1		3,4,6		2,6	1	1,2	1	12	33	214	147	+	16		13.74	11.8	
162			4		2,3,6	1	4,5,6	2,4,5,6		110	119	212		26		23	16.8	
163	1,2		4	4,6	2,5	1,6	2	1	200	340	76	60	+	24		25	25.9	
164	1		4		1,6	1	2	1		154	202	98		10		8.3		
165	1		4		2,3,6	1	2,5,6	1	16	93	190	82		18		20.3		
166	1		4		1,6	1	1	1	2		1	285	+	20		27	26.8	
167			4		1,6	1	2,6	1		94	67	21	+			24		

IH CPSS

Patient	Crystalluria		Bladder ultrasonography		Complications		Check-up		Ultrasonography post-operative	Mortality	Notes
	before	after	before	after	i.o.	p.o.	number	months p.o.			
130	yes			1		yes	3	3	open		
131			2		yes	yes	2	4	unknown		
132						yes				3(1 day p.o.)	Portal hypertension and hypoglycemia
133		yes	2	2,3			1	1,5	open		
134							2	2	unknown		
135									unknown		
136							3	4	closed		
137			1			yes	2	3	open		
138			4		yes		2	3	unknown		
139							unknown	6	unknown		
140		yes	1,2				unknown	6	unknown		Urine was negative on crystals before surgery
141	yes		1						unknown		
142			1	4			1	1	open		
143							1	1	closed		
144			4				2	1	open		
145							2	4	closed		
146			4				2	3	unknown		
147			2				1	1	closed		Complete ligation in two surgeries
148			4	4			1	1	closed		
149			1,2	1,2			3	4	open		
150			1,2			yes				3(2 days p.o.)	Cerebral necrosis
151						yes			unknown		
152			4	4	yes		1	2	unknown		
153			2				2	3	open		
154			4	4			2	2	closed		
155			2		yes	yes				3(11 days p.o.)	Euthanasia because of lethargy
156					yes					3(25 days p.o.)	
157							3	6	open	4(unknown)	
158			2,3						unknown		
159	yes		1,2,5				unknown	6	open	4(unknown)	
160			1						unknown		
161			4			yes	1	1,5	open		
162			2				1	1	open	4(3 years p.o.)	Euthanasia because of heartfailure
163		yes	4				2	3	open		
164			1,2				2	3	open	4(3 years p.o.)	Euthanasia because of chonical hepatitis
165	yes			2			1	2	open	4(unknown)	
166			1,5	1			1	1	open		
167			1,2	2			2	5	open		



1d: Caption for 1a-c

	Medical management	General impression	Neurological signs	GI signs	Micturition	Appetite	Activity	Bladder ultrasono	Mortality
1	Low protein/ kidney/liver diet	Attentive	Depression	Vomiting	Stranguria	As usual	As usual	Uroliths	Pre-operative
2	Lactulose	Calm	Abnormal behaviour	Diarrhea	Hematuria	Changing	Decreased	Sediment	Intra-operative
3	Antibiotics	Losing weight	Head pressing	Melena	Pollakisuria	Decreased	Increased	Cystitis	Post-operative (# days)
4		Gain weight	Circling	Gagging	Polyuria	Anorexia	Indolent	Normal	Died of different cause
5		Lazy/ indolent	Ataxia	Coprophagia	Dysuria	Unknown	Sleeps a lot	Polyp	
6		Soporosis	Wandering around		Incontinence	Polydipsia	Tired		
7		Bad breath	Muscle tremors		Nocturia	Severe polydipsia			
8		Nervous	Seizures			Increased			
9		Excitation	Blindness			Pica			
10		Pain	Deafness						
11		Restless	Ptyalism						
12		Coma	Aggression						
13			Desorientation						
14			Vacant						
15			Weakness						
16			Compulsive movements						
17			Fear						
18			Insomnia						
19			Slip						
20			Fall over						
21			Staggering						
22			Looking for support						
23			Wobbliness						
24			Not walking						

## 2a: General information



Universiteit Utrecht

Naam:

Patiëntnummer:

### Vragenlijst voor de kwaliteit van leven bij een leverschunt

Hartelijk dank dat u onze vragenlijsten wilt invullen.

Er zijn twee verschillende vragenlijsten:

Deel 1 gaat over hoe het met uw hond ging voor de operatie van de leverschunt.

Deel 2 gaat over hoe het nu met uw hond gaat.

Wilt u onderstaande vragen alstublieft beantwoorden voordat u met deel 1 begint?

#### **1: Hoe oud was uw hond toen u hem/haar hebt gekregen?**

Jonger dan 6weken    6-12weken    12weken-6maanden    Ouder dan 6maanden

#### **2: Hoe bent u aan uw hond gekomen?**

Fokker (bedrijfsmatig)    Particulier    Asiel    Vrienden/familie

Internet    anders:

#### **3: Heeft uw hond een stamboom (uitgegeven door de Raad van Beheer)?**

Nee    Ja

Registratienummer of transpondernummer (chip):

Anders (bijvoorbeeld buitenlandse stamboom of registratie door een kennel club):

## 2b: Quality of Life questionnaire before surgery



Universiteit Utrecht

Naam:

Patiëntnummer:

### Vragenlijst voor de kwaliteit van leven bij een levershunt:

#### Deel 1: voor de operatie

#### **1: Hoe vaak vertoonde uw hond de volgende verschijnselen ?**

**(voor de operatie en voordat er een medische behandeling werd ingesteld door een dierenarts)**

Zet alleen een streepje op de lijn onder uw antwoord over een verschijnsel indien uw hond dit ook vertoonde

##### **a: Toevallen** *(of epileptische aanvallen)*

A1A1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1A2 Helemaal niet |—————| Kan niet erger

##### **b: Dringen** *(met het hoofd tegen muren of dingen aanduwen)*

A1B1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1B2 Helemaal niet |—————| Kan niet erger

##### **c: Cirkelen** *(rondjes lopen)*

A1C1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1C2 Helemaal niet |—————| Kan niet erger

##### **d: Desoriëntatie** *(de weg kwijt zijn, niet goed weten waar hij naar toe moet)*

A1D1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1D2 Helemaal niet |—————| Kan niet erger



**e: Agressie** (*bijten, happen, snappen of grommen zonder reden*)

A1E1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1E2 Helemaal niet |—————| Kan niet erger

**f: Flauw vallen** (*het verliezen van het bewustzijn en in elkaar zakken*)

A1F1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1F2 Helemaal niet |—————| Kan niet erger

**g: Schommelende gang** (*moeite met normaal lopen, lopen als een dronkenman of omvallen*)

A1G1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1G2 Helemaal niet |—————| Kan niet erger

**h: Afwezigheid** (*niet of verminderd reageren op de omgeving, zitten suffen*)

A1H1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1H2 Helemaal niet |—————| Kan niet erger

**i: (Schijnbare) blindheid** (*tegen muren of obstakels oplopen*)

A1I1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1I2 Helemaal niet |—————| Kan niet erger



**j: Moeheid of zwakte** (*snel gaan liggen, geen puf hebben, veel slapen*)

A551 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A552 Helemaal niet |—————| Kan niet erger

**k: Braken** (*spugen van voedsel, gal of maagsap*)

A181 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A182 Helemaal niet |—————| Kan niet erger

**l: Diarree** (*dunnere ontlasting*)

A191 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A192 Helemaal niet |—————| Kan niet erger

**m: Niet willen eten** (*geen of verminderde eetlust*)

A131 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A132 Helemaal niet |—————| Kan niet erger

**n: Moeite met plassen** (*persen bij het plassen en/of vaak kleine beetjes plassen*)

A141 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A142 Helemaal niet |—————| Kan niet erger



**o: Bloed in de urine (rode plasjes)**

A1P1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1P2 Helemaal niet |-----| Kan niet erger

**p: Opvallend veel kwijlen (spontaan speeksel uit de bek laten lopen)**

A1P1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1P2 Helemaal niet |-----| Kan niet erger

**q: Duidelijk veel meer drinken (in combinatie met vaker en meer moeten plassen)**

A1Q1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend was dit voor uw hond?

A1Q2 Helemaal niet |-----| Kan niet erger

**A2 2: Heeft uw hond last gehad van blaasstenen of een obstructie in de urinewegen (plasbuis) waarvoor u hem of haar naar de dierenarts heeft gebracht voor diagnose en behandeling? Zo ja, kunt u hieronder wat meer details geven**

Ja Nee

**A3 3: Vond u uw hond voor de operatie te klein of te dun voor het ras en de leeftijd?**

Ja Nee Twijfel

**A4 4: Hoe actief was uw hond?**

Totaal niet actief |-----| Maximaal actief



**5: Hoe graag wilde uw hond:**

A5a

**a: spelen?**

Totaal niet | \_\_\_\_\_ | Maximaal

A5b

**b: interactie (contact) met u als eigenaar?**

Totaal niet | \_\_\_\_\_ | Maximaal

A5c

**c: zich lichamelijk inspannen?**

Totaal niet | \_\_\_\_\_ | Maximaal

A5d

**d: interactie (contact) met andere honden?**

Totaal niet | \_\_\_\_\_ | Maximaal

A6

**6: Heeft uw hond een medische behandeling (bijvoorbeeld lactulose) of een speciaal dieet gekregen voorafgaand aan de operatie?**

Nee          Dieet          Medicatie          Medicatie en dieet

*Zo ja, kunt u dit specificeren:*

A7

**7: Zo ja, kunt u op onderstaande schaal aangeven hoeveel verbetering dit gaf voor uw hond?**

Totaal geen | \_\_\_\_\_ | Maximaal

A8

**8: Indien uw hond een speciaal dieet kreeg, hoe graag at uw hond deze voeding vergeleken met de normale voeding?**

Totaal niet | \_\_\_\_\_ | Uitstekend

A9

**9: Heeft u het gevoel dat het speciale dieet de kwaliteit van leven van uw hond heeft beïnvloedt vergeleken met normale voeding?**

Totaal niet | \_\_\_\_\_ | Maximaal



**10: Indien uw hond dieet of medicijnen kreeg voor de leverproblemen voorafgaand aan de operatie, kunt u aangeven of dit een NEGATIEF effect heeft dit gehad op zijn of haar kwaliteit van leven?**

Totaal niet | \_\_\_\_\_ | Maximaal

Dank u wel voor het invullen van deze vragenlijst.

Als u vragen heeft na het invullen van deze lijst, kunt u altijd contact met ons opnemen via email: [chirurgieUKG@uu.nl](mailto:chirurgieUKG@uu.nl) onder vermelding van Onderzoek Levershunt.



2c: Quality of Life questionnaire after surgery



Universiteit Utrecht

Naam:

Patiëntnummer:

**Vragenlijst voor de kwaliteit van leven bij een leverschunt:**

Deel 2: na de operatie

**1: Hoe vaak vertoont uw hond de volgende verschijnselen?**

Zet alleen een streepje op de lijn onder uw antwoord over een verschijnsel indien uw hond dit ook vertoonde

**a: Toevallen** *(of epileptische aanvallen)*

B1A1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1A2 Helemaal niet | \_\_\_\_\_ | Kan niet erger

**b: Dringen** *(met het hoofd tegen muren of dingen aanduwen)*

B1B1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1B2 Helemaal niet | \_\_\_\_\_ | Kan niet erger

**c: Cirkelen** *(rondjes lopen)*

B1C1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1C2 Helemaal niet | \_\_\_\_\_ | Kan niet erger

**d: Desoriëntatie** *(de weg kwijt zijn, niet goed weten waar hij naar toe moet)*

B1D1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1D2 Helemaal niet | \_\_\_\_\_ | Kan niet erger



**e: Agressie** (*bijten, happen, snappen of grommen zonder reden*)

B1E1

Nooit    Minder dan 1 keer per maand    Maandelijks    Wekelijks    Dagelijks

Hoe vervelend is dit voor uw hond?

B1E2

Helemaal niet | \_\_\_\_\_ | Kan niet erger

**f: Flauw vallen** (*het verliezen van het bewustzijn en in elkaar zakken*)

B1F1

Nooit    Minder dan 1 keer per maand    Maandelijks    Wekelijks    Dagelijks

Hoe vervelend is dit voor uw hond?

B1F2

Helemaal niet | \_\_\_\_\_ | Kan niet erger

**g: Schommelende gang** (*moeite met normaal lopen, lopen als een dronkenman of omvallen*)

B1G1

Nooit    Minder dan 1 keer per maand    Maandelijks    Wekelijks    Dagelijks

Hoe vervelend is dit voor uw hond?

B1G2

Helemaal niet | \_\_\_\_\_ | Kan niet erger

**h: Afwezigheid** (*niet of verminderd reageren op de omgeving, zitten suffen*)

B1H1

Nooit    Minder dan 1 keer per maand    Maandelijks    Wekelijks    Dagelijks

Hoe vervelend is dit voor uw hond?

B1H2

Helemaal niet | \_\_\_\_\_ | Kan niet erger

**i: (Schijnbare) blindheid** (*tegen muren of obstakels oplopen*)

B1I1

Nooit    Minder dan 1 keer per maand    Maandelijks    Wekelijks    Dagelijks

Hoe vervelend is dit voor uw hond?

B1I2

Helemaal niet | \_\_\_\_\_ | Kan niet erger



**j: Moeheid of zwakte** (*snel gaan liggen, geen puf hebben, veel slapen*)

B1J1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1J2 Helemaal niet | \_\_\_\_\_ | Kan niet erger

**k: Braken** (*spugen van voedsel, gal of maagsap*)

B1K1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1K2 Helemaal niet | \_\_\_\_\_ | Kan niet erger

**l: Diarree** (*dunnere ontlasting*)

B1L1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1L2 Helemaal niet | \_\_\_\_\_ | Kan niet erger

**m: Niet willen eten** (*geen of verminderde eetlust*)

B1M1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1M2 Helemaal niet | \_\_\_\_\_ | Kan niet erger

**n: Moeite met plassen** (*persen bij het plassen en/of vaak kleine beetjes plassen*)

B1N1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1N2 Helemaal niet | \_\_\_\_\_ | Kan niet erger



**o: Bloed in de urine** (*rode plasjes*)

B1G1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1G2 Helemaal niet |—————| Kan niet erger

**p: Opvallend veel kwijlen** (*spontaan speeksel uit de bek laten lopen*)

B1P1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1P2 Helemaal niet |—————| Kan niet erger

**q: Duidelijk veel meer drinken** (*in combinatie met vaker en meer moeten plassen*)

B1Q1 Nooit Minder dan 1 keer per maand Maandelijks Wekelijks Dagelijks

Hoe vervelend is dit voor uw hond?

B1Q2 Helemaal niet |—————| Kan niet erger

B2 **2: Heeft uw hond na de operatie last gehad van blaasstenen of een obstructie in de urinewegen (plasbuis) waarvoor u hem of haar naar de dierenarts heeft gebracht voor diagnose en behandeling?** Zo ja, kunt u hieronder wat meer details geven

Ja Nee

B3 **3: Vindt u uw hond te klein of te dun voor het ras en de leeftijd?**

Ja Nee Twijfel

B4 **4: Hoe actief is uw hond?**

Totaal niet actief |—————| Maximaal actief



**5: Hoe graag wil uw hond:**

**a: spelen**

B5a

Totaal niet | \_\_\_\_\_ | Maximaal

**b: interactie (contact) met u als eigenaar**

B5b

Totaal niet | \_\_\_\_\_ | Maximaal

**c: zich lichamelijk inspannen?**

B5c

Totaal niet | \_\_\_\_\_ | Maximaal

**d: interactie (contact) met andere honden**

B5d

Totaal niet | \_\_\_\_\_ | Maximaal

B6

**6: Indien uw hond na de operatie nog steeds verschijnselen had, hoelang heeft hij/zij hier last van gehad?**

Tot 4 weken na de operatie

Minder dan 6 maanden na operatie

Meer dan 6 maanden na operatie

Nog steeds aanwezig

B7

**7: Heeft uw hond na de operatie een terugval gehad, waarbij de verschijnselen waren zoals voor de operatie? Zo ja, kunt u hierover iets meer vertellen?**

Ja

Nee

B8

**8: Krijgt uw hond op dit moment nog medicatie of een speciaal dieet? Zo ja, graag specificeren.**



**9: Vindt u dat de voedingstoestand van uw hond is verbeterd sinds de operatie?**

89a

Totaal niet | \_\_\_\_\_ | Maximaal

*Welke waarde had uw hond voor de operatie (zie bijlage):*

89b

*Welke waarde heeft uw hond nu (zie bijlage):*

**10: Hoeveel is uw hond in het geheel vooruit gegaan sinds de operatie?**

89c

Totaal niet | \_\_\_\_\_ | Maximaal

**11: Hoe tevreden bent u met het resultaat van de operatie?**

89d

Totaal niet | \_\_\_\_\_ | Maximaal

**12: Geef op onderstaande lijn aan hoe de kwaliteit van leven van uw hond was, voor de leverschunt diagnose**

89e

Zeer slecht | \_\_\_\_\_ | Uitstekend

**13: Geef op onderstaande lijn aan hoe de kwaliteit van leven van uw hond was, voor de operatie met dieet en/of medicatie**

89f

Zeer slecht | \_\_\_\_\_ | Uitstekend

**14: Geef op onderstaande lijn aan hoe de kwaliteit van leven van uw hond nu is**

89g

Zeer slecht | \_\_\_\_\_ | Uitstekend

**15: Maakt u zich nog zorgen over de gezondheid van uw hond?**

89h

Totaal niet | \_\_\_\_\_ | Heel veel

Dank u wel voor het invullen van deze vragenlijst.

Als u vragen heeft over de gezondheid van uw hond na het invullen van deze lijst, kunt u altijd contact met ons opnemen.



**Bijlage vraag 9.**



### 3a: Results of Quality of Life Questionnaire

Patient	Days between surgery and questionnaire		Quality of Life Questionnaire Scores (A and B)																	
	A1A1	B1A1	A1A2	B1A2	A1B1	B1B1	A1B2	B1B2	A1C1	B1C1	A1C2	B1C2	A1D1	B1D1	A1D2	B1D2	A1E1	B1E1	A1E2	B1E2
3	5	1	8,9	0	1	1	0	0	5	1	5,4	0	5	1	9,5	0	1	1	0	0
6	1	1	0	0	5	1	10	0	5	1	10	0	5	5	10	3,2	1	4	0	0
8	1	1	0	0	5	1	8,4	0	5	1	8,4	0	5	1	8,4	0	1	1	0	0
11	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
15 (died)	1	1	0	0	5	1	8,1	0	1	1	0	0	4	4	8,2	0	1	4	0	0
20	4	1	8	0	4	1	9	0	1	1	0	0	4	1	8,7	0	1	1	0	0
21	1	1	0	0	5	1	5,5	0	5	1	5,5	0	5	1	5,2	0	1	1	0	0
24	5	1	7,5	0	1	1	0	0	1	3	0	2,3	5	1	6,8	0	1	2	0	0
27	1	1	0	0	4	1	0,6	0	4	1	1	0	4	1	5,5	0	1	1	0	0
28	1	1	0	0	4	1	5,4	0	5	1	9,8	0	5	1	9,4	0	1	1	0	0
29	2	1	10	0	2	1	10	0	1	1	0	0	5	1	10	0	2	1	10	0
31	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
37	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
39 (died)	1	2 (1x)	0	9,4	3	1	1,4	0	1	1	0	0	1	1	0	0	1	1	0	0
42 (died)	1	1	0	0	5	1	9,7	0	5	1	9,8	0	5	1	9,6	0	1	1	0	0
45	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
49	2	1	3,4	0	4	1	5,1	0	4	1	8,5	0	2	1	4,9	0	1	1	0	0
50	1	1	0	0	1	1	0	0	4	1	8	0	4	1	7	0	1	5	0	0
51	1	1	0	0	5	1	9	0	5	1	9,2	0	5	1	9	0	1	1	0	0
52	5	2	7,6	4,2	5	3	8,1	5,2	4	2	7,2	3,4	5	2	8,3	3,2	5	4	9	0
55	1	1	0	0	5	1	2,4	0	5	1	2,7	0	4	1	3,2	0	4	1	2,8	0
60	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	3	13	5,1	0
65	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	4	0	0
66	1	1	0	0	3	1	8	0	1	1	0	0	4	1	0	0	1	3	0	0
68	1	1	0	0	1	1	0	0	1	1	0	0	4	1	7	0	1	3	0	0
70	4 of 5	1	10	0	1	1	0	0	1	1	0	0	1	1	0	0	1	5	0	0
71	1	1	0	0	1	1	0	0	5	1	10	0	5	1	10	0	1	1	0	0
73	2	1	9,5	0	1	1	0	0	3	3	1,8	0,2	1	1	0	0	1	1	0	0
74	5	1	9,5	0	1	1	0	0	1	1	0	0	5	1	7,7	0	1	1	0	0
75	1	1	0	0	4	1	8	0	4	1	8	0	4	1	8	0	1	1	0	0
76	1	1	0	0	5	1	10	0	5	1	10	1	5	0	10	0	1	1	0	0
77	5	1	9,6	0	5	1	9,7	0	5	1	9,7	0	5	1	9,7	0	1	1	0	0
78	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
81	1	1	0	0	1	1	0	0	5	1	10	0	5	1	10	0	1	1	0	0
83	1	1	0	0	1	1	0	0	1	4	0	4,7	4	5	1,8	1,9	1	5	0	0
84	5	1	6,6	0	5	2	7,2	0	5	5	5,1	6,8	5	4	5,1	7,4	5	5	5,3	0
86	1	1	0	0	5	1	8,6	0	5	1	8,6	0	5	1	9,3	0	1	1	0	0
87	1	1	0	0	3	3	0,3	0,3	1	1	0	0	1	1	0	0	1	1	0	0

1 Days between surgery and passing away

no answer or no interpretation possible  
not applicable



Patient	B1E2	A1F1	B1F1	A1F2	B1F2	A1G1	B1G1	A1G2	B1G2	A1H1	B1H1	A1H2	B1H2	A1I1	B1I1	A1I2	B1I2	A1J1	B1J1	A1J2	B1J2	
3	0	1	1	0	0	5	1	4,8	0	5	1	5	0	0	5	1	0	0	5	2	5	0,5
6	0	5	1	10	0	5	1	10	0	5	5	10	0	0	5	4	10	0	5	5	10	0
8	0	1	1	0	0	5	1	8,1	0	5	1	8,2	0	0	1	1	8	0	1	1	0	0
11	0	1	1	0	0	1	1	0	0	1	1	0	0	0	1	1	0	0	1	1	0	0
15 (died)	9	1	1	0	0	4	4	8,2	8,9	5	1	8,8	0	0	3	4	7	0	5	5	9,4	8,9
20	0	1	1	0	0	1	1	0	0	5	1	9,4	0	0	1	1	0	0	5	1	9,2	0
21	0	1	1	0	0	4	1	3	0	5	1	4,3	0	0	5	1	4,7	0	5	1	1,7	0
24	0	1	1	0	0	5	1	9,3	0	4	1	5,5	0	0	1	1	0	0	5	1	8	0
27	0	1	1	0	0	1	1	0	0	1	1	0	0	0	1	1	0	0	4	1	5,7	0
28	0	1	1	0	0	5	1	9,7	0	5	1	9,7	0	0	1	1	0	0	5	1	9,6	0
29	0	2	1	10	0	5	1	10	0	4	1	10	0	0	2	1	10	0	5	1	10	0
31	0	1	1	0	0	5	1	0	0	1	1	0	0	0	1	1	0	0	1	1	0	0
37	0	1	1	0	0	1	1	0	0	1	1	0	0	0	1	1	0	0	1	1	0	0
39 (died)	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	1	0	0	1	1	0	0
42 (died)	0	1	1	0	0	5	1	9,8	0	5	1	9,4	0	0	1	1	0	0	5	1	9,1	0
45	0	1	1	0	0	1	1	0	0	1	1	0	0	0	1	1	0	0	1	1	0	0
49	0	1	1	0	0	1	1	0	0	4 of 5	4 of 5	1	0	0	1	1	0	0	5	4 of 5	0,7	0
50	0	1	1	0	0	4	1	6	0	1	1	0	0	0	1	1	0	0	4	1	0	0
51	0	1	1	0	0	5	1	9,3	0	5	1	7,8	0	0	5	1	9,6	0	5	1	6,4	0
52	8,1	1	1	0	0	2	2	5,7	4,8	1	1	0	0	0	1	1	0	0	2	2	3,4	3,6
55	0	1	1	0	0	4	1	5	0	4	1	3,9	0	0	1	1	0	0	4	1	5,8	0
60	5,3	1	1	0	0	1	1	0	0	1	1	0	0	0	1	1	0	0	2	2	2,8	5,3
65	9,3	1	1	0	0	1	4	0	0,1	1	1	0	0	0	1	1	0	0	5	5	8,8	0
66	0	1	1	0	0	5	1	0	0	5	1	0	0	0	4	1	10	0	5	1	0	0
69	1	4	1	6,7	0	5	1	6,7	0	4	1	6,9	0	0	1	1	0	0	5	5	8,4	4
70	8,1	1	1	0	0	1	1	0	0	4	4 of 5	4,8	6,6	0	1	1	0	0	5	4 of 5	4,8	6,3
71	0	1	1	0	0	1	1	0	0	4	0	8,4	0	0	1	1	0	0	4	1	7,5	0
73	0	2	1	7,5	0	4	1	9,5	0	5	1	9,1	0	0	2	1	5,2	0	5	1	9,8	0
74	0	1	1	0	0	5	1	7,6	0	5	1	8	0	0	5	1	5,5	0	5	1	6,1	0
75	0	1	1	0	0	4	1	6	0	4	1	8	0	0	4	1	9	0	1	1	0	0
76	0	1	1	0	0	5	1	10	0	5	1	10	0	0	5	1	10	0	5	1	10	0
77	0	1	1	0	0	5	1	9,7	0	5	1	9,8	0	0	5	1	9,6	0	5	1	8,1	0
78	0	1	1	0	0	2	1	10	0	1	1	0	0	0	1	1	0	0	5	5	1,5	4,5
81	0	1	1	0	0	5	1	8,3	0	5	1	10	0	0	5	1	10	0	5	1	9,2	0
83	4,9	1	1	0	0	2	4	0,8	0	4	5	4,4	0,6	0	2	1	0,7	0	1	1	0	0
84	9,2	2	1	1,4	0	5	5	7,2	8,4	5	1	7	0	0	5	1	7,1	0	5	1	7,2	0
86	0	1	1	0	0	5	1	9,1	0	5	1	9,4	0	0	5	1	9,7	0	5	5	9,3	4
87	0	1	1	0	0	1	1	0	0	1	1	0	0	0	1	1	0	0	4	3	0,4	0,3

Patient	ATK1	BTK1	ATK2	BTK2	A1L1	B1L1	A1L2	B1L2	AMM1	BMM1	A1M2	B1M2	A1M1	B1M1	A1M2	B1M2	A1O1	B1O1	A1O2	B1O2
3	5	1	5.5	0	5	5	4.8	6.4	4	4	5.3	1.3	1	1	0	0	1	1	1	0
6	5	1	10	0	5	2	10	0	5	1	10	0	1	1	0	0	1	1	1	0
8	1	1	0	0	1	2	0	0.2	1	1	0	0	1	1	0	0	1	1	1	0
11	3	2	0.4	0	1	1	0	0	1	1	0	0	1	1	0	0	5	1	0.2	0
15 (died)	4	3	9.3	8.8	3	1	8.2	0	5	4	9.1	6.8	3	5	7.1	8	0	1	0	0
20	3	2	5.7	0.5	1	2	0	0.5	1	1	0	0	1	1	0	0	1	1	0	0
21	5	2	6.5	5.2	4	2	3.3	6.5	5	2	10	3.9	4	1	5.6	0	1	1	0	0
24	5	4	8.9	7.8	4	2	5.8	0	1	1	0	0	4	3	7	4.9	3	2	6.6	0
27	2	1	4.2	0	4	1	5	0	5	1	5.4	0	4	1	5.3	0	1	1	0	0
28	5	1	9.7	0	4	1	6.4	0	1	1	0	0	1	1	0	0	1	1	0	0
29	4	1	10	0	1	1	0	0	4	1	0	0	1	1	0	0	1	1	0	0
31	2	2	0	0	1	1	0	0	1	3	0	0	2	1	10	0	1	1	0	0
37	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
39 (died)	3	1	6.5	0	1	1	0	0	1	1	0	0	1	1	0	0	3	1	7.4	0
42 (died)	1	1	0	0	1	1	0	0	5	1	6.2	0	1	1	0	0	1	1	0	0
45	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0	1	0	0	0
49	3	1	1.5	0	1	1	0	0	5	4 of 5	1	0	1	1	0	0	1	1	0	0
50	4	2	4	0	2	1	0	0	4	1	0	0	1	1	0	0	1	1	0	0
51	1	1	0	0	1	3	0	0	5	2	3.8	0	1	1	0	0	1	1	0	0
52	2	2	6	4.8	1	1	0	0	1	1	0	0	3	2	6	3.5	1	1	0	0
55	4	1	6.8	0	2	1	0	0	2	1	0	0	3	1	4.7	0	1	1	0	0
60	4	3	1.2	1.8	3	3	1.2	0.9	2	1	4.7	0	4	2	7.4	9.3	4	2	2.6	1.2
65	1	3	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
66	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
69	4	2	3.7	2.8	5	2	8	0	4	3	8.2	8	5	5	9.6	7.8	1	1	0	0
70	3	3	5.3	5	2	2	5.4	5.4	4	1	5.1	0	1	1	0	0	1	1	0	0
71	4	1	9.6	0	1	1	0	0	1	1	0	0	5	1	9.8	0	1	1	0	0
73	5	2	9.6	5.2	5	2	9.3	5.7	5	3	9.5	5.5	5	1	9.5	0	4	1	4.4	0
74	1	1	0	0	5	1	9.8	0	5	1	7.5	0	1	1	0	0	0	1	0	0
75	3	1	7	0	1	1	0	0	4	1	6	0	1	1	0	0	1	1	0	0
76	1	1	0	0	1	1	0	0	5	1	10	0	1	1	0	0	2	1	0	0
77	1	1	0	0	1	1	0	0	5	1	9.6	0	1	1	0	0	1	1	0	0
78	2	1	1.2	0	5	1	0.2	0	1	3	0	0	1	1	0	0	1	1	0	0
81	4	1	10	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
83	2	2	0.9	0	2	2	0.7	0	2	1	0.7	0	1	1	0	0	1	1	0	0
84	4	4	5.7	8.1	4	4	6.1	7.8	5	1	6.4	0	2	1	0	0	1	1	0	0
86	5	4	9.3	3	1	1	0	0	5	1	9.4	0	5	1	9.8	0	1	1	0	0
87	2	1	4.9	0	2	1	5	0	4	4	4.8	0.3	5	1	8.1	0	5	1	0.3	0

Patient	A1P1	B1P1	A1P2	B1P2	A1O1	B1O1	A1O2	B1O2	A2	B2	A3	B3	A4	B4	A5a	B5a	A5b	B5b	A5c	B5c	A5d	B5d
3	5	1	4,5	0	4	1	2,8	0	1	1	3	3	1,8	3,3	0,3	5,6	1,1	1,1	1,2	7,4	1,1	1,1
6	1	1	0	0	5	5	0	0	1	1	1	1	10	8	10	8,1	10	10	4,5	7,9	10	10
8	1	1	0	0	1	1	0	0	1	1	1	1	1,1	9,3	1,6	9,2	0,6	9	1,4	8,9	1,2	1,2
11	1	1	0	0	1	1	0	0	1	1	1	1	9,8	9,8	10	10	10	10	10	10	10	10
15 (died)						5		6,1						7,6		6,9		9,4			6	
20	5	1	8,3	0	1	1	0	0	1	1	2	1	1,8	9,2	4,7	9,6	4,7	9,6	4,7	9,6	4,7	4,7
21	5	1	5,1	0	4	1	3,1	0	1	1	2	1	10	10	4,9	10	5,2	10	5	10	2,8	2,8
24	5	2	9,5		4	2			2	2	1	1	4,3	5,6	5	5,4	8,2	8,7	5,1	5,6	1,6	1,6
27	1	1	0	0	1	1	0	0	1	1	1	1	5,6	6,9	5,3	6,6	5,4	6,1	2,8	0	0,8	0,8
28	5	1	9,4	0	5	1	9,3	0	1	1	2	1	0,4	8,7	0,5	7,2	9,6	9,8	3,3	10	0,4	0,4
29	4	1	10	0	4	1	0	0	1	1	2	1	0	10	0	10	10	10	0	10	0	0
31	1	1	0	0	1	1	0	0	2	1	1	1	5	5	5	5	5	7,5	5	7,5	5	5
37	1	1	0	0	1	1	0	0	1	1	1	1	10	10	10	10	10	10	10	10	10	10
39 (died)	1	1	0	0	1	1	0	0	1	1	3	1	4	7,8	7,6	8,2	7,6	8,2	5,3	8,2	5,5	5,5
42 (died)	1	1	0	0	1	1	0	0	1	1	1	1	0,7	9,4	0,2	9,1	6,8	9,3	0,5	9,3	6	6
45	1				5				1		1		10		7		7		8		5	5
49	1	1	0	0	1	1	0	0	1	1	2	1	3,9	3,9	4	4	6,9	7,8	3,7	3,3	5	5
50	3	1	0	0	1	1	0	0	1	1	1	1	5	10	0	10	5	10	5	10	0	0
51	5	1	8,5	0	1	1	0	0	1	1	2	1	4,7	8,6	2	9,4	8,7	9,4	3,6	9,4	3,2	3,2
52	1	1	0	0	2	2	3,5	3,8	1	1	2	1	4	5,2	4	4,8	7,2	4,2	6,2	5,2	1,9	1,9
55	5	1	4,4	0	5	1	5,4	0	1	2	2	1	10	10	10	10	10	10	5,3	10	0	0
60	1	1	0	0	1	2	0	2,6	2	2	1	1	6,5	5,2	6,3	6,7	8,9	8,3	6,7	5	3	3
65	1	1	0	0	1	1	0	0	1	1	2	1	0	0,4	2,5	9,7	9,8	9,8	2,6	9,7	9,4	9,4
66	5	1			1	1	0	0	1	1	1	1	10	10	10	10	10	10	10	10	10	10
69	1	1	0	0	1	3	0	2,5	1	1	1	1	5,4	2,2	3,7	10	4,9	10	4,9	10	10	10
70	1	1	0	0	5	4 of 5	10	4,6	1	1	2	2	1,9	5,2	3,5	7,6	10	10	2,4	6,6	8,9	8,9
71	1	1	0	0	5	1	4,8	0	1	1	2	1	4,9	10	5,2	10	10	10	4,4	8,1	10	10
73	1	1	0	0	1	1	0	0	1	1	2	1	0,2	8	1,9	6,3	9,6	9,9	1,1	9,6	0,6	0,6
74	1	1	0	0		1			1	1	2	2	2	9,6	4,8	8,3	7,7	9,4	5,2	6,6	6,8	6,8
75	1	1	0	0	5	1	8	0	1	1	1	1	8	10	6	10	6	10	8	10	9	9
76	1	1	0	0	1	1	0	0	2	1	2	1	0	10	0	10	10	10	0	10	0	0
77	5	1	9,3	0	1	1	0	0	1	1	2	2	6,1	8,6	3,9	8,2	0,7	8,3	4,8	8,4	4,7	4,7
78	1	1	0	0	1	1	0	0	1	2	1	1	0	2,6	4,9	3,1	6,5	8,3	2,9	2,4	0,6	0,6
81	5	1	10	0	1	1	0	0	1	1	1	1	1,8	10	1	10	7,6	10	1,3	10	0,5	0,5
83	1	1	0	0	1	5	0		1	1	2	2	8,6	6,3	8,5	5,2	8,7	9,3	8,8	9,4	9,5	9,5
84	1	1	0	0	1	1	0	0	1	1	2	2	2,3	9,4	1,4	5,1	8,4	8,7	2,1	8,8	9,8	9,8
86	4	1	6,2	0	5	1	9,8	0	1	1	2	3	0	5	0,6	4,7	4,7	9,9	1,1	4,7	0,8	0,8
87	1	1	0	0	1	1	0	0	2	1	1	1	5,6	5,7	4,4	2,1	1	4,7	0,5	2,3	3,3	3,3

Patient	B50	A6	A7	A8	A9	A10	B6	B7	B8	B9a	B9c	B10	B11	B12	B13	B14	B15
3	3	1	-	-	-	-	-	1	1	7.8	-	10	10	0	-	7.3	0
6	0	4	1.7	10	4	-	-	1	1	0	4	10	10	0	0	10	1.8
8	9	2	4.3	9.8	9.5	0.2	-	1	1	0.8	0	9.9	9.9	0.2	3.2	9.8	0.1
11	10	2	0.1	10	0.1	0	-	1	1	0	0	0	10	10	10	10	0
15 (died)	0.9	1	-	-	-	-	1	2	2	9	+4	9.7	9.2	0.5	0.7	0	9.3
20	9.6	2	9.6	9.6	9.6	5	-	1	1	6.8	+4	9.8	9.8	0.7	3	9.8	0.5
21	10	2	3.9	8.5	10	0	3	1	1	10	3	10	10	0	2.9	10	1.8
24	0.9	4	4.3	7.3	1.3	1.4	2	1	1	0.8	+4	8.7	8.9	0.5	2.3	9.4	6
27	2.8	1	-	-	-	-	-	1	1	5.9	+6	9.7	9.3	0	0	8	5.4
28	5.1	4	9.4	9.3	9.5	0.4	-	1	1	10	+8	10	10	0.2	7.4	9.8	0.2
29	10	3	5	-	-	0	3	1	1	10	+6	10	10	0	0	10	0
31	7.5	1	-	-	-	-	-	1	1	-	2	7.5	7.5	7.5	-	7.5	0
37	10	1	-	10	10	0	-	1	1	-	0	-	10	10	10	10	0
39 (died)	8.2	4	0.5	9.8	0.5	9.3	-	1	1	-	+1	8.2	9.1	3.8	1.1	8.8	2.3
42 (died)	9.6	2	8.8	9.2	9.2	0	-	1	1	9.7	2	9.8	9.8	0.2	6.9	9.9	0.1
45	-	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
49	5.6	4	-	2.8	0.7	1.1	1	1	1	-	4	7.7	9.3	3.1	6.4	8.8	0.2
50	0	4	7	1	0	0.5	1	1	1	10	+4	10	10	0	0	10	2
51	9.4	4	8.1	4.9	9.5	1.5	1	1	1	9.3	+2	9.7	9.7	3.3	1.4	9.7	0.7
52	0.9	4	2.5	2.9	2.9	3.2	3	2	2	5.3	+2	5.6	6.3	0.8	0.8	6.4	8.4
55	0	1	-	-	-	-	-	1	1	-	4	10	10	0	0	10	0
60	1.9	1	0.7	5.1	0.5	0	3	1	1	7.1	+2	6.3	7.5	5	5	7.5	6.15
65	9.6	2	-	9.8	9.9	0	-	1	2	9.8	4	4.4	9.9	5	5.2	5.2	10
66	10	4	10	10	10	0	-	1	1	0	-	10	10	0	3	10	0
69	1.7	-	8.8	10	9.1	1	-	2	2	8.8	0	9	7.8	2.6	4.8	7.8	3
70	9.2	2	7.9	10	7.9	0	4	1	2	-	2	6.8	4.8	2	6.7	6.8	5.8
71	10	4	10	10	10	0	-	1	1	0	2	10	10	4.6	8.1	10	2.7
73	9.4	1	-	-	-	-	-	1	1	9.6	7	9.7	9.7	0.2	0.3	9.7	0.4
74	7	4	7.5	7.5	9.7	0.4	-	1	1	9.8	+4	9.6	9.7	0.3	2.9	9.7	0.5
75	10	2	7	9	10	0	2	1	2	-	0	-	10	2	10	9	2
76	10	4	10	10	10	0	1	1	10	10	+4	10	10	0	10	10	0
77	8.6	1	-	-	-	-	-	1	1	9.8	+2	9.8	9.7	0.4	-	9.8	0.2
78	2.4	4	9.6	9.3	8.8	0.5	-	1	2	7	+1	7	9.8	4	6.3	8.7	7
81	7.7	2	5.8	7.9	5.4	0	-	1	1	0.1	0	10	10	0	0.8	10	0
83	7.6	1	-	-	-	-	4	1	2	-	2	5.7	9.1	8.6	5.8	9.1	4
84	9.6	3	1.5	0.8	1.6	5.1	4	2	1	8.1	-	5.6	6.8	1.3	2	5.2	6.8
86	4.8	2	5.2	5	6	0	-	1	1	10	-	10	10	0	0	10	4
87	4.7	2	2.1	8.6	0.4	0.4	-	1	1	4.4	-2	7.4	9.8	9.4	8.6	8.8	0.3

Patient	Days between surgery and questionnaire																		
	A1A1	B1A1	A1A2	B1A2	A1B1	B1B1	A1B2	B1B2	A1C1	B1C1	A1C2	B1C2	A1D1	B1D1	A1D2	B1D2	A1E1	B1E1	A1E2
88	1	1	0	0	5	1	9.4	0	5	1	9.7	0	5	1	9.8	0	1	1	0
89	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
90	2	1	8.7	0	1	1	0	0	1	1	0	0	4	1	6.8	0	1	1	0
91	1	1	0	0	1	1	0	0	2	1	7	0	2	1	6.8	0	1	1	0
92	1	1	0	0	2	1	7	0	4	2	6.7	1.3	4	1	7.3	0	1	1	0
94	1	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0
95	1	1	0	0	1	1	0	0	1	5	0	9.6	1	4	0	7.1	1	1	0
96	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
97 (died)	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
98	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
104	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
109	1	1	0	0	5	1	4.8	0	5	1	3.2	0	5	1	6	0	4	5	4.5
121	1	1	0	0	5	1	0.4	0	5	4	0	0	2	1	0	0	4	5	0
122	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
123	2	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
125	5	1	0	0	5	1	0	0	5	1	0	0	5	1	0	0	5	5	0
127	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
129	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
130 (died)	1	1	0	0	4	2	2.5	0	4	2	5.1	0	4	4	6.7	5.4	1	1	0
141	2	1	3	0	1	1	0	0	1	1	0	0	3	1	2.9	0	1	1	0
146	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
147	1	1	0	0	1	1	0	0	5	1	0	0	5	1	5.5	0	1	1	0
148	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0
160	1	1	0	0	5	1	10	0	5	1	7.1	0	5	1	7.3	0	2	1	7.2
162 (died)	1	1	0	0	3	2	7.8	2.1	3	2	8.2	2.1	3	2	8.3	2.2	4	2	7.3
163	1	1	0	0	2	1	0	0	1	1	0	0	1	2	0	4.5	1	3.4	0

<sup>1</sup> Days between surgery and passing away

█

no answer or no interpretation possible  
not applicable

Patient	B1E2	A1F1	B1F1	A1F2	B1F2	A1G1	B1G1	A1G2	B1G2	A1H1	B1H1	A1H2	B1H2	A1I1	B1I1	A1I2	B1I2	A1J1	B1J1	A1J2	B1J2
88	0	4	1	5.8	0	5	1	9.5	0	5	3	8.1	1	4	1	8.8	0	5	2	9.6	2.2
89	0	1	1	0	0	1	1	0	0	4	1	5.2	0	1	1	0	0	5	1	6.2	0
90	0		1		0		1		0		1		0		1		0		1		0
91	0	1	1	0	0	2	1	7	0	2	1	7.5	0	2	1	7.4	0	2	1	7.8	0
92	0	1	1	0	0	4	2	5.8	1.1	4	1	6.7	0	3	1	7	0	4	1	6.1	0
94	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	5		3	2
95	0	1	1	0	0	1	5	0	8.5	5	5	0.4	4.9	5	1	0.4	0	5	5	9.5	9.1
96	0	1	1	0	0	5	1	8.7	0	5	1	2.6	0	1	1	0	0	5	1	7.7	0
97 (died)	0	1	1	0	0	1	1	0	0	-	1	-	0	1	1	0	0		1		0
98	0		1		0		1		0		1		0		1		0		5		0
104	0	1	1	0	0	1	1	0	0	5	1	8	0	1	1	0	0	5		5	
109	5.8	1	1	0	0	3	1	3.1	0	1	1	0	0	5	1	3.3	0	5	5	3.9	5
121	0	1	1	0	0	4	1	0	0	4	1	0	0	5	1	10	0	5	1	0	0
122	0	1	1	0	0	1	1	0	0	5	1		0	1	1	0	0	5	3	7.5	0
123	0	2	1	0	0	2	1	0	0	1	1	0	0	1	1	0	0	5	1		0
125		5				5				5				5				5			
127	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
129	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	5	4	9.1	7.4
130 (died)	0	1	1	0	0	5	4	6.5	3.3	5	4	6.3	2.3	1	1	0	0	2	2	0.7	1
141	0	3	1	4.9	0	4	1	5.4	0	4	1	5.7	0	1	1	0	0	5	2	6.8	1
146	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
147	0	1	1	0	0	5	1	5	0	5	1	4.8	0	1	1	0	0	5	5	4.2	
148	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	4	2	8.6	5.9
160	0	1	1	0	0	5	1	5.9	0	5	1	8.5	0	5	1	10	0	5	1	10	0
162 (died)	1.8	1	1	0	0	3	2	7.7	1.7	4	2	6.1	1.8	3	2	7.9	1.7	4	2	5.2	1.8
163		1	1	0	0	1	1	0	0	1	2	0	3.3	1	1	0	0	1	2	0	0

Patient	ANKT	BNKT	ANK2	BNK2	ATLT	BTLT	ATL2	BTL2	ATMT	BTMT	ATM2	BTM2	ATMT	BTMT	ATM2	BTM2	ATOT	BNOT	ANOT	BNOT
88	4	1	8,5	0	5	3	4,5	3	4	1	5,7	0	2	1	3,8	0	1	1	0	0
89	4	2	6,5	0,5	2	1	2,9	0	5	2	5,1	1,1	1	1	0	0	1	1	0	0
90	3	3	7,2	7,2	3	3	3,7	3,7	4	4	8,2	1,3	4	1	0	0	3-4	1	3,7	0
91	4	1	9,5	0	1	1	0	0	4	1	8,2	0	1	1	0	0	1	1	0	0
92	3	4	0	2,5	2	2	0	0,7	4	1	0,5	0	1	1	0	0	1	1	0	0
94	1	1	0	0	1	1	0	0	4	4	2	2,1	1	1	0	0	1	1	0	0
95	5	3	9,6	5	2	2	0,6	0,6	5	5	9,6	9,5	1	1	0	0	1	1	0	0
96	5	1	9,8	0	1	1	0	0	5	1	5,3	0	1	1	0	0	1	1	0	0
97 (died)	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	0	0	0	0
98	2	2	0	0	2	2	0	0	3	3	0	0	1	1	0	0	0	0	0	0
104	2	2	0	0	5	2	0	0	1	1	0	0	1	1	0	0	1	1	0	0
109	5	1	8,2	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
121	4	3	10	0	3	1	0	0	2	1	0	0	5	1	10	0	1	1	0	0
122	5	1	8,8	0	5	1	8,8	0	5	4	10	0	1	1	0	0	1	1	0	0
123	1	1	0	0	1	1	0	0	5	1	0	0	1	1	0	0	1	1	0	0
125	4	4	0	0	4	4	0	0	5	5	0	0	5	5	0	0	5	5	0	0
127	1	2	0	3,4	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
129	2	4	1	1	2	2	1,1	1	1	1	0	0	5	5	9	5,4	4	1	7,5	0
130 (died)	1	1	0	0	2	1	0,6	0	2	2	0,8	0	4	1	1,7	0	4	1	0	0
141	1	1	0	0	3	3	6,3	2,1	5	1	4,1	0	1	1	0	0	1	1	0	0
146	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0	1	1	0	0
147	1	1	0	0	2	2	0	0	1	1	0	0	1	1	0	0	1	1	0	0
148	3	5	6,7	6,7	2	2	0	1,5	5	1	5,4	0	1	1	0	0	0	0	0	0
160	2	1	5,2	0	1	1	0	0	5	1	10	0	1	1	0	0	1	1	0	0
162 (died)	2	1	2,5	0	5	5	7,9	6,9	4	4	3,7	3,1	4	4	5	4,8	1	1	0	0
163	4,5	2	5,5	0	4,5	2	5,5	0	4,5	2	5,9	0	1	1	0	0	1	1	0	0

Patient	A1P1	B1P1	A1P2	B1P2	A1Q1	B1Q1	A1Q2	B1Q2	A2	B2	A3	B3	A4	B4	A5a	B5a	A5b	B5b	A5c	B5c	A5d
88	5	1	9,6	0	5	1	7,2	0	1	1	2	2	2,4	6,7	3,6	7	6	9,7	2,6	9	0,3
89	1	1	0	0	5	1	10	0	1	1	2	1	0,1	10	1,6	9,4	1,9	10	1,9	10	1,8
90	1	1	0	0	5	1	6	0	1	1	2	1	3,4	8,1	3,4	8,4	3,2	8,8	3,7	8,9	6,7
91	2	1	3,5	0	4	1	0,9	0	1	1	2	1	4,4	9,6	5	9,7	9,5	9,6	4,7	9,4	9,4
92	1	1	0	0	1	1	0	0	1	1	2	3	9,6	9,3	8,5	9,7	8,7	9,6	8,8	9,7	4,7
94	1	1	0	0	5	1	8,9	2,3	1	1	2	2	1,3	6,3	2,1	4,6	8	6,8	1,6	5,2	1,7
95	1	1	0	0	5	1	6	0	1	1	3	2	0,7	1,3	2,2	1,9	8,7	8,9	0,6	1,8	5,3
96	1	1	0	0	1	1	0	0	1	1	3	2	2,2	10	0	10	10	10	0	4,9	2,1
97 (died)	1	1	0	0	5	1	10	0	1	2	3	1	4,9	4,3	4,6	4,1	4,7	4,1	4,5	4,1	4,5
98										1	1	1		5		5	5			2	
104	1	1	0	0	5	1	6	0	1	1	1	1	0	3	0	0	1	8	0	3	1
109	1	1	0	0	1	1	2,1	0	1	1	1	1	2,1	2	1,8	1	9	8,9	3	1,5	1,2
121	5	5	10	10	5	1	10	0	1	1	1	1	4	6,5	2	4	10	10	3	6	3
122	1	1	0	0	5	1	0	0	1	1	2	2	0	4	2	3,8	10	10	3,7	3,9	4,2
123	1	1	0	0	5	1		0	1	1	2		0	0	0	5,9	10	10	0	6	0
125	5				5	1			1	2	2		0		0		4,9		2,9		0,7
127	1	1	0	0	4	1	0,9	0	1	1	1	1	10	10	9,8	10	9,8	10	10	10	9,9
129	1	1	0	0	1	1	0	0	2	1	1	1	2,8	7,5	1,1	5,3	10	10	5,6	6,5	4,5
130 (died)	2	2			4	1		0	2	1	3	3	4,6	5,3	5,3	6,7	3,6	6,9	3,8	6	1
141	1	1	0	0	1	1	0	0	1	1	1	1	1,6	7,6	1,4	5,2	5,7	8,8	1,3	8,8	5,5
146	1	1	0	0	1	1	0	0	1	1	1	1		10	10	10	10	10		10	10
147	1	1	0	0					1	1	1	1	5	4	5,2	6,4	2,3	6,7	2,6	7,7	3,4
148					5	1	9,2		1	1	1	2	4,5	7	8,5	10	9,3	10	6	8	7,3
160	1	1	0	0	1	1	0	0	1	1	2	1	0,4	9,6	0	10	0	10	0,4	10	0
162 (died)	1	1	0	0	5	5	4,1	2,7	1	1	1	1	4,7	8	3,2	7,2	6	8,4	2,5	3,7	5,7
163	1	1	0	0	5	2,3	10	4,8	1	1	3	1	8,4	10	8,5	10	10	10	10	10	10



Patient	B50	A6	A7	A8	A9	A10	B6	B7	B8	B9a	B9b	B10	B11	B12	B13	B14	B15
88	3.6	4	6.7	9	9.1	0.7		1	2	7.8	+2	9.4	8.7	1.8	4.4	8	2.4
89	10	4	6.5	7.6	7.8	1	1	1	1	10	+4	10	10	0	2.3	10	0.7
90	9.8	4	7.8	9.6	7.9	0.8		1	1	8.7	+2	9.3	10	0.8	4.9	9.3	2.8
91	9.4	4	0.4	0.6	0.6	4.7	1	1	1	9.6		9.6	9.6	2.4	2.4	9.4	0.4
92	7.7	4	9.7	3.7	9.4	0.2	1	1	1	9.3	+2	9.4	9.4	1.9	7.9	9.4	6.7
94	2.4	3	0	0	0	0	-	1	1	5.8	0	9.2	9.9	1.3	-	5.4	7.8
95	8.3	2	1.9	1.8	1.9	0.7		2	1	0	0*	5.3	9.2	9.1	0.4	2.2	9.1
96	1.1	1	-	-	-	-	-	1	2	10	3	10	10	0	0	10	6.6
97 (died)	4.1	1	-	-	-	-	-	1	1	10	+2	10	10	4.4	-	10	-
98	5							1	1	7	0	7	9	0	10	10	0
104	1	2	7	0	0	0	-	1	1			7	9	0	0	8	0
109	2	4	3.1	8.2	4	2.7	1	1	1	4	+4	5.4	6.7	3.2	4.2	4.1	3.1
121	8	2	0	10	0	0	4	1	1	5	+2	10	10	0	0	10	2
122	1.7	4	2.6	2.3	3.3	0	2	1	1	7.2		8	10	0	2.4	10	0
123	5.7	1	-	-	-	-	-	1	1		+4	10	10	1.1	10	10	0
125		4	7.4	5.9	9.6	0											
127	10	1	-	-	-	-	-	1		5	0	10	10	10	10	10	2.3
129	6.4	4	5.3	10	5	0	1	1	1	7	0	8	10	1.4	2.2	8.2	1.5
130 (died)	3.1	1	-	-	-	-	4	2	2		0	4.1	4	4.1	4.4		
141	8.8	1	-	-	-	-	1	1	1	9.4	+4	8.9	8.9	0.8	0.8	9.4	0.3
146	10	1	-	-	-	-	-	-	1	-	-	-	7	10	10	10	0
147	7.6	2					1	1	1	0		6.7	8.4	2	2.2	6.9	10
148	10	1	-	-	-	-	-	1	1	9.4	+2	10	10	2.7	2.8	10	8.3
160	10	2	1.6	2	0	0	1	1	1		+6	10	10	1.1	1.6	9.3	0
162 (died)	8.3	1	-	-	-	-	4†	2	2	0.4	0	0.8	0.7	0.2	6.7	6.3	9.7
163	10	4	0	0.3	0	0	4	1	2	9.7	+2	6	8.8	2.4	2.1	10	10

**3b:** Caption for 3a, Results of Quality of Life Questionnaire (letters referring to the question are also given in appendix 2b-c)

<b>A1.1/B1.1</b> (. = A-Q)	0: Never	1: Less than once a month	2: Monthly	3: Weekly	4: Daily
<b>A1.2/B1.2</b> (. = A-Q)	The number in the table is where the owner put down an X, on a scale of 0-10				
<b>A2/B2/B7/B8</b>	1: No	2: Yes			
<b>A3</b>	1: No	2: Yes	3: Doubt		
<b>A4-A5/B4-B5</b>	The number in the table is where the owner put down an X, on a scale of 0-10				
<b>A6</b>	1: No	2: Diet	3: Medication	4: Medication and Diet	
<b>A7-A10</b>	The number in the table is where the owner put down an X, on a scale of 0-10				
<b>B6</b>	- : No signs after surgery	1: Until 4 weeks after surgery	2: Less than 6 months after surgery	3: More than 6 months after surgery	4: still remaining/ signs remained till death
<b>B9a/B10-15</b>	The number in the table is where the owner put down an X, on a scale of 0-10				
<b>B9b</b>	The body condition score given after surgery minus the body condition score before surgery				

#### 4: Results of patients at long-term check-up

Patient	Neurological signs	HE classification	Shunt anatomy	Days surgery-check-up	Fasting Bile acids $\mu\text{mol/L}$	Fasting ammonia $\mu\text{mol/L}$	Ammonia tolerance test	Urine sample	Last check-up after surgery	Shunting long-term	Recovery	Compare shunting
6	Vacant		1 PC	3689	8	29	-	Not deflecting	No shunting	No shunting	Partial recovery	No shunting
11	-		0 PC	3509	15	36	-	Not deflecting	No shunting	No shunting	Complete recovery	No shunting
20	-		0 PC	3079	3	28	-	Not deflecting	No shunting	No shunting	Complete recovery	No shunting
52	Aggression		1 PC	1900	12	42	+	Not deflecting	Asymptomatic shunting	Symptomatic shunting	No recovery	Persistent shunting
65	Lazy		1 PC	1129	172	84	+		Asymptomatic shunting	Symptomatic shunting	No recovery	Persistent shunting
66	-		0 PC	1088	1	8	-	Not deflecting	No shunting	No shunting	Complete recovery	No shunting
69	Lazy / Indolent		1 PC	928	121	28	+	Erythrocytes	Symptomatic shunting	Symptomatic shunting	No recovery	Persistent shunting
70	Aggression		1 PC	939	285	201			Symptomatic shunting	Symptomatic shunting	No recovery	Persistent shunting
71	-		0 PC	925	1	39	-	Struvite	Asymptomatic shunting	No shunting	Complete recovery	No shunting
75	-		0 PC	802	19	39	-	Not deflecting	Symptomatic shunting	No shunting	Complete recovery	No shunting
83	-		0 PC	398	269	179		Ammonia urate	Symptomatic shunting	Asymptomatic shunting	Partial recovery	Persistent shunting
87	-		0 PC	259	1	26	-	Erythrocytes		No shunting	Complete recovery	
88	-		0 PC	282	156	189		Not deflecting	Asymptomatic shunting	Asymptomatic shunting	Partial recovery	Persistent shunting
90	-		0 PC	214	68	29	-	Not deflecting	Asymptomatic shunting	No shunting	Complete recovery	No shunting
95	Compulsive movements		2 PAZ	3382	1	31	-	Not deflecting	No shunting	No shunting	Partial recovery	No shunting
96	-		0 PAZ	3255	1	31	-	Not deflecting		No shunting	Complete recovery	
106	-		0 PAZ	2075	3	30	-	Not deflecting	No shunting	No shunting	Complete recovery	No shunting
112	Lazy / Aggression		2 PAZ	1677	27	34	+	Not deflecting	Symptomatic shunting	Symptomatic shunting	No recovery	Persistent shunting
127	-		0 PAZ	445	3	13	-	Struvite	Asymptomatic shunting	No shunting	Complete recovery	No shunting
148	Lazy / Indolent		1 IH	994	28	47	+		No shunting	Symptomatic shunting	No recovery	Recurrent shunting
163	-		0 IH	1762	146	32	-	Epithelium	Symptomatic shunting	No shunting	Complete recovery	No shunting

Patient number corresponds with the patient number in appendix 1a-c.

HE classification is done by the classification system of Rothuizen (1993)

Shunt anatomy: portocaval (PC); portoazygos (PAZ); intrahepatic (IH)

The number of days between the day of surgery and the day they came back for this study

Ammonia tolerance test was called positive when there was a more than two fold increase in ammonia concentration, and this concentration was over 45 mmol/L.

No tolerance test was performed in 3 patients, because the fasting ammonia concentration was very high.

Shunting at last check-up after surgery and Shunting long-term were determined by table 17 on page 15.

Recovery was determined by table 18 on page 16.

Shunting was compared between the last check-up after surgery and the long-term check-up.