Effects of mechanical and pharmacologic manipulations on portal pressure, central venous pressure, and heart rate in dogs

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SUMMARY

Central venous pressure (CVP), portal pressure (PP), and heart rate (HR) were monitored in 6 female, sexually intact, middle-age Beagles during temporary portal vein obstruction, anesthetic recovery, abdominal bandaging, and propranolol administration. Intraoperative baseline PP was 7.3 mm of Hg (±1.7 SD). Portal pressure was significantly increased throughout portal vein occlusion, but returned to baseline values 2 minutes after release of the ligature. Central venous pressure was significantly decreased throughout portal vein occlusion, but did not differ significantly from baseline values 3 minutes after release of the portal vein ligature. Portal pressure increased significantly (8 ± 3.3 mm of Hg) over baseline values after application of an abdominal bandage; however, CVP did not change significantly. During postoperative monitoring, CVP and PP did not change significantly from respective 18-hour mean postoperative values in resting dogs. At 60 and 75 minutes after surgery, heart rate was significantly increased over the 18-hour mean. Portal pressure and CVP, respectively, were significantly increased over intraoperative baseline values in the first hour and the first 8 hours after surgery. Postoperative CVP and HR were significantly correlated. Individual measurements of PP in dogs that were abdominal pressing during barking or defecation were significantly increased (9 ± 3 mm of Hg) above measurements taken after cessation of abdominal press. Portal pressure measurements in standing dogs decreased 7.5 ± 2 mm of Hg, compared with measurements of the same dog in lateral recumbency. Central venous pressure was inaccurate in dogs performing abdominal press. Portal pressure did not decrease significantly from baseline after injection of propranolol (2 mg/kg, IV). Central venous pressure was significantly decreased at 2.5 and 3.0 hours after propranolol injection, and HR was significantly decreased from 1 to 3.5 hours after injection. Heart rate quickly returned to normal values if the dogs became excited. After propranolol administration, significant correlations were found between PP and HR, and between CVP and HR.

Received for publication Nov 2, 1989.

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Supported by the State of Ohio Canine Research Fund and the Straumann Laboratory for Surgical Research.

This report represents a portion of a thesis submitted by the senior author to the graduate school as partial fulfillment of the requirements for the MS degree.

Complete occlusion of congenital portosystemic shunts (PSS) results in portal hypertension and splanchnic venous pooling when the hepatic portal system is poorly developed or resistant to hepatopetal blood flow. Circulating blood volume is lost, potentially leading to hypovolemic or endotoxic shock and death.[1] Recent advances in anesthetic, surgical, and postoperative management of PSS have resulted in improved surgical outcomes. Several methods have been advocated to determine whether portal hypertension will develop following shunt ligation. Some methods rely on subjective evaluation by the surgeon and may not be reliable in all cases.[2] Currently, intraoperative monitoring of portal pressures (PP) is used routinely to help determine whether shunt attenuation should be partial or complete. However, intraoperative postligation PP that consistently result in patient survival, or in fatal portal hypertension, have not been determined, to our knowledge. Also, intraoperative portal pressures may vary with the depth and type of anesthesia, position of the intestines, vascular volume, or placement of the portal pressure catheter, and do not always correlate with postoperative development of portal hypertension.[1,3-7] Increases in splanchnic visceral vascular volume may not immediately cause PP changes because of the large compliance and subsequent venous pooling in the splanchnic vascular bed.[8] Little is known about systemic and cardiovascular changes that occur during and after PSS ligation in dogs that develop postoperative portal hypertension. Because central venous pressure (CVP) is a function of capacitance vessel tone, cardiac function, and blood volume, we hypothesized that CVP may be used as an indicator of splanchnic vascular pooling when portal resistance is increased and, thus, may predict postoperative hypertension following PSS ligation.

Portal hypertension may lead to several sequelae. Obstruction of intestinal venous return secondary to decreased portal flow results in mucosal devitalization, allowing transmural passage of bacteria and endotoxins. Endotoxins produce changes in vascular endothelial cells and WBC, and may cause circulatory collapse and death.[9] Postligation portal hypertension and subsequent cardio-

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vascular collapse are currently diagnosed after surgery by evaluating clinical signs because simple, reliable, inexpensive methods for monitoring postoperative PP have not, to our knowledge, been developed. Treatment of postoperative portal hypertension includes cardiovascular stabilization, systemic administration of antibiotics, and immediate removal of the PSS occlusion.[10] Pharmacologic treatments for increased PP have not, to our knowledge, been tested on dogs with postligation portal hypertension; however, in human beings with portal hypertension attributable to hepatic cirrhosis, propranolol has been used to decrease portal and wedged hepatic vein pressures.[11,a] The objectives of the study reported here were to: develop a safe method of monitoring PP during and after surgery, using a catheter that can be removed without further anesthesia or surgery; evaluate changes in heart rate (HR), CVP, and PP during temporary complete portal vein occlusion under general anesthesia; evaluate changes in these values during abdominal bandaging, anesthetic recovery, and conscious activity; determine whether correlations between these variables exist such that they may be used to indicate increased intraoperative portal vascular resistance or predict development of postoperative hypertension; and determine the effects of propranolol on PP, CVP, and HR in dogs after surgery.

Materials and Methods

Six healthy, female, sexually intact, middle-age Beagles were included in the study. Mean weights were 18 ± 1.4 kg. All dogs received humane care in compliance with the principles of laboratory animal care formulated by the *"Guide for the care and use of laboratory animals."*[12] Anesthesia was induced with a combination of 0.3 mg of diazepam/kg and 5 mg of ketamine hydrochloride/kg injected via the cephalic vein. The dogs were maintained under a surgical plane of anesthesia with isoflurane (1.5 to 2%) and oxygen (1 L/min) and were allowed to ventilate spontaneously. The ventral cervical region of each dog was aseptically prepared and an 18.5-gauge 12-inch catheter[b] was placed aseptically in the jugular vein. The catheter was threaded distal to the level of the thoracic inlet and sutured in place. A neck bandage was applied to secure the catheter, and the catheter was flushed with heparinized saline solution. The dog was placed in dorsal recumbency, and baseline CVP was measured by use of a water manometer with the zero point at the level of the thoracic inlet. Lactated Ringer solution (10 ml/kg/h) was administered via the catheter during anesthesia. Heart rate and CVP were initially monitored every 15 minutes.

The ventral portion of the abdomen was aseptically prepared for surgery. The ventral abdominal midline was incised from the xiphoid to 5 cm cranial to the pubis. The portal vein was exposed by ventral retraction of the duodenum and mesoduodenum. In 3 dogs, the portal veins were isolated and 2-0 silk suture was loosely placed around the vein cranial to the entrance of the splenic and gastroduodenal veins before placement of the transducer-tipped catheters. In 3 dogs, dissection and ligature placement were performed after catheter placement to determine whether PP was altered by dissection. A 1-cm right paramedian incision was made through the skin near the umbilicus. The abdominal wall was bluntly perforated 4 cm off midline and a transducer-tipped catheter[c] was tunnelled through the incision into the abdomen. The catheter was attached to a pressure monitor[d] and zeroed. A 1-mm incision was made at the bifurcation of an isolated terminal jejunal vein. The transducer-tipped catheter was inserted into the jejunal vein and threaded into the portal vein caudal to the encircling silk ligature. The location of the catheter in the portal vein was verified by observation of the catheter through the vessel wall and by palpation. The catheter was secured in place with 3-0 chromic gut ligatures, and 3-0 chromic gut stay sutures were placed around the vein distal to the catheter's insertion (Fig 1). The stay sutures and venous insertion site were tunnelled through the paramedian abdominal incision and secured to the external abdominal wall musculature (Fig 2). Baseline PP measurements were recorded.

A 2-0 silk ligature was passed around the portal vein cranial to the gastrosplenic vein and through a section of tubing to form a tourniquet. The portal vein was completely obstructed for 7 minutes. Central venous pressure and PP were monitored once each minute. The portal ligature was released and PP and CVP were recorded each minute (approx 8 minutes) until pressures stabilized for at least 2 recording periods. Heart rates and spontaneous respiration rates were monitored intermittently during temporary total portal vein ligation and after cessation of portal vein occlusion. The abdominal incision was closed in a routine pattern. A 3-cm piece of adhesive tape was folded over the transducer-tipped catheter and secured to the skin with nylon sutures. Anesthesia was discontinued. An abdominal bandage was gently applied, and pressure readings were immediately taken. Dogs were extubated and considered recovered when they were able to swallow voluntarily. Lactated Ringer solution was administered through the jugular catheter after surgery at 60 ml/kg/24 h. Central venous pressure, PP, HR, respiratory rate, mucous membrane color, capillary refill time,

[a] Lebrec D, Bercoff E, Menu Y, et al. Comparison of the effect of propranolol on wedged hepatic venous pressure and portal venous pressure in patients with cirrhosis (abstr). *Gastroenterology* 1983;84:1380.

[b] I-Cath, Delmed Inc, New Brunswick, NJ.

[c] Catheter, model 110-4, size 4 F 120 cm, Camino Laboratories, San Diego, Calif.

[d] Pressure monitor, model 420, Camino Laboratories, San Diego, Calif.

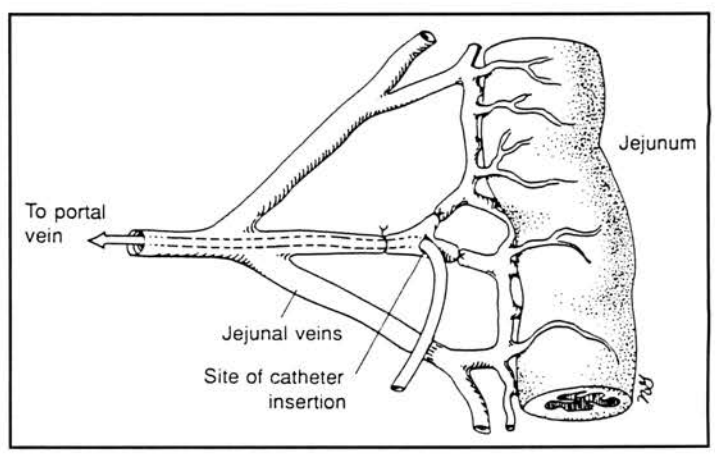

Figure 1—Top view illustration of catheter insertion. Transducer-tipped catheter is inserted through an incision at jejunal vein bifurcation and threaded into portal vein.

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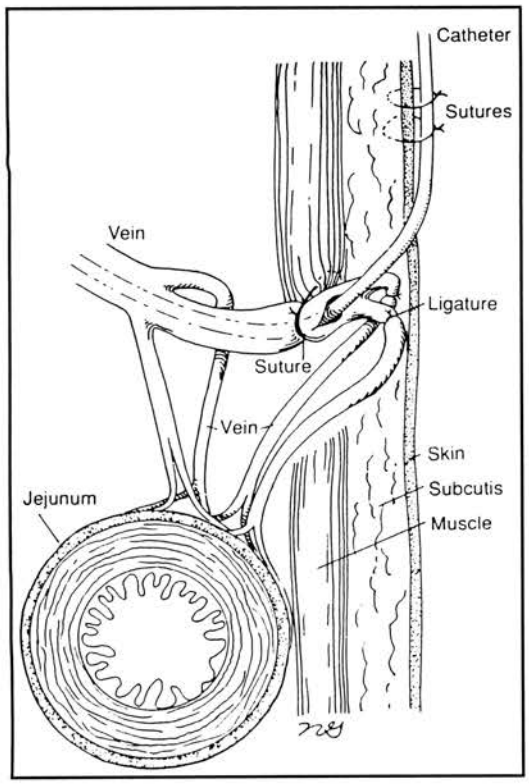

Figure 2—Side view illustration of catheter placement. Jejunal vessels are ligated, brought out through paramedian incision, and secured to body wall.

vocalization and sensitivity to abdominal palpation, and episodes of vomiting were recorded every 15 minutes for 4 hours, every 30 minutes for 4 hours, every hour for 4 hours, and every 2 hours for 6 hours, successively. The jugular catheter was flushed with heparinized saline solution after every measurement of CVP. Because of the length of time required for CVP measurements to plateau and the excessive activity level of the dogs in recumbency, CVP was measured with the dogs in standing position after surgery. Portal pressures were measured with the dogs in left lateral recumbency. One dog was excluded from monitoring after recovery because of catheter breakage.

Twenty hours after surgical recovery, dog 1 was given 1 mg of propranolol/kg IV; dogs 2 to 5 were given 2 mg of propranolol/kg, IV. With the dogs standing, PP, CVP, mucous membrane color, capillary refill time, and any episodes of vomiting, lethargy, signs of pain, or hypersalivation were recorded every 10 minutes for a half hour, every 15 minutes for 2 hours, every 30 minutes for 2.5 hours, and a final reading at 6 hours after injection. Portal catheters were removed percutaneously following completion of the propranolol study. Manual compression was applied to the paramedian insertion site for 5 minutes after catheter removal. Dogs were monitored for 2 hours for potential complications, such as hemorrhage, and then they were euthanatized with pentobarbital (3,250 mg, IV). Complete necropsies were performed. Jejunal segments at the site of the jejunal vessel ligation and dissection sites around the portal vein were grossly examined for abnormalities. Intestinal wall and perivascular portal vein tissue samples were obtained for histologic evaluation.

Data involving portal vein occlusion, abdominal bandaging, postoperative monitoring, and propranolol administration were analyzed by use of analysis of variance (repeated measures). After this analysis, all means were compared with the appropriate baseline mean *by use of* the Dunnett test. For the postoperative data, comparison was made to the intraoperative baseline mean and to the postoperative 18-hour mean.

For each set of data, correlations were made between PP, CVP, and HR by use of Pearson product moment correlation. An analysis of variance for linear/curvilinear regression was used to regress either mean PP, mean CVP, or HR on hours after each set of interventions.

Paired *t* tests were used to compare standing and recumbent measurements after surgery, and to compare measurements taken during abdominal press and during rest.

Results

Results were divided into the following categories: intraoperative manipulation and bandaging, postoperative monitoring to 18 hours, and propranolol administration.

Intraoperative baseline PP measurement was 7.33 mm of Hg (± 1.7 SD). As expected, PP did not change ($P < 0.05$) with dissection and preplacement of the silk ligature around the portal vein. Occlusion of the portal vein resulted in significant differences in PP ($F = 114.2$, $df = 7/35$, $P < 0.001$, Table 1; Fig 3 and 4) and in CVP ($F = 20.09$, $df = 7/35$, $P < 0.001$; Table 1; Fig 5 and 6). As indicated by comparisons with baseline values, for 7 minutes after occlusion, every value was significantly ($P < 0.05$) different from its respective baseline value (Table

Table 1—Results of comparing* the means of portal pressure and central venous pressure in dogs at various times after the occlusion and release of the portal vein to a baseline value and after abdominal bandaging

Minutes occluded	Mean portal pressure (mm Hg)	Mean central venous pressure (cm H_2O)
0 (baseline)	7.33	1.47
1	31.83[a]	0.21[a]
2	33.5[a]	−0.39[a]
3	34.33[a]	−0.88[a]
4	34.83[a]	−1.28[a]
5	35.17[a]	−1.45[a]
6	35.83[a]	−1.53[a]
7	35.17[a]	−1.50[a]
Minutes released		
0 (baseline)	7.33	1.47
1	9.67[a]	−0.85[a]
2	7.83	−0.07[a]
3	7.33	0.483
4	7.16	0.63
5	7.0	0.73
6	6.83	0.92
7	6.83	0.92
8	6.83	1.03
Bandage	15.33[a]	0.97

* Dunnett test.
[a] Significantly ($P < 0.05$) different than the baseline value.

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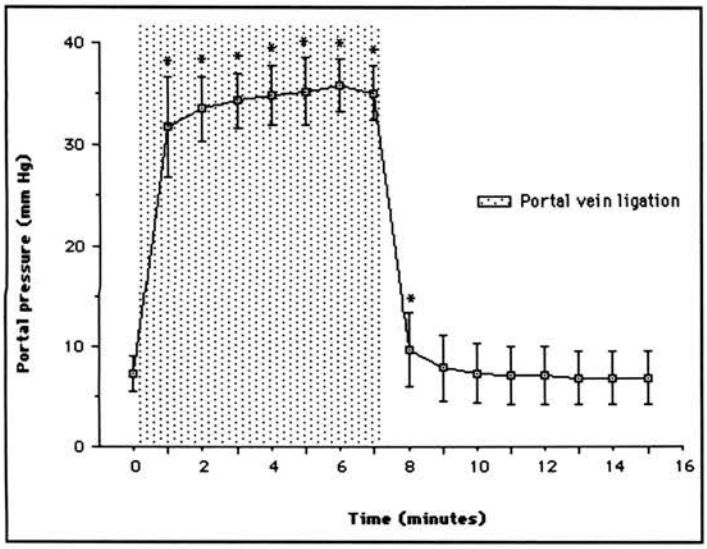

Figure 3—Mean portal pressure vs time during experimental test ligation of portal vein (n = 6). Notice significant increase * ($P < 0.05$) over baseline at 1 minute through 7 minutes of portal vein occlusion.

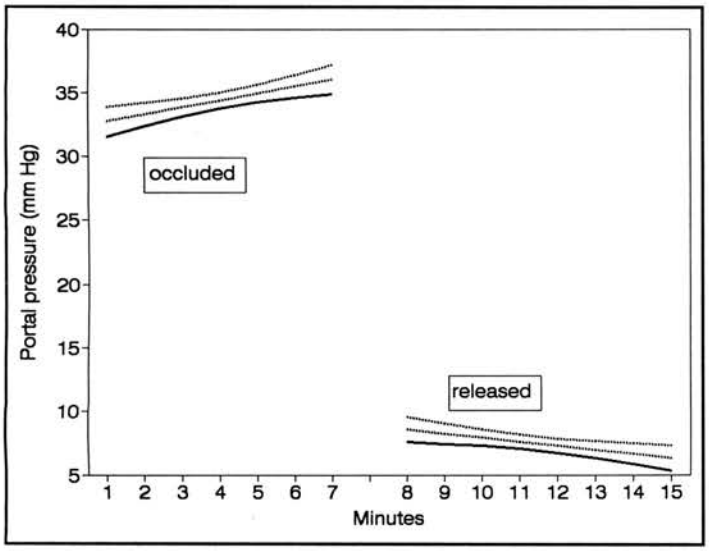

Figure 4—The regression, with 95% confidence interval, of portal pressure on minutes following portal vein occlusion and release.

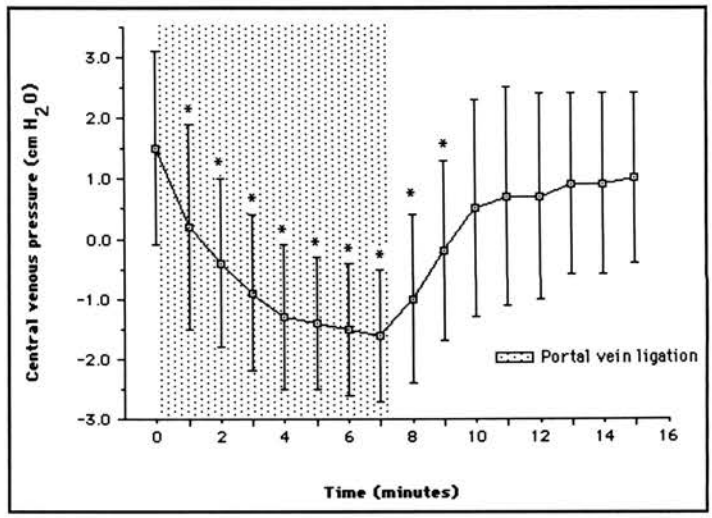

Figure 5—Mean central venous pressure vs time during experimental test ligation of portal vein (n = 6). Notice significant decrease * ($P < 0.05$) below baseline at 1 minute through 7 minutes of portal vein occlusion.

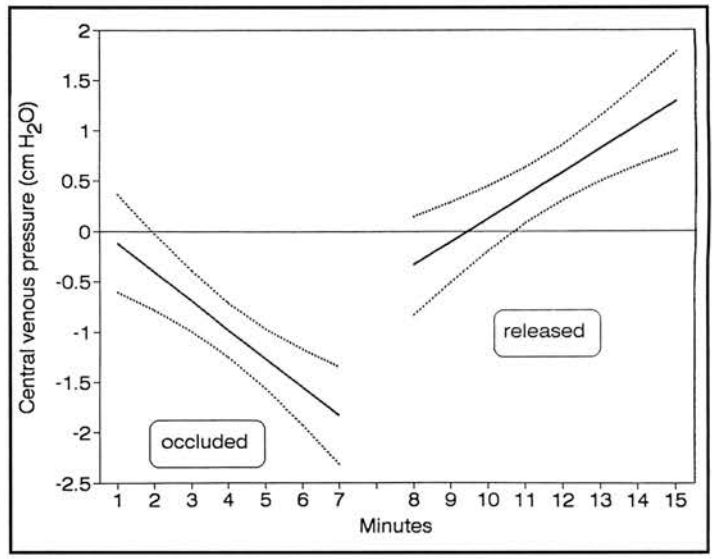

Figure 6—The regression, with 95% confidence interval, of central venous pressure on minutes following portal vein occlusion and release.

1). Upon release from occlusion, the portal vein pressure changed significantly (F = 29.24, df = 9/45, $P < 0.001$), as did CVP (F = 3.02, df = 9/45, $P < 0.01$). For PP, the values for 1 minute after ligature release and when the dogs were bandaged were significantly larger than baseline values. For CVP, the values at 1 and 2 minutes after release were significantly ($P < 0.05$) smaller than baseline values (Table 1).

When the portal vein was occluded, there was a significant (t = 4.56, df = 6, $P < 0.01$) negative correlation (−0.88) between the PP values and the CVP values. When the occlusion was released, there was almost no correlation (t = −0.10, df = 8, p = 0.28) between these 2 variables.

When the portal vein was occluded, PP regressed on time of occlusion in a significant (F = 20.3, df = 1/5, $P < 0.01$) positive linear manner (Fig 4). Central venous pressure responded negatively on time of occlusion in a significant way (F = 29.7, df = 1/5, $P < 0.005$) and regressed positively in a significant (F = 23.6, df = 1/6, $P < 0.005$) manner (Fig 6). Blanching and cyanosis of the intestinal walls, intestinal hypermotility, vascular distension, and pancreatic congestion occurred within 2 minutes of portal vein occlusion, but resolved within 5 minutes of ligature removal.

Mean PP of individual dogs averaged from 33 measurements taken during the 18-hour postoperative period ranged from 7.0 ± 4.7 mm of Hg to 15.3 ± 2.1 mm of Hg. There were significant differences in PP (F = 1.72, df = 31/124, $P < 0.025$; Table 2), CVP (F = 1.96, df = 32/128, $P < 0.01$), and HR (F = 1.172, df = 31/124, $P < 0.025$) after surgery. Compared with intraoperative baseline PP, all the values from 0 to 1.00 hour were significantly ($P < 0.05$) higher (Table 2). Compared with intraoperative baseline CVP, almost all of the values from

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Table 2—Results of comparing* the means of portal pressure, central venous pressure, and heart rate for various time increments postoperatively to their respective baseline values

Hours after surgery	Mean portal pressure (mm of Hg)	Mean central venous pressure (cm of H_2O)	Mean heart rate (beats/min)
Intraoperative baseline	7.2	1.32	...
0	16.4[a]	9.48	125.6
0.25	16.6[a]	8.36	127.2
0.50	16.1[a]	11.86[a]	142.4
0.75	16.9[a]	12.84[a]	154.4
1.00	16.9[a]	9.5	166.4[b]
1.25	12.7	9.44	165.6[b]
1.50	12.6	9.08	149.6
1.75	11.7	10.98[a]	156.8
2.00	11.3	10.44[a]	143.2
2.25	10.7	9.92[a]	152.8
2.50	10.1	11.74[a]	138.4
2.75	10.0	12.58[a]	146.0
3.00	10.2	11.12[a]	137.6
3.25	9.8	11.0[a]	152.8
3.50	9.7	11.7[a]	144.8
3.75	10.5	11.72[a]	137.6
4.00	10.4	10.08[a]	131.6
4.50	9.8	11.66[a]	145.2
5.00	10.6	11.26[a]	145.6
5.5	10.2	11.38[a]	143.2
6.0	10.2	11.36[a]	150.4
6.5	10.2	12.96[a]	138.4
7.0	9.8	10.28[a]	138.4
7.5	10.6	8.7	136.8
8.0	10.0	10.22[a]	134.4
9.0	10.4	8.12	142.4
10.0	10.4	9.38	134.4
11.0	10.4	8.84	130.4
12.0	10.2	8.48	131.6
14.0	9.9	6.36	130.4
16.0	10.3	6.84	133.6
18.0 (postoperative baseline)	10.8	8.72	120.8

* Dunnett test ($\alpha = 0.05$). This test was used to: compare every mean to the intraoperative baseline mean and compare every mean to the 18-hour baseline mean. [a] Significantly ($P < 0.05$) different from the intraoperative baseline mean. [b] Significantly ($P < 0.05$) different from the 18-hour mean.

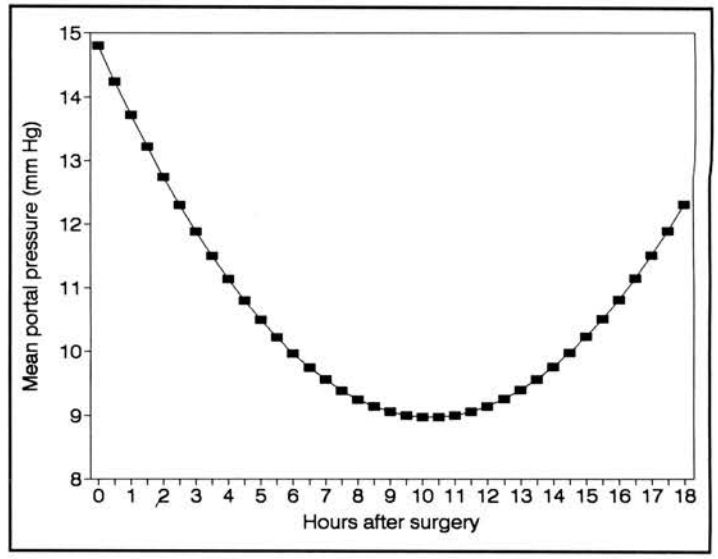

Figure 7—Curvilinear regression of mean portal pressure (mm of Hg) on hours during postoperative monitoring.

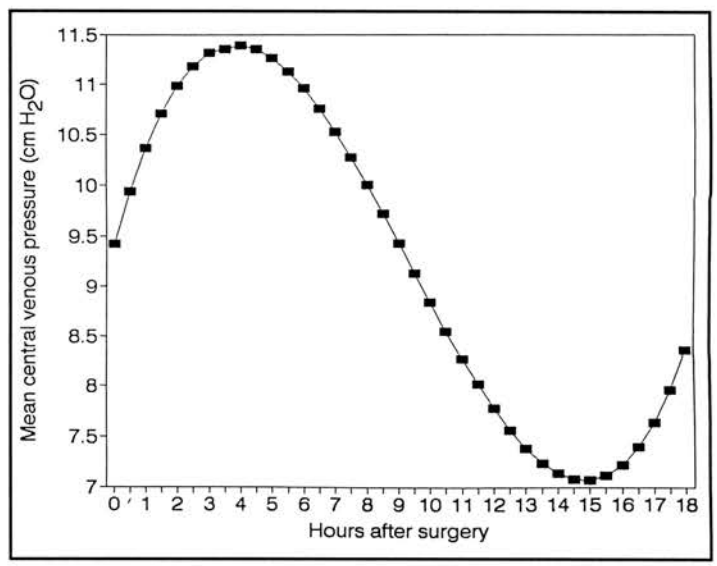

Figure 8—Curvilinear regression of mean central venous pressure (cm of H_2O) on hours during postoperative monitoring.

0.5 hour to 8 hours were significantly ($P < 0.05$) higher (Table 2). Heart rate did not change significantly from intraoperative baseline values.

Significant differences were not found between postoperative PP and CVP and their respective 18-hour mean. Heart rates at 60 and 75 minutes after surgery were significantly greater than the 18-hour mean.

Mean PP values regressed with time after surgery in a significant ($F = 21.09$, $df = 2/29$, $P < 0.001$) inverse parabolic pattern (Fig 7). Mean CVP values regressed in a significant ($F = 21.01$, $df = 2/29$, $P < 0.001$) cubic pattern (Fig 8). Mean HR regressed in a significant ($F = 22.19$, $df = 2/29$, $P < 0.001$) sixtic parabolic pattern (Fig 9).

There is a correlation of 0.37 ($Z = 2.06$, $P = 0.04$) between postoperative CVP and HR. Central venous pressure was inaccurate in dogs performing abdominal press; saline solution in the jugular catheter manometer rose 4 to 6 cm with each abdominal wall contraction, whereas it receded 1 to 2 cm in the shorter period of relaxation between abdominal presses.

Individual measurements of PP in dogs that were abdominal pressing during barking or defecation were significantly increased (9 ± 3 mm of Hg) above measurements taken after cessation of abdominal pressing. Portal pressure measurements in standing dogs decreased 7.5 ± 2 mm of Hg ($P < 0.05$), compared with measurements of the same dog taken in lateral recumbency.

Dosages of propranolol were selected on the basis of recommendations in human beings, which suggest the PP will decrease significantly when HR is decreased by 25% below resting HR, thus decreasing cardiac output.[13] Portal pressure, HR, and CVP did not change significantly in dog 1 after IV infusion of 1 mg of propranolol/kg, therefore, 2 mg of propranolol/kg was infused in dogs 2 through 5.

Analysis of PP responses to propranolol injection over time determined that there was a significant ($F = 2.92$, $df = 17/51$, $P < 0.005$) difference in these responses. The Dunnett test failed to indicate any difference between

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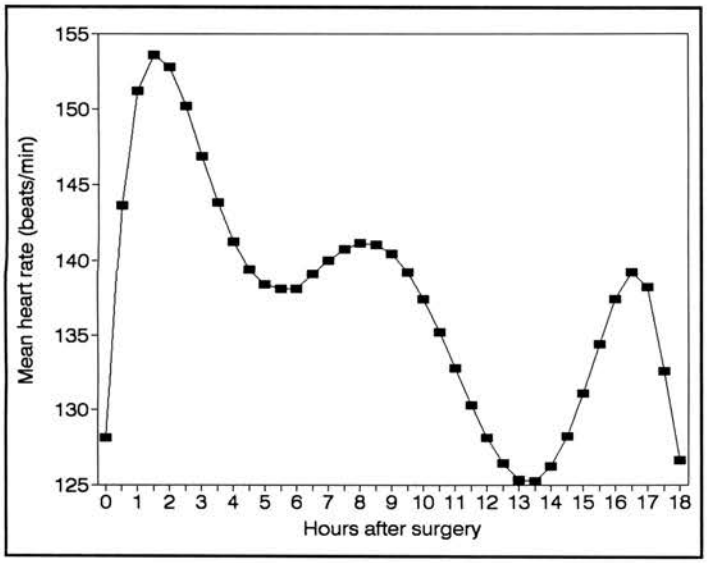

Figure 9—Curvilinear regression of mean heart rate (beats/min) on hours during postoperative monitoring.

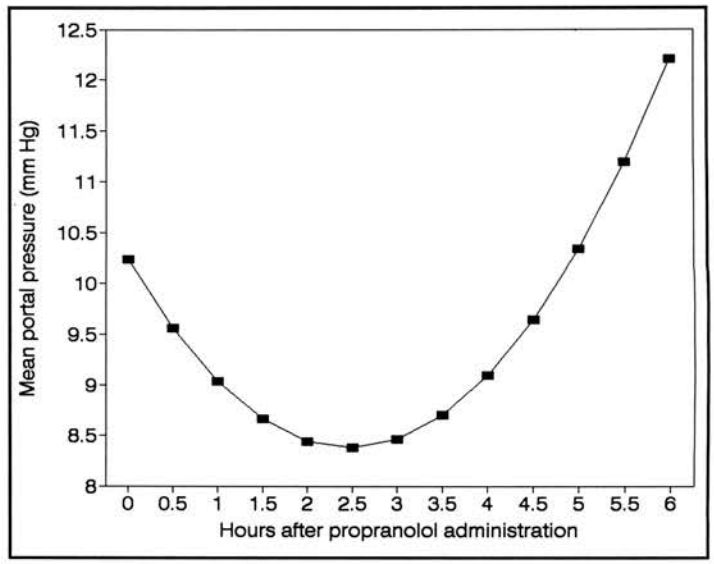

Figure 10—Curvilinear regression of mean portal pressure (mm of Hg) on hours after injection of 4 dogs with propranolol.

Table 3—Results of comparing* the means of portal pressure, central venous pressure, and heart rate for various time increments following injection of propranolol† to their respective baseline values prior to the injection

Hours after propranolol injection	Mean portal pressure (mm of Hg)	Mean central venous pressure (cm of H_2O)	Mean heart rate (beats/min)
0 (baseline)	10.5	10.5	124.0
0.2	10.4	10.2	119.0
0.35	10.6	11.9	112.5
0.5	10.0	9.4	109.5
0.75	9.6	9.2	110.0
1.00	7.9	8.3	103.0[a]
1.25	7.8	8.1	102.5[a]
1.50	8.5	8.3	102.0[a]
1.75	8.3	8.0	102.0[a]
2.00	7.8	7.4	99.0[a]
2.25	7.4	7.3	98.5[a]
2.50	8.6	6.7[a]	95.5[a]
3.00	8.5	6.3[a]	100.0[a]
3.50	9.0	7.6	103.0[a]
4.0	9.9	7.4	106.0
4.5	11.5	7.5	105.0
5.0	10.5	7.4	106.0
6.0	10.9	8.3	106.0

* Dunnett test ($\alpha = 0.05$). † Dosage = 2 mg of propranolol/kg of body weight, IV.
[a] Significantly ($P < 0.05$) different than the baseline mean.

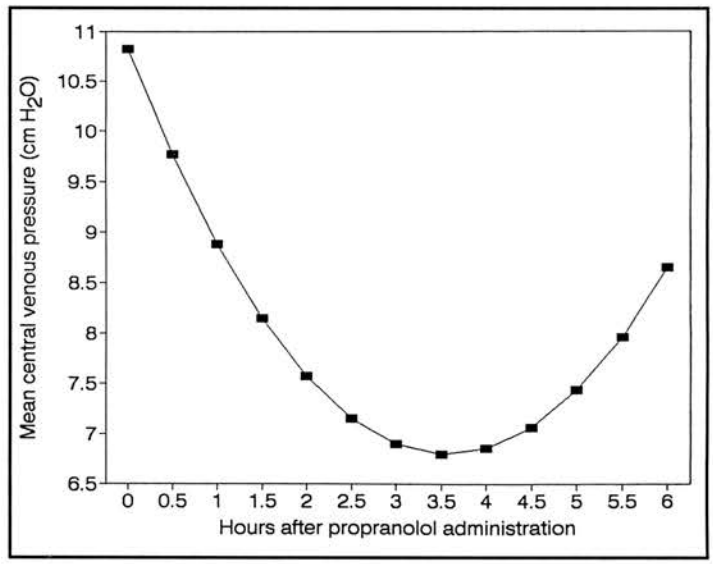

Figure 11—Curvilinear regression of mean central venous pressure (cm of H_2O) on hours after injection of 4 dogs with propranolol.

baseline PP mean and the mean of any time interval (Table 3). Mean PP regressed in a significant ($F = 10.52$, $df = 2/15$, $P < 0.005$) inverse parabolic pattern over hours after propranolol injection (Fig 10).

Central venous pressure responded to the propranolol injection in a significant ($F = 3.31$, $df = 17/51$, $P < 0.001$) pattern over time. Central venous pressure means at 2.5 and 3.0 hours after injection were significantly smaller than the baseline mean (Table 3). Mean CVP regressed in a significant ($F = 36.84$, $df = 2/15$, $P < 0.001$) inverse parabolic pattern over hours after propranolol injection (Fig 11).

After propranolol injection, HR responded significantly ($F = 2.38$, $df = 17/51$, $P < 0.05$) over time. For 1 to 3.5 hours after injection, HR regressed in a significant ($F = 19.59$, $df = 2/15$, $P < 0.001$) inverse parabolic pattern over hours after propranolol injection (Fig 12).

A correlation was not found between PP and CVP after propranolol infusion. However, there is a correlation of 0.65 between PP and HR ($t = 3.42$, $df = 16$, coefficient of determination = $r^2 \times 100 = 42.2\%$) and a correlation of 0.82 between CVP and HR ($t = 5.75$, $df = 16$, $r^2 \times 100 = 67.2\%$). Those correlation values are highly significant ($P < 0.001$).

Portal pressure and HR quickly increased if the dogs became excited. Three dogs became lethargic and weak 1.25 hours after propranolol administration, and 2 dogs developed hypersalivation.

Complications were not noticed after prolonged portal

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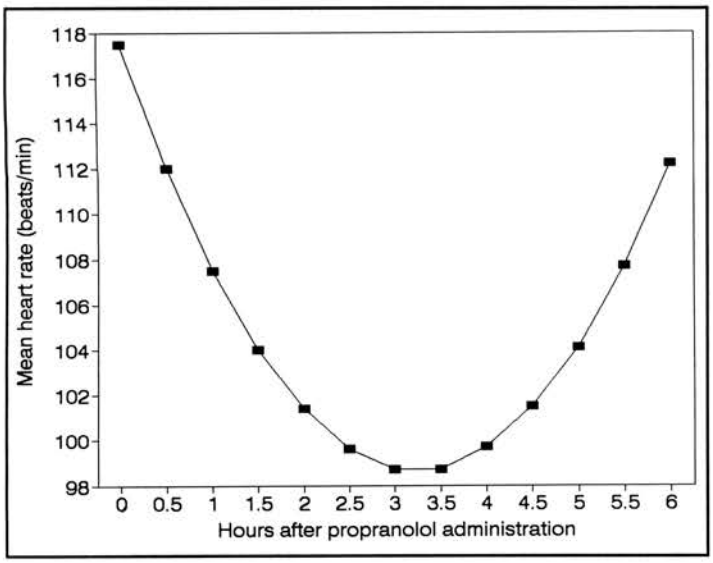

Figure 12—Curvilinear regression of mean heart rate (beats/min) on hours after injection of 4 dogs with propranolol.

vein catheterization and percutaneous catheter removal. Minimal hemorrhage was found around the paramedian area where the cathether was secured. Other gross lesions were not noticed during necropsy. Histologic evaluation was performed by a board-certified pathologist. Microscopic examination of the portal vein revealed mild focal areas of perivascular inflammation. Abnormalities associated with temporary portal vein occlusion were not noticed in the intestines or pancreas.

Discussion

During surgery, PP significantly increased and CVP significantly decreased during portal vein ligation, whereas HR remained unchanged. Portal venous pressures rapidly increased and plateaued because of iatrogenically increased prehepatic portal venous resistance, resulting in intraoperative signs of portal hypertension, which have been previously described.[2] Increased portal resistance and portal hypertension resulted in rapid decrease in CVP, which may be explained by changes in venous return and splanchnic venous capacitance. Central venous pressure is an indirect measure of right atrial pressure and cardiac preload and, thus, is controlled by the tone of capacitance vessels and the intrathoracic pressure when right ventricular function is adequate.[3] Normal CVP is approximately 0 cm of H_2O, which is equivalent to extracorporeal atmospheric pressure. Central venous pressure will decrease toward intrapleural pressure (-4.1 to -6.8 cm of H_2O) when heart contractions are exceptionally vigorous or when venous return is decreased.[5] Sudden obstruction of the hepatopetal portal system leads to a sudden decrease in caudal vena caval flow. For a given increase in hepatic venous resistance, the decrease in venous return is 8 times greater than when arterial resistance is increased by the same amount, because of the larger total capacitance of the veins.[5,8] This change in venous tone, which compensates for the change in venous volume, results in splanchnic pooling with continued decrease in venous return and decrease in CVP. Eventually, enough-volume is added to the capacitance vessels to reach an elastic recoil pressure that exceeds right atrial pressure, reestablishing venous return if prehepatic portal obstruction is not present.[3-5,8,14-16] Heart rate remained unchanged, possibly because of effects of anesthesia. Under isoflurane anesthesia, baroreceptor reflexes may be attenuated, and cardiac output may change without concomitant alterations in HR.[17,e,f]

Abdominal bandaging did not change CVP or HR, but did increase PP significantly. When the abdominal cavity is closed, studying venous and intra-abdominal pressures alone will not indicate the amount of portal blood flow. However, portal venous pressure must be greater than intra-abdominal pressure to allow hepatopetal portal flow.[18,19] An increase in intra-abdominal pressure of 25 to 39 cm of H_2O (18 to 28 mm of Hg) in dogs has been shown to decrease portal flow 70 to 93%.[18] With a closed abdomen, splanchnic blood flow is reduced because of increased resistance in the intestinal, mesenteric, portal, and hepatic veins, and inferior vena cava.[18] High intra-abdominal pressure (30 to 35 cm of H_2O) also results in hepatic compression, decreased portal vascularity, and decreased portal vein diameter.[18] Thus, although we cannot conclude from our study that portal flow was affected by increased PP of 8 to 9 mm of Hg attributable to bandaging or abdominal press, we suggest that abdominal compression resulting in large increases in intra-abdominal pressure should be avoided because of previously documented effects on splanchnic and portal venous resistance and blood flow.

After surgery, CVP was inaccurate and HR was increased during periods of abdominal press. Increased intra-abdominal and intrathoracic pressures during forced expirations result in large rapid elevations in CVP, with relatively prolonged recession of saline solution values in the manometer during subsequent inspiration.[3,18] Postoperative PP in recumbent dogs did not change significantly; however, a trend toward high PP immediately following recovery was noticed, with gradual decrease and stabilization of PP over time. In the immediate postoperative period, barking with abdominal pressing during anesthetic recovery was noticed. Portal pressures with the dogs relaxed were not available in many of these animals; therefore, high PP attributable to high intra-abdominal pressure were recorded and included in the data. Portal pressures with the dogs standing were significantly decreased, compared with PP measurements taken with dogs in lateral recumbency. Abdominal visceral and vascular compression may occur in recumbent dogs, causing increases in PP. Further studies are needed to compare postoperative PP with abdominal pressure and portal flow to determine effects of abdominal bandaging and portal hypertension on these measurements. Because of the aforementioned problems, reliable percutaneous PP monitoring in clinical patients after shunt ligation may not be feasible with this system of measurement. Wedged hepatic venous pressure measurement gives an indirect estimate of portal venous pressure, averaging 1 cm of H_2O higher than concomitant portal venous pressure

[e] Gelman S, Fowler K, Smith L. Cardiac output distribution and regional blood flow during isoflurane anesthesia (abstr). *Anesth 1983;59:A68.*

[f] Van der Linden P, Gilbart E, Engelman E, et al. Hemodynamic effects of anesthesia in a canine septic shock model: comparison of four anesthetic agents (abstr). *Anesth* 1987;67:A413.

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measurements in dogs.[20] However, heavy sedation or light anesthesia necessary to perform the technique may alter PP measurements.

Administration of propranolol (2 mg/kg, IV) resulted in transient decrease in HR and CVP, but no change in mean PP. Propranolol is a synthetic nonselective beta-adrenergic receptor blocking agent. The peak effect of intravenously administered propranolol hydrochloride is 1 to 1.5 hours.[21] Propranolol has been successfully used in treatment of chronic hypertension in human beings. Several mechanisms are thought to contribute to its antihypertensive effects. Decrease in cardiac output caused by propranolol secondary to decreased HR will eventually be followed by decrease in peripheral pressure. Propranolol blocks the release of norepinephrine initiated by β-agonists at adrenergic nerve endings, decreasing sympathetic nerve stimulation. In man and other animals, propranolol has been shown to decrease portal pressures in patients suffering from portal hypertension attributable to cirrhosis and has advantages over other drugs because it can be given orally, has a prolonged effect, and does not induce many serious side effects.[4,13,22] Intravenous administration of a single dose of propranolol was not shown to have a prolonged effect on PP in this study.

Portal venous pressures are determined by hepatic vascular resistance to blood perfusion, portal venous tributary flow, and portal and splanchnic venous compliance. Propranolol decreases portal venous pressures by diminishing the flow within portal tributaries by decreasing cardiac output via blockade of β-cardiac receptors, and by its extracardiac β-blocking effects on such sites as the spleen, where it may cause vasoconstriction.[23] In man, propranolol significantly decreases mean systolic and diastolic pressures, resting pulse rates, wedged hepatic venous pressure, and the gradient between wedged and free hepatic venous pressure.[24,a,g,h] It is questionable whether propranolol induces the same effects in dogs. One study reported significant changes of systemic hemodynamics following propranolol treatment in dogs with intrahepatic portal hypertension, but only minimal effects on hepatic circulation (PP, wedged and free hepatic pressures, and portal blood flow).[25] Portal flow and vascular resistance will not be directly affected by propranolol because of the absence of β-receptors in the portal tributaries.[11,16] In our study that used dogs with normal PP, effects of propranolol were easily overridden by sympathetic stimulation, and the degree of lethargy in 3 of 4 dogs was severe. Studies were not performed on dogs with portal hypertension following shunt ligation; therefore, no recommendations can be given as to its use in clinical situations. However, because propranolol decreases cardiac output, it may be detrimental in portal hypertensive patients when cardiac preload is already reduced. Because the portal vein contains α-receptors, further research should be done to determine whether α-receptor blockers will decrease resistance in the portal venous tributaries without detrimental side effects.[11,16]

Intraoperative CVP may be used to indicate reduced portal flow attributable to increased hepatic venous resistance during test occlusion of the portal vein. Thus, measurement of CVP may potentially be useful during occlusion of single congenital portal systemic shunts because it may indicate which animals have high portal resistance or which animals might be predisposed to developing postoperative portal hypertension. Abdominal bandages should be avoided in animals with increased risk of portal hypertension. Postoperative portal pressures can be monitored easily and safely with transducer-tipped catheters. If combined with concomitant measurement of intra-abdominal pressure, this method of PP measurement may be of value in monitoring animals for postoperative portal hypertension after PSS ligation. Portal catheters can be removed percutaneously from the conscious animal with minimal complications. Portal pressures are affected by intra-abdominal pressure and positioning of the animal. Portal pressure measurements do not directly indicate the amount of portal blood flow, but may demonstrate trends in PP values during cardiovascular and hemodynamic stability. Propranolol has minimal indirect transient effects on PP in clinically normal dogs and its effect on cardiac output and its side effects may preclude its use in dogs with portal hypertension following PSS ligation.

[g] Rector WG. Propranolol for portal hypertension (abstr). *Hepatology* 1982;2:698.

[h] Nakayama T, Ohnishi K, Saito M, et al. Effects of propranolol on portal vein pressure, portal blood flow, hepatic blood flow, and cardiac output in patients with chronic liver disease (abstr). *Hepatology* 1983;3:812.

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