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Effect of Abdominal Distension on Central and Regional Hemodynamics in Neonatal Lambs

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ABSTRACT. Elevations of intraabdominal pressure (IAP) can occur during surgical repair of gastroschisis and omphalocele and lead to ischemia of abdominal organs. We examined the effect of elevated IAP on central hemodynamics and regional abdominal organ blood flow, measured by radiolabeled microspheres, in 11 pentobarbital-anesthetized neonatal lambs. Stepwise increases in IAP were obtained by inflating a large bag placed intraperitoneally with air to pressures of 15, 20, and 25 mm Hg. Measurements were made at 30 min of elevated IAP and 30 min after deflating the bag. Mean aortic pressure was not significantly altered at an IAP of 15 mm Hg (78 ± 4 mm Hg) (±SE) or 20 mm Hg (76 ± 4 mm Hg) compared to baseline (81 ± 4 mm Hg), but was decreased at the highest IAP (68 ± 5 mm Hg). Stepwise decreases in blood flow to all abdominal organs, except adrenal gland, occurred with elevated IAP, and blood flows to these organs (except spleen) returned to or above baseline on bag deflation. At IAP of 15, 20, and 25 mm Hg, cardiac output was reduced by 14, 21, and 35%, respectively. Similar percent reductions of renal blood flow occurred. However, regional gastrointestinal blood flow decreased by a greater extent (35, 50, and 54% at each respective IAP). Hepatic arterial blood flow more than doubled at each IAP, but this was not sufficient to maintain total liver blood flow, or presumably total oxygen delivery to liver. The marked curtailment of splanchnic blood flow suggests that prolonged periods of elevated IAP at or above 15 mm Hg should be avoided in repair of gastroschisis and omphalocele or in other situations that can produce profound abdominal distension, such as swelling from liver transplantation or gaseous distension. (*Pediatr Res* 19: 1244–1249, 1985)

Abbreviations

IAP, intraabdominal pressure
GI, gastrointestinal

Surgical repair of gastroschisis and omphalocele carries the risk of placing the abdominal contents under pressure (1, 2). Pathophysiological sequelae may occur. Impairment of cardiovascular dynamics with elevated abdominal pressure include hypotension (3) and reduction in cardiac output (3, 4); respiratory function may deterioriate with loss of ability to maintain adequate gas exchange and acid-base equilibrum (3), necessitating the use of postoperative ventilation (5, 6). Ischemic bowel problems have also been observed (5, 7–9). Attempts have been made to reduce these effects of performing staged repairs to limit the rise in IAP; however, these carry increased risk of infection (5, 8–11) and necessitate repeat anesthetic and surgical procedures.

Studies in adult animals indicate that renal blood flow and function are impaired when IAP is elevated (12, 13). However, less is known about regional splanchnic blood flow, particularly in the neonate, or whether there is an IAP threshold that could be tolerated without curtailing abdominal organ blood flow. Such knowledge may help guide decisions concerning the number of staged repairs which could be tolerated. This study was designed to examine cardiovascular and pulmonary function during acute, graded increases in IAP in the neonatal lamb. Regional organ blood flows were studied specifically to determine if raised IAP caused any selective alteration in perfusion to abdominal organs. We examine whether a significant IAP threshold exists at which blood flows are maintained and ischemic bowel problems are less likely to occur.

METHODS

Eleven lambs of both sexes, 2.7 to 7.9 kg in weight, and 3 to 14 days old, were anesthetized with intravenous sodium pentobarbital (25 mg/kg), intubated, paralyzed with pancuronium (0.1 mg/kg), and ventilated with a Harvard small animal ventilator. End-tidal CO_2 was maintained at 4.5 to 5.0% throughout the experiment. Temperature was monitored with a rectal temperature probe and maintained at 39 ± 0.5° C with infrared heating lamps. Catheters were placed in the abdominal aorta via a femoral artery, in the inferior vena cava via a femoral vein and in both subclavian arteries via the brachial arteries. A 5 French Swan-Ganz catheter was advanced into the pulmonary artery via a femoral or external jugular vein. For injection of microspheres, a left ventricular catheter was inserted via the other femoral artery. The position of all lines was checked at autopsy. A nasogastric tube with esophageal balloon was passed into the stomach. Simulation of a repair of gastroschisis or omphalocele was produced by spreading a 3-liter anesthetic reservoir bag intraperitoneally over most of the ventral surface and lateral aspects of the abdomen. The midline incision was sutured closed and the lambs were studied in the supine position. To increase IAP, the bag was inflated with a volume of air that was less than the unstressed volume of the bag. Although IAP was not directly measured, intrabag pressure was assumed to reflect IAP along the outer surface of the unstressed bag. IAP surrounding distal organs may have differed somewhat from intrabag pressure

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because the abdomen does not behave perfectly as a fluid-filled container.

Arterial blood pressure, intraabdominal inferior vena cava pressure, intrathoracic central venous pressure (from the central venous pressure port of the Swan-Ganz catheter), pulmonary artery and wedge pressures, airway, esophageal, and intragastric and intraabdominal (intrabag) pressures were recorded. All pressures were referenced to atmosphere at the level of the right atrium. Cardiac output was measured by the thermodilution technique using the average of triplicate determinations of 3 ml of iced saline injectate. Others have validated this technique in lambs (14). Systemic vascular resistance was calculated using central venous pressure as the downstream pressure. Arterial blood gases and pH were measured with Radiometer BMS3 electrodes and analyzer, and arterial O_2 content was measured with a CO-Oximeter (Instrumentation Laboratories).

For measurement of regional cerebral and peripheral blood flow, approximately 1.3×10^6 radiolabeled microspheres (15 ± 1.5 μ in diameter) were injected into the left ventricle over 30 s, as previously reported from this laboratory (15, 16). Arterial reference samples were simultaneously withdrawn from both the subclavian artery and abdominal aortic catheters at a rate of 2.5 ml/min. The subclavian arterial reference sample was used in the calculation of cerebral blood flow, and the abdominal aortic reference sample was used in the calculation of blood flow to abdominal organs. A random sequence of five different isotopes were used: ^{153}Gd, ^{113}Sn, ^{103}Ru, ^{95}Nb, and ^{46}Sc (New England Nuclear). Following each reference sample withdrawal, transfusion of the animal with an equal volume of adult sheep blood was made to maintain circulating blood volume.

After killing the animal by intravenous injection of a saturated solution of potassium chloride, the following entire organs were dissected and placed in scintillation vials: kidney, liver, spleen, stomach, duodenum, jejunum and ileum, large bowel, pancreas, omentum, mesentery, adrenals, diaphragm, and brain (dissected into cerebellum, brain stem, and hemispheres). The radioactivity was analyzed in a Packard Auto-Gamma Scintillation Spectrometer (model 9042) and the overlap of activity from the high energy to the low energy isotopes was subtracted to obtain corrected counts for each isotope (C_t) (17). Blood flow to each organ was calculated as $\dot{Q}_t = \dot{Q}_r \times C_t/C_r$ where $\dot{Q}_r$ is the arterial reference sample withdrawal rate and C_r the reference sample counts.

The experimental protocol consisted of first taking a set of baseline measurements with the bag deflated at 0 mm Hg. Intraabdominal bag pressure was then elevated stepwise to 15, 20, and 25 mm Hg, allowing 30 min equilibration at each pressure before taking another set of measurements. Pressure in the bag was then returned to baseline (0 mm Hg), and measurements were again obtained after 30 min equilibration.

In three additional animals the same protocol was followed, inserting the bag intraperitoneally, but not inflating it. Pressure and flow measurements were followed over a 2-h period after obtaining baseline measurements. These animals represented the time control series.

All results were analyzed for statistical significance at the $p < 0.05$ level using analysis of variance. Comparisons with baseline measurements were made with the Duncan multiple range test. Values are presented as means ± SE.

RESULTS

Raising intraabdominal bag pressure (IAP) to 15, 20, and 25 mm Hg produced a graded decrease in cardiac output of 14 ± 5, 21 ± 8, and 35 ± 4%, respectively, from a control value of 0.99 ± 0.11 liter/min. This was the result of a decrease in both stroke volume and heart rate, both of which returned to baseline 30 min after deflating the bag (Fig. 1). Mean aortic pressure fell significantly from 81 ± 4 to 68 ± 5 mm Hg at the highest IAP, but did not completely return to baseline at 30 min of recovery.

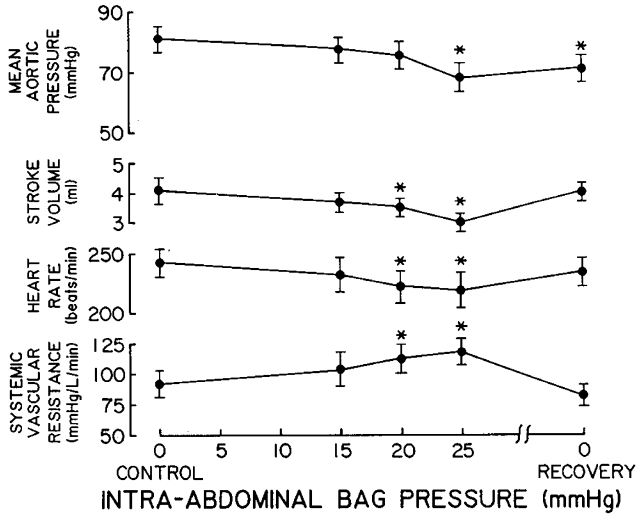

Fig. 1. Responses of mean aortic pressure, stroke volume, heart rate, and systemic vascular resistance to increased IAP. Values are means ± SE (*bars*). *Asterisks* indicate significant differences from control.

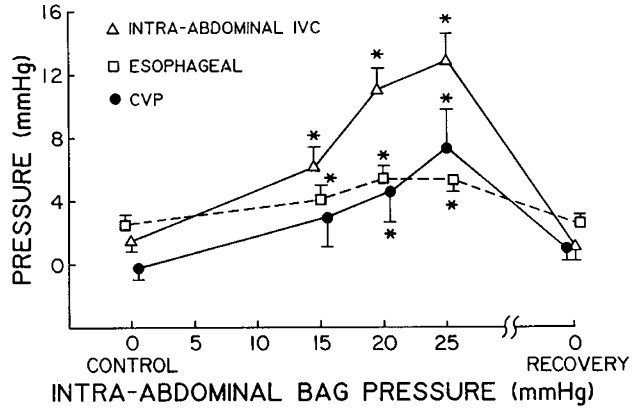

Fig. 2. Responses of intraabdominal inferior vena cava (*IVC*) pressure, esophageal balloon pressure, and intrathoracic central venous pressure (*CVP*) to increased intraabdominal pressure. Values are means ± SE (*bars*). *Asterisks* indicate significant differences from control.

Calculated systemic vascular resistance was elevated by 23 and 29% at 20 and 25 mm Hg IAP, respectively.

Reversible increases in abdominal inferior vena caval pressure, central venous pressure, and esophageal balloon pressure were observed with bag inflation (Fig. 2). The increase was greater in abdominal vena cava pressure than in intrathoracic central venous pressure, which in turn was greater than the increase in esophageal presure. Intragastric pressure rose from 3.3 ± 0.5 to 5.5 ± 1.0 mm Hg at the highest bag inflation. This rise was parallel to esophageal balloon pressure rather than to intraabdominal bag pressure.

Pulmonary wedge pressure increased essentially in parallel with esophageal balloon pressure (Fig. 3). Peak airway pressure on intermittent positive pressure ventilation also increased. There were no significant changes in systolic, mean, or diastolic pulmonary artery pressure. There were no changes in arterial blood gas tensions or oxygen content, but a metabolic acidosis developed during bag inflation (Table 1).

Regional blood flow to all tissues sampled are presented in Table 2. Relative responses, expressed as a percent of control, of selected organs are compared in Figure 4 and 5. Blood flow to all abdominal organs except the adrenal gland, decreased in a graded fashion to elevated IAP. Blood flow then returned to

baseline or above baseline in all of these organs, except spleen, 30 min after deflating the bag (Table 2). Adrenal blood flow was sustained with elevated IAP, but then increased during the recovery phase. Brain blood flow, including blood flow to cerebellum, brainstem, and cerebral hemispheres, remained unchanged indicating intact autoregulation (Table 2).

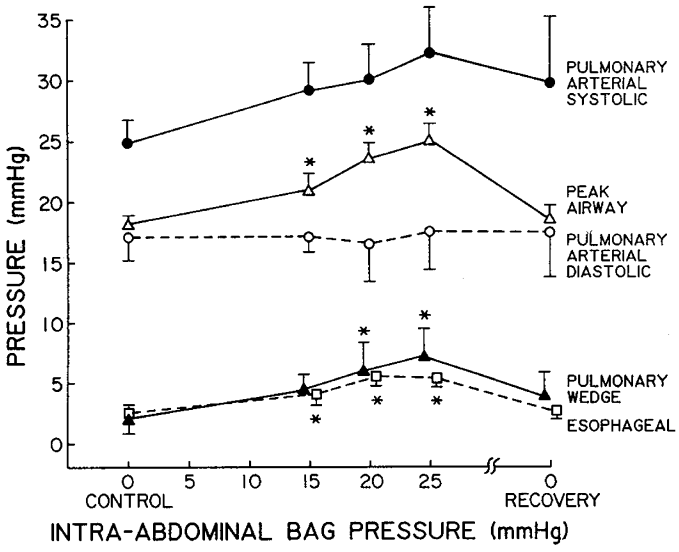

Fig. 3. Responses of pulmonary arterial systolic and diastolic pressures, peak airway pressure on intermittent positive pressure ventilation, pulmonary wedge pressure, and esophageal balloon pressure to increased IAP. Values are means ± SE (*bars*). *Asterisks* indicate significant differences from control.

Portal vein blood flow was estimated by summing blood flow to the entire GI tract (including spleen). This approximation does not take into account transient, intrinsic volume changes or transcapillary fluid shifts. Portal vein blood flow decreased at each elevation of IAP and was reduced by more than half at the highest IAP (Fig. 4). The decrease was also significant when expressed as a percent of cardiac output. Hepatic arterial blood flow (as assessed by microsphere concentration in the liver) more than doubled by 15 mm Hg of IAP. Nevertheless, total liver blood flow was markedly depressed at all three bag inflation pressures (Table 2). Renal blood flow also fell, but the percent fall was not significantly greater than the percent decrease in cardiac output (Fig. 4). At 15 mm Hg of IAP, renal blood flow was better preserved than gastrointestinal blood flow. Within the GI tract, the decrease in blood flow was similar among organs (Fig. 5). However, splenic blood flow decreased substantially more than other abdominal organs.

A graded decrease in diaphragmatic blood flow down to 55 ± 7% of control occurred, which then returned to control on bag deflation. However, in the time control lambs in which the bag was inserted but not inflated, diaphragmatic blood flow was decreased to 64 ± 3% of control at the fourth measurement time and remained depressed at the fifth time point. Thus, the interpretation of diaphragmatic blood flow is not clear. No other organs had significant alterations in the time control group.

DISCUSSION

This study demonstrates that stepwise increases in IAP produced by distending an intraperitoneal bag in neonatal lambs resulted in: 1) graded decreases in cardiac output, the magnitude of which was not totally reflected by solely measuring central venous or arterial pressure, 2) graded decreases in blood flow to all abdominal organs, except adrenal glands, 3) a greater reduc-

Table 1. *Arterial blood gases and pH with increased IAP (mean ± SE)*

IAP (mm Hg)	0 Control	15	20	25	0 Recovery
pH	7.42 ± 0.015	7.40 ± 0.015	7.31 ± 0.015*	7.31 ± 0.024*	7.33 ± 0.015*
$Paco_2$ (mm Hg)	35 ± 1	33 ± 1	33 ± 1	34 ± 1	33 ± 2
Pao_2 (mm Hg)	70 ± 6	69 ± 4	69 ± 5	65 ± 5	73 ± 4
CaO_2 (vol %)	14.4 ± 0.8	14.3 ± 0.8	14.4 ± 0.9	14.0 ± 0.8	13.9 ± 0.8

* $p < 0.05$ from control; $Paco_2$, arterial CO_2 tension; Pao_2, arterial O_2 tension; CaO_2, arterial O_2 content.

Table 2. *Regional blood flow with increased IAP (mean ± SE expressed as ml·min^{-1}·100 g^{-1})*

	Control	15 mm Hg	20 mm Hg	25 mm Hg	Recovery
Stomach	63 ± 5	46 ± 5*	37 ± 4*	29 ± 2*	67 ± 7
Duodenum	229 ± 22	160 ± 22*	150 ± 20*	133 ± 14*	321 ± 29*
Jejunum and ileum	139 ± 10	98 ± 9*	87 ± 10*	73 ± 8*	212 ± 16*
Large bowel	66 ± 7	53 ± 6*	43 ± 3*	35 ± 3*	82 ± 11*
Pancreas	61 ± 6	54 ± 12	40 ± 6*	25 ± 3*	59 ± 9
Omentum	56 ± 13	48 ± 8	30 ± 12	19 ± 4*	52 ± 12
Mesentery	50 ± 7	29 ± 5*	22 ± 5*	19 ± 4*	44 ± 6
Spleen	248 ± 39	161 ± 31*	104 ± 24*	50 ± 16*	77 ± 22*
Total portal vein†	252 ± 19	182 ± 19*	154 ± 18*	122 ± 12*	330 ± 26*
Hepatic artery†	20 ± 5	33 ± 9*	33 ± 6*	33 ± 6*	25 ± 5
Total liver flow†	272 ± 23	216 ± 23*	187 ± 22*	155 ± 16*	354 ± 26*
Kidney	264 ± 20	242 ± 17	212 ± 17*	155 ± 22*	251 ± 23
Adrenal	263 ± 35	293 ± 64	240 ± 29	212 ± 28	370 ± 31*
Diaphragm	14 ± 2	10 ± 1	9 ± 1*	7 ± 1*	12 ± 2
Cerebellum	76 ± 39	67 ± 14	71 ± 24	81 ± 21	58 ± 11
Brainstem	61 ± 14	54 ± 8	52 ± 15	61 ± 18	45 ± 9
Cerebral hemispheres	47 ± 11	42 ± 6	41 ± 12	49 ± 15	35 ± 7
Total brain	52 ± 12	46 ± 7	45 ± 13	53 ± 15	38 ± 7

* $p < 0.05$ from control.

† Flow per 100 g of liver weight.

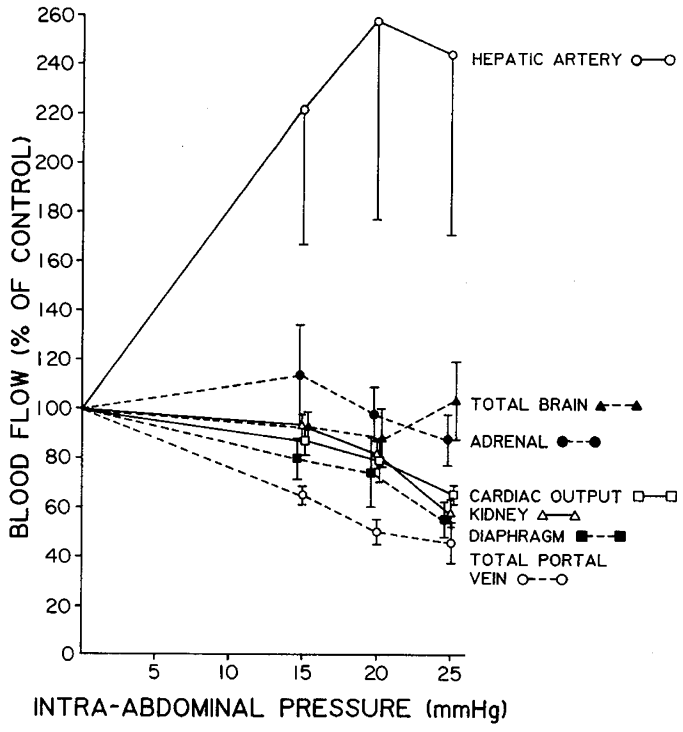

Fig. 4. Regional blood flow response, expressed as percent of control, to increased IAP. Values are means ± SE (*bars*).

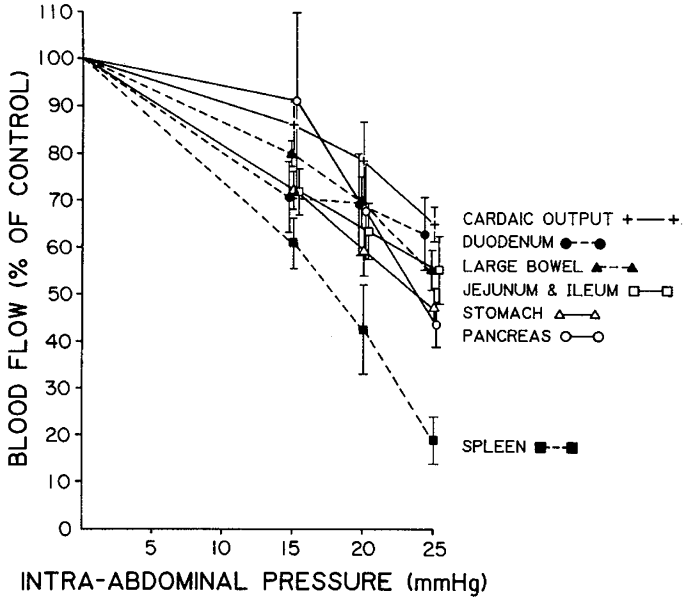

Fig. 5. Regional splanchnic blood flow response, expressed as a percent of control, to increased IAP. Values are means ± SE (*bars*).

tion of splanchnic blood flow than that reflected by the percent change in cardiac output, 4) a substantial decrease in splanchnic blood flow with as little as a 15 mm Hg increase in IAP, and 5) an increase in hepatic artery blood flow which did not totally compensate for the decreased portal vein flow for maintaining total liver blood flow, and which appeared to be maximal by only 15 to 20 mm Hg IAP.

Regional blood flow. Wesley *et al.* (3) have suggested IAP be kept below 15 to 20 mm Hg during surgical repair of gastroschisis and omphalocele. At an IAP of 15 mm Hg in our model, regional gastrointestinal (GI) blood flow was reduced 20–40%. The question of whether a reduction of this magnitude causes ischemic bowel problems was not addressed in this model. Studies of neonatal lambs indicate that O_2 extraction across the GI tract is approximately 25 to 30% in the fasted state (18) and is somewhat greater during digestion (19). Although these values indicate some reserve for increasing O_2 extraction, the observed decreases in GI blood flow at even 15 mm Hg IAP are probably close to compromising GI O_2 uptake.

Increased GI O_2 extraction together with decreased portal vein blood flow will have even greater consequence on liver O_2 availability. The neonatal liver extracts a greater percent of delivered oxygen than the intestines (18). The relatively low hepatic arterial contribution to total liver blood flow (less than 10% in this study) in neonatal lambs compared to adults (20, 21), and the relatively high O_2 saturation of portal vein blood in the neonate (18, 19) indicates the liver O_2 extraction relies heavily on portal vein O_2 delivery. Thus, the decreased portal vein blood flow, which is presumably associated with decreased O_2 content from increased GI O_2 extraction, should quickly exhaust the O_2 extraction reserve of the liver when IAP is elevated. Hepatic arterial blood flow more than doubled, as seen with other stresses that reduce portal vein inflow (21). However, due to the small contribution of hepatic arterial blood flow to total liver blood flow in the lamb, this hepatic autoregulation is insufficient to maintain total liver O_2 delivery even if GI O_2 extraction were not to increase. Therefore, our data suggest than the liver may be at even greater risk of depressed O_2 uptake than the intestines. This would further suggest that elevations of IAP to as little as 15 mm Hg for prolonged periods should probably be avoided, particularly because the compensatory increase in hepatic arterial flow is nearly maximal at this IAP.

We also observed substantial decreases in renal blood flow, although the percent drop was not as great as that in the GI tract. A preliminary study using nitrogen gas to distend the abdomen in adult dogs reported similar percent changes in renal and GI blood flow (although hepatic arterial flow fell in contrast to the lamb) (13). Thus, the major findings of this study are probably not specific to the lamb, or in general, to the method of raising IAP. Decreases in renal blood flow and function have been reported using inflatable intraperitoneal bags in adult dogs (12). Others have reported similar decreases in cardiac output using an inflatable bag in piglets (4) and using intraperitoneal gas infusion in puppies (3) and adult dogs (22). We used an inflatable bag rather than gas instillation to stimulate the traction placed on the ventral surface of the GI tract during surgical repair of gastroschisis. Nevertheless, our results probably have bearing on a variety of situations arising in pediatric and adult populations, such as gaseous abdominal distension, swelling from liver transplantation (13), ascites, portal hypertension (23), peritoneal dialysis, and gas instillation for laparoscopy (22, 24). However, the magnitude of the decrease in splanchnic and renal blood flows may be greater in the neonate than the adult because a given increase in IAP represents a larger percent decrease in perfusion pressure at the normally lower neonatal arterial pressure. Moreover, the low IAP threshold for reducing splanchnic blood flow raises questions whether treatment of the more common neonatal problem of gaseous abdominal distension should not be more aggressive.

Several mechanisms may contribute to the decrease in splanchnic and renal blood flow with elevated IAP. First, high levels of IAP probably collapse large veins. While physiological rises in IAP may assist venous return transiently (25), levels of IAP of 15 mm Hg and greater would be expected to collapse large veins as they empty and to possibly cause engorgement upstream from the site of compression. Our observation that the rise in intraabdominal vena cava pressure was less than the rise of IAP is consistent with venous collapse at the site of measurement and along the venous system further upstream. In this situation IAP acts as the effective downstream pressure for perfusion. There is some evidence from pressure-flow curves in the liver that the

effective downstream pressure is normally higher than hepatic vein pressure (26). Second, elevated IAP may increase arterial resistance by direct arterial compression. Compensation by metabolic and myogenic autoregulation may be incomplete in the neonate (27). The observed increase in calculated systemic vascular resistance, which utilized central venous pressure as the downstream pressure, does not distinguish between arteriolar constriction and elevated downstream pressure in the abdominal compartment. Third, active reflex vasoconstriction may occur in response to decreased arterial pressure and venous return. This possibility is supported by the observation that in certain splanchnic organs, particularly spleen, the percent decrease in blood flow was greater than the percent decrease in perfusion pressure (assuming IAP as a downstream pressure). Splenic vasoconstriction is prominent in the lamb during the stress of arterial hypoxia (16). In addition, portal vein occlusion elicits a greater renal vasoconstriction than inferior vena caval occlusion at similar reductions in arterial pressure, suggesting the presence of a GI to renal vascular reflex (23). Fourth, humoral-induced vasoconstriction may occur. Decreased venous return from portal vein or inferior vena caval occlusion is thought to stimulate vasopressin secretion (23). Moreover, the maintained blood flow in the adrenal gland with elevated IAP suggests active vasodilation which may be associated with hormonal secretion. Adrenal vasodilation is known to occur with other stresses such as hypoxia in the lamb (16).

The 36% decrease in diaphragmatic blood flow in the time control group makes the 45% decrease at an IAP of 25 mm Hg difficult to interpret. However, the return of blood flow to baseline levels on deflating the bag suggests that elevated IAP does impair blood flow to the diaphragm to some extent. Others have recently reported impaired diaphragmatic blood flow during contraction when IAP was elevated by binding the abdomen (28).

We elected to increase IAP in a stepwise fashion rather than in a random sequence for evaluating any threshold in the IAP response. We believed that the potential order effect on a moderate elevation of IAP following a more severe elevation could evoke a large secondary effect of decreasing IAP rather than the primary effect of increasing IAP. The large hyperemia found throughout the intestines 30 min after decreasing IAP to zero indicates the presence of a significant and prolonged compensatory phase. On the other hand, it should be recognized that the 30-min step durations in the current design may have allowed fluid shifts, humoral responses, and other adaptations to come into play which may have modified the response seen if IAP were suddenly elevated from 0 to 25 mm Hg. Nevertheless, the observation that blood flow to most abdominal organs decreased monotonically as a function of IAP suggests that any compensatory mechanisms were not completely overriding the primary response.

Central hemodynamics. The decrease in cardiac output with elevated IAP is presumably the result of decreased venous return from abdominal organs. The question of why central venous pressure increased more than esophageal pressure then arises. A significant increase in right ventricular afterload is not a likely explanation because there was no significant change in pulmonary artery pressure. A more likely explanation is that the increase in esophageal pressure does not totally reflect the increase in pressure surrounding the heart. Several studies have found that the change in pressure surrounding the heart may be greater than the change in esophageal pressure during lung inflation with positive end-expiratory pressure (29–31). In addition, Robotham *et al.* (32) demonstrated in open-chest dogs on right heart bypass that approximately 20% of the increase in IAP produced by external abdominal compression is reflected in an elevated left atrial pressure. This pressure transmission was dependent on cephalad movement of the diaphragm and not on changes in left ventricular venous return or afterload. Therefore, esophageal balloon pressure may not be a valid measure of pericardial pressure when IAP is raised.

Similarly for assessing left ventricular function in our experiments, the equivalent rises in pulmonary wedge pressure and esophageal pressure in the face of decreased aortic pressure does not necessarily imply that cardiac filling is unchanged and contractility is depressed. Rather, esophageal pressure is likely underestimating pericardial pressure and left ventricular filling is likely diminished. We observed a decrease in both stroke volume and heart rate with a decline in aortic pressure. Because unloading the high pressure baroreceptors should elicit tachycardia, the observed bradycardia may result from unloading cardiac low pressure baroreceptors. Thus, the decrease in stroke volume and heart rate are both consistent with the hypothesis that cardiac filling is decreased.

Wesley *et al.* (3) have advocated use of intragastric pressure measured through a gastrostomy tube as a guide for reducing and closing a Silastic chimney in the repair of omphalocele and gastroschisis. Although we found that intragastric pressure did not track IAP, these results are probably unique to the ruminant with its four-chambered stomach and not relevant to the human neonate. It should be appreciated, however, that the abdomen does not necessarily behave mechanically as a fluid-filled container. A recent study employed a variety of maneuvers to increase IAP and demonstrated differences in intragastric, intraintestinal, and subdiaphragmatic pressures, unless the abdomen was first filled with a large volume of fluid (33). Therefore, the concept of a single value of IAP may not always be valid.

In summary, our results demonstrate reduced renal blood flow and profound reductions of regional splanchnic blood flow, even with moderate elevation of IAP in neonatal lambs. The data suggest that care should be exercised in minimizing elevations of IAP at or above 15 mm Hg for prolonged periods in the repair of gastroschisis and omphalocele. Although blood volume expansion may help maintain cardiac output, it may not necessarily restore renal blood flow (12, 22). Whether fluid infusion can be used to restore splanchnic blood flow under conditions of elevated IAP is not clear at the present time (34, 35).

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