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Changes in splanchnic blood flow and cardiovascular effects following peritoneal insufflation of carbon dioxide

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Summary. Laparoscopic surgery has rapidly become a popular and widely used technique. Although this procedure has been shown to be generally safe, cardiovascular derangement related to carbon dioxide pneumoperitoneum has been reported. There are few data available on the relationship between systemic and regional hemodynamics in cases of pneumoperitoneum. Changes in splanchnic blood flow and cardiovascular effects following a moderate increase of intraabdominal pressure (IAP) to 16 mmHg during a 3-h period were analyzed in six anesthetized dogs. After insufflation, cardiac output and blood flow in the superior mesenteric artery and portal vein decreased progressively and returned to the preinsufflation values following deflation. Hepatic arterial blood flow did not change significantly, perhaps due to compensatory mechanisms for maintenance of hepatic blood flow. Mechanical compression of the splanchnic capillary beds due to the elevated IAP may possibly reflect the increase in systemic vascular resistance causing the decrease in cardiac output. To prevent this impairment, intermittent decompression of gas during surgical laparoscopy is recommended.

Key words: Laparoscopic surgery – Intraabdominal pressure – Pneumoperitoneum – Carbon dioxide – Cardiovascular response – Splanchnic blood flow

Recently, operative laparoscopy has become widely used for general surgery including laparoscopic cholecystectomy [7, 16]. Although the respiratory and cardiovascular derangements produced by carbon dioxide (CO_2) pneumoperitoneum are well described [10, 11, 19], less attention has been focused on the change in visceral blood flow, other than that of the kidney, in this condition. Moreover, there are no detailed data on the correlations between systemic and regional hemodynamics. This study was therefore designed to ascertain changes in splanchnic blood flow due to the elevation of intraabdominal pressure (IAP) accompanying CO_2 pneumoperitoneum using a transit-time ultrasonic blood flow meter [6].

Materials and methods

Six adult mongrel dogs, weighing 11.5–15 kg, were fixed in a supine position under anesthesia induced by intravenous injection of 30 mg/kg sodium pentobarbital. The animals were then intubated with a cuffed endotracheal tube, paralyzed with 0.1 mg/kg pancuronium bromide, and ventilated with a mechanical ventilator (Bird Mark 7, Bird Products Co., U.S.A.). The ventilation was adjusted to maintain the arterial blood gases and pH within normal limits. Additional sodium pentobarbital and pancuronium bromide were given throughout the experiment whenever spontaneous respiratory movement occurred.

Experimental preparation and measured parameters

1. Mean arterial blood pressure (MAP): A polyethylene catheter was inserted into the femoral artery and connected to a pressure transducer (78342A Monitor, Hewlett-Packard Inc., U.S.A.) to obtain MAP.
2. Cardiac output (CO): A 5-French Swan-Ganz catheter was advanced into the pulmonary artery via an external jugular vein. CO was measured by the thermodilution technique using the average of triplicate determinations after injection of 2 ml of iced saline (REF-1, Edwards Inc., Critical Care Division, U.S.A.).
3. Central venous pressure (CVP): CVP was recorded from the central venous pressure port of the Swan-Ganz catheter.
4. Inferior vena caval pressure (IVCP): The inferior vena cava was cannulated from a femoral vein for measurement of IVCP.
5. Systemic vascular reistance (SVR): SVR was calculated using central venous pressure as the downstream pressure.
6. Portal venous pressure (PVP): Measurement of PVP was accomplished by isolating a small venous branch from the mesentery and inserting a catheter into the portal vein.
7. Hepatic arterial blood flow (HAF), superior mesenteric arterial blood flow (SMAF), portal venous blood flow (PVF), total hepatic blood flow (THF): The common hepatic artery, superior mesen-

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teric artery, and portal vein were exposed. The gastroduodenal artery was ligated at its origin from the common hepatic artery. HAF, SMAF, and PVF were obtained with a transit-time ultrasonic blood-flow meter (Transonic T201 ultrasonic blood flow meter, Transonic System Inc., U.S.A.) using probes of appropriate size. THF was calculated as the sum of PVF and HAF.

Experimental protocol

The abdominal wall was closed in two layers. Purse-string sutures were tied securely around each catheter and line to prevent loss of air and subsequent loss of pressure. A Veress needle was inserted into the peritoneal cavity. This needle was attached to a CO_2 inlet (PNE-N, Olympus Optical Inc., Japan), which could be adjusted to increase IAP and then to maintain the pressure at a constant 16 mmHg that was confirmed occasionally by a coeliotometer of the CO_2 inlet for an indefinite period. Each animal was observed for a 30-min period, during which control readings were obtained. All hemodynamic parameters were measured at 30 min and 1, 2, and 3 h after peritoneal insufflation of CO_2, and at 30 min after decompression, and were compared with the preinsufflation values. Statistical analysis of all data was performed using nonparametric ranking for paired data (Wilcoxon's sign-rank test).

Results

There were no significant changes in MAP and HR during the 3-h insufflation period. CO was decreased significantly at 30 min after the introduction of CO_2 gas and then decreased progressively and significantly during the 3 h of insufflation. SVR was elevated 30 min after the start of insufflation and thereafter increased gradually during the procedure. Following the deflation of CO_2 gas, CO and SVR returned to the preinsufflation values (Fig. 1).

Figure 2 shows the effect of CO_2 pneumoperitoneum on intravascular pressure, CVP, IVCP, and PVP. CVP did not increase significantly throughout the procedure. IVCP and PVP increase significantly in parallel with the elevation of IAP and were almost constant throughout the procedure. Deflation caused an immediate fall in IVCP and PVP.

The results of splanchnic blood-flow measurements are summarized in Fig. 3. SMAF and PVF were decreased even at 30 min after the start of insufflation, and these declines persisted during the course of pneumoperitoneum. HAF had a tendency to decrease, but not to a significant extent. THF also declined after insufflation. Although this change was not significant until 30 min after the start of insufflation, it became significant after 1 h. After deflation, it reverted to the preinsufflation value.

Discussion

The findings of the present study confirmed that moderate IAP of 16 mmHg produced by CO_2 insufflation for a 3-h period resulted in a significant rise of intraabdominal venous pressure and impairment of blood flow in the superior mesenteric artery and portal vein. Caldwell et al. have also reported that an IAP of more than 20 mmHg created by an inflatable bag caused a decrease of tissue blood flow in all organs measured except for

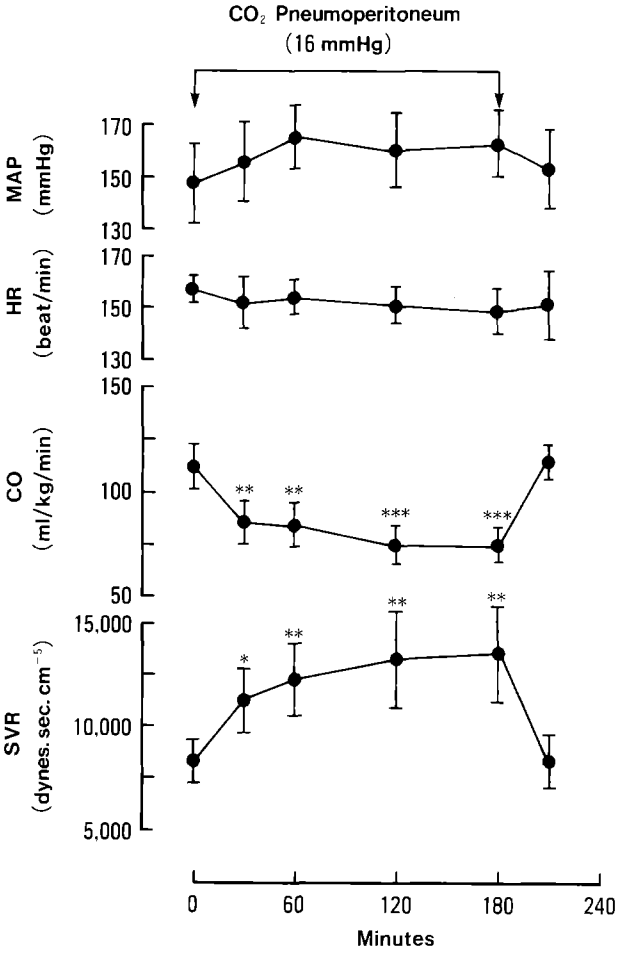

Fig. 1. Responses of mean arterial blood pressure (MAP), heart rate (HR), cardiac output (CO), and systemic vascular resistance (SVR) following CO_2 pneumoperitoneum (n = 6) Data are means ± SD. $^{***}P < 0.005$. $^{**}P < 0.01$. $^{*}P < 0.05$

the adrenal glands as determined by using radioactive microspheres [3].

Several mechanisms may contribute to the decrease in splanchnic blood flow due to elevated IAP caused by CO_2 insufflation. First, an IAP of 16 mmHg would be expected to collapse the splanchnic veins. The direct mechanical compression caused by CO_2 insufflation easily decreases the diameter of these blood vessels. Poiseuille's law states that blood flow in a vessel is proportional to the fourth power of its radius. Thus, a small decrease in vessel radius leads to a significant decrease in blood flow and a large increase in vessel resistance. These changes would possibly cause engorgement upstream from the site of compression. If it is assumed that the pressure-gradient-driving flow is given by the difference between systemic arterial pressure and IAP, then vascular resistance in each bed would rise as IAP increased.

Second, humoral-induced vasoconstriction may occur. Increased IAP and subsequent peritoneal distension have a direct stimulating effect on the release of vasopressin, which is a well-known constrictor of re-

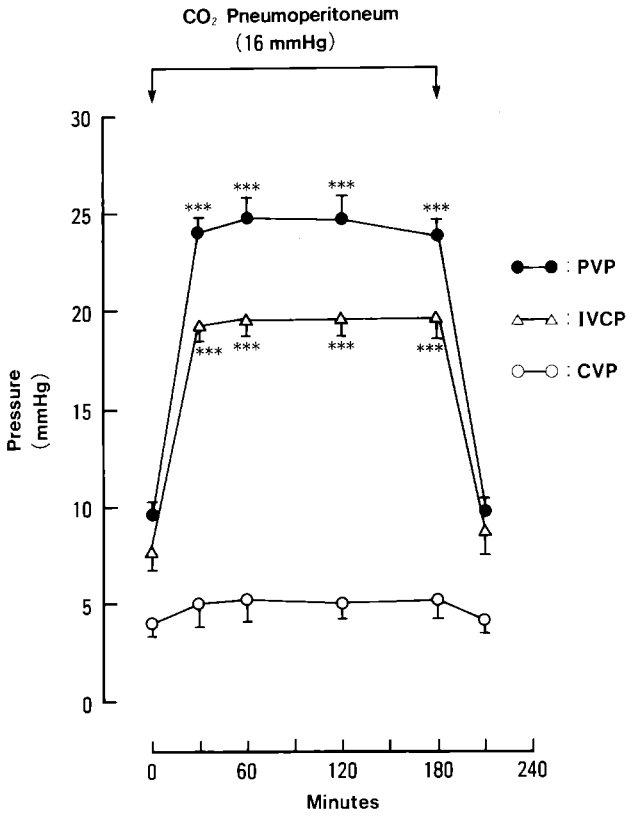

Fig. 2. Responses of central venous pressure (CVP), inferior vena caval pressure (IVCP), and portal venous pressure (PVP) following CO_2 pneumoperitoneum (n = 6) Data are means ± SD. ***$P < 0.005$

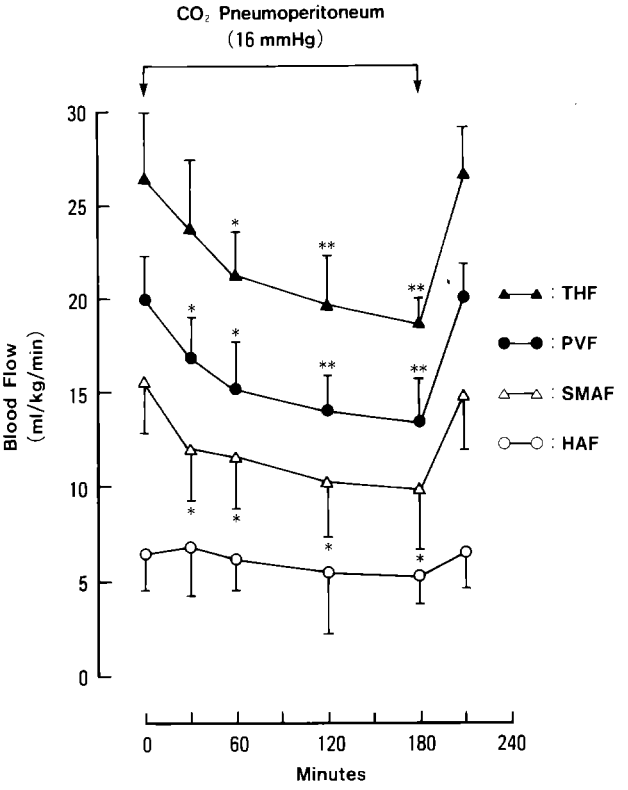

Fig. 3. Responses of hepatic arterial blood flow (HAF), superior mesenteric arterial blood flow (SMAF), portal venous blood flow (PVF), and total hepatic blood flow (THF) following CO_2 pneumoperitoneum (n = 6) Data are means ± SD. **$P < 0.01$. *$P < 0.05$

nal, superior mesenteric, and celiac vasculature [15]. Decreased venous return from the occluded portal vein or inferior vena cava is also thought to stimulate vasopressin secretion [1].

Third, the splanchnic vessels are known to possess a well-developed myogenic mechanism for local control of vascular tone [12]. Thus compression of the venous outflow tract could elevate intravascular pressure and thereby trigger intrinsic myogenically mediated vasoconstriction.

Finally, CO_2 itself may influence blood circulation in the liver. Although in this study time course of P_{CO2} was not estimated, it is well known that CO_2 pneumoperitoneum induces a significant increase in P_{CO2} which is transferred across the peritoneum as well as a significant decrease in pH [11, 14, 19]. Hepatic blood flow is reduced by hypercapnia produced by breathing CO_2 at concentrations of less than 5%, which increases portal venous pressure and causes mesenteric vasoconstriction [8].

In the present study, hepatic arterial blood flow remained unchanged, and the decline in total hepatic blood flow was caused mainly by the decrease in portal venous blood flow. A reciprocal relationship between blood flow to the liver through the hepatic artery and portal vein has been suggested. Previous studies of hepatic arterial blood flow in experimental animals have confirmed that reduction or elimination of the portal venous flow increases the hepatic arterial flow [4, 9]. The fact that the reduction of hepatic arterial flow was insignificant throughout the present procedure may be explained by this compensatory mechanism.

The effects of changes in IAP on cardiac output have usually been evaluated with respect to the net effects of changes in systemic venous return and systemic vascular resistance [13]. In our hemodynamic observations, while central venous pressure did not change significantly, systemic vascular resistance increased progressively. Under the condition of elevated IAP, central venous pressure may not reflect the venous return precisely because of an increase in pleural pressure caused by elevation of the diaphragma [11]. The mechanism of the increases in systemic vascular resistance was probably mechanical compression of the splanchnic capillary beds and the subsequent rise in splanchnic vascular resistance caused by the elevated intraabdominal pressure.

In our study, the reduction of each blood flow was gradual, and the greatest change was recognized 3 h after the start of insufflation. This may be related to the decrease in intravascular volume produced by the rise in capillary pressure and subsequent transcapillary fluid filtration into the interstitial space. Barnes et al. examined the cardiovascular response to elevated IAP using a dog model in which Tyrode solution was infused

into the peritoneal cavity and found that the lower body was grossly engorged due to edema at the end of the experiment [2].

The mortality rate associated with laparoscopic cholecystectomy is generally agreed to be very low [18]. From our results, however, it is clear that undue strain is exerted on the intestinal and liver blood flow by continuous moderate elevation of IAP to even 16 mmHg, which is usually applied during laparoscopic surgery. Expansion of the blood volume or treatment with a vasoconstrictor may easily correct the deficit in cardiac output [5, 17]. Cullen et al. found that in the patients with high IAP, the infusion on either lactated Ringer's or colloid solutions (5% albumin or hetastarch) at a rate of 10 ml/kg over 10–15 min increased central venous pressure, cardiac output, and stroke volume significantly [5]. Also, intermittent deflation of CO_2 during laparoscopic surgery is recommendable.

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