FULL PAPER *Surgery*

Influence of pneumoperitoneum and postural change on the cardiovascular and respiratory systems in dogs

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ABSTRACT. We investigated the influence of pneumoperitoneum#(PP) and postural change under inhalation anesthesia with isoflurane, which is routinely used in dogs, on the cardiovascular and respiratory systems. As test animals, 6 adult beagles were used. To induce anesthesia, atropine, butorphanol and propofol were intravenously injected. Anesthesia was maintained with 1.3 MAC (1.7%) isoflurane. The following were the experiment conditions: I:E ratio, 1:1.9; tidal air exchange, 20 m*l*/kg; and ventilation frequency, 14 times/min. Respiration was regulated so that the $PaCO_2$ was approximately 35 to 40 mmHg before the start of the experiment. PP with CO_2 (intraperitoneal pressure 15 mmHg) and a postural change (15°C) was performed during the experiment. As parameters of circulatory kinetics, heart rate (HR), mean aortic pressure (MAP), mean pulmonary arterial pressure (MPAP), central venous pressure (CVP), femoral venous pressure (FVP) and cardiac output (CO) were measured. As parameters of respiratory kinetics, airway pressure (PAW) and blood gas (BG) were measured. There were significant increases in HR, MAP, MPAP, CVP, FVP, CO, PAW and $PaCO_2$ after PP in the horizontal position. There were significant increases in CVP, FVP, PAW and $PaCO_2$ after PP in the Trendelenburg position. There were significant increases in the MPAP, CVP, FVP, PAW and $PaCO_2$ after PP in the inverse Trendelenburg position. There was a significant difference in FVP after PP between the Trendelenburg position and inverse Trendelenburg position. The results of this experiment suggest that appropriate anesthesia control, such as changing the ventilation conditions after PP, is required for laparoscopic surgery under inhalation anesthesia with isoflurane.

KEY WORDS: cardiovascular system, laparoscopic surgery, pneumoperitoneum, postural change, respiratory system

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For laparoscopic surgery, pneumoperitoneum and postural changes are performed to maintain the visual field. Pneumoperitoneum increases the airway pressure and $PaCO_2$ and decreases pulmonary compliance and PaO_2, influencing the respiratory system [1]. In addition, it increases the heart rate, aortic pressure, pulmonary arterial pressure and vascular resistance and decreases venous return and cardiac output, affecting the cardiovascular system [8]. For laparoscopic surgery, the posture is changed, considering the position of the target organ and the necessary manipulations. This inhibits the cardiovascular and respiratory functions in comparison with those in a horizontal position [4]. However, these studies reported results under anesthesia with pentobarbital sodium and a marked increase in the pneumoperitoneum pressure; the effects of pneumoperitoneum and postural change under inhalation anesthesia with isoflurane (intraperitoneal pressure, 8 to 15 mmHg; postural tilting angle, 15°C or less), which is commonly employed in clinical practice, on the cardiovascular and respiratory systems remain to be clarified. In this study, we investigated the influence of these manipulations under inhalation anesthesia with isoflurane, which is routinely used in dogs, on the cardiovascular and respiratory systems.

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MATERIALS AND METHODS

As test animals, 6 adult beagles (7 to 10 kg, average 8.6 kg, and age 3 to 4 years old) were used. For preanesthetic medications, atropine sulfate at 0.025 mg/kg (Tanabe Seiyaku Co., Ltd., Osaka, Japan) and butorphanol tartrate at 0.1 mg/kg (Vetorphale, Meiji Seika Pharma Co., Ltd., Tokyo, Japan) were intravenously injected. To induce anesthesia, 6 mg/kg of propofol (Rapinovet, Takeda Schering-Plough Animal Health, Tokyo, Japan) was intravenously injected. After intratracheal intubation, 0.1 mg/kg of pancuronium bromide (Mioblock, Sankyo Co., Ltd., Tokyo, Japan) was additionally administered if necessary. Furthermore, anesthesia was maintained with 1.3 MAC (1.7%) isoflurane. Controlled respiration was conducted using a ventilator (KV-1a, Kimura Medical Instrument Co., Ltd., Tokyo, Japan) under the following ventilation conditions: I:E ratio, 1:1.9; tidal air exchange, 20 m*l*/kg; and ventilation frequency, 14 times/min. Respiration was regulated so that the $PaCO_2$ was approximately 35 to 40 mmHg before the start of the experiment. Lactated Ringer's solution was transfused at 5 m*l*/kg/hr. To measure the aortic pressure, blood gas and central venous pressure, 5 F vascular catheters were inserted into the common carotid artery and posterior vena cava, respectively. A 5 F Swan-Ganz catheter was inserted into the pulmonary artery to determine the pulmonary arterial pressure and cardiac output. A 22 G indwelling needle was inserted into the femoral vein for femoral venous pressure measurement. A minor incision was made around the median umbilical region. After puncture with a pneumoperitoneum needle, pneumoperitoneum with carbon dioxide was performed. A

trocar (Core) measuring 5 mm in diameter was inserted into the abdominal cavity. Subsequently, carbon dioxide was deaerated, and the test animals were acclimated for about 1 hr.

Circulatory parameters: As parameters of circulatory kinetics, heart rate, aortic pressure, pulmonary arterial pressure, central venous pressure, femoral venous pressure and cardiac output were measured. Heart rate was determined based on waveforms obtained on a monitor (Bio-scope AM120, Fukuda M-E Kogyo, Tokyo, Japan). To measure the aortic, pulmonary arterial and central venous pressures, an indwelling catheter was connected to a transducer (TP400T, Nihon Kohden Corp., Tokyo, Japan), and a bedside monitor (Life Scope 8, Nihon Kohden Corp.) was employed. Cardiac output was measured using a thermodilution cardiac output meter (MTC-6100, Nihon Kohden Corp.).

Respiratory parameters: As parameters of respiratory kinetics, airway pressure and gas parameters were measured. For airway pressure measurement, an airway pressure meter was used. For blood gas analysis, pH, $PaCO_2$, HCO_3^- and BE were measured using an automatic blood gas analyzer (M278, Chiba Corning, Tokyo, Japan).

Pneumoperitoneum and postural change: The test dogs were horizontally fixed in a supine position. After pre-pneumoperitoneum values were measured in the horizontal position, pneumoperitoneum with carbon dioxide (intraperitoneal pressure: 15 mmHg) was performed in the test dogs for 20 min. After 20 min, post-pneumoperitoneum values were determined. After deaeration, the posture was changed to the Trendelenburg position (low-head position) with 15-degree tilting, and the test dogs were acclimated for 10 min. Subsequently, pre-pneumoperitoneum values were measured, and pneumoperitoneum was carried out for 20 min, as described for the horizontal position. After 20 min, post-pneumoperitoneum values were determined, and deaeration was conducted. The posture was changed to the inverse Trendelenburg position (high-head position) with 15-degree tilting, and the animals were acclimated for 10 min. Subsequently, similar measurements were performed.

Statistical analysis: The results are expressed as the mean ± standard deviation. For statistical analysis, the paired *t*-test and one-way analysis of variance# (ANOVA) followed by a post hoc Bonferroni's multiple comparison test were employed to compare values before and 20 min after pneumoperitoneum in each posture. $P<0.05$ was regarded as significant.

RESULTS

Circulatory parameters: The results for the circulatory parameters during the experiment are shown in Table 1. There was a significant increase in heart rate after pneumoperitoneum in the horizontal position ($P<0.01$). In other postures, there were no significant post-pneumoperitoneum changes. The mean aortic pressure significantly increased after pneumoperitoneum in the horizontal position ($P<0.01$). In other postures, there were slight increases after pneumoperitoneum. The mean pulmonary arterial pressure significantly increased after pneumoperitoneum in the horizontal and inverse Trendelenburg positions ($P<0.01$ and $P<0.05$, respectively). In the Trendelenburg position, there was a slight increase after pneumoperitoneum. Central venous pressure significantly increased after pneumoperitoneum regardless of the posture ($P<0.05$). There was also a significant increase in femoral venous pressure regardless of the posture ($P<0.01$). Furthermore, there was a significant difference after pneumoperitoneum between the Trendelenburg and inverse Trendelenburg positions ($P<0.01$). Mean cardiac output significantly increased after pneumoperitoneum in the horizontal and inverse Trendelenburg positions ($P<0.05$ and $P<0.01$, respectively). There were no significant changes in the total or pulmonary vascular resistance coefficients regardless of the posture.

Respiratory parameters: The results for the respiratory parameters during the experiment are shown in Table 2. The airway pressure significantly increased after pneumoperitoneum regardless of the posture ($P<0.01$). In the Trendelenburg position, post-pneumoperitoneum changes were more marked than in the horizontal position. In the inverse Trendelenburg position, they were less marked. There was a significant increase in $PaCO_2$ after pneumoperitoneum regardless of the posture ($P<0.01$).

DISCUSSION

In this experiment, there was a significant increase in airway pressure after pneumoperitoneum irrespective of the posture. This change was possibly associated with pneumoperitoneum/postural change-related physical factors related to the abdominal pressure. Carbon dioxide is used as the pneumoperitoneum gas, because it is nonflammable and unlikely to cause gas embolism. An increase in $PaCO_2$ is observed after pneumoperitoneum [6]. This change may be primarily related to carbon dioxide absorbed through the peritoneum, that is, the area of the peritoneum exposed to carbon dioxide [3]. Carbon dioxide absorption-related excitation of the sympathetic nervous system and physical compression of the vascular system related to a rise in the intraperitoneal pressure have been reported as influences of the pneumoperitoneum operation on the cardiovascular system [5]. In this experiment, there was a significant increase in heart rate after pneumoperitoneum in the horizontal position. This may have appeared as a sympathetic nerve-mediated change related to abdominal wall extension associated with a rise in intraperitoneal pressure and an increase in blood carbon dioxide level. However, there was no significant increase after pneumoperitoneum in the Trendelenburg or inverse Trendelenburg positions. This suggests the influence of carbon dioxide and postural change *in vivo*, considering that the blood carbon dioxide level remains high for a specific period after deaeration and that post-pneumoperitoneum changes in heart rate depend on changes in posture [9]. The arterial and pulmonary arterial pressures rose after pneumoperitoneum. A previous study reported that, when the tilting angle was 30°C, the mean arterial pressure increased in both the Trendelenburg and inverse Trendelenburg [7]. A rise in pulmonary arterial pressure may be intricately associated with

Table 1. Fluctuation of circulatory parameters

Position	Horizontal		Trendelenburg		Inverse Trendelenburg	
	Before	After	Before	After	Before	After
HR (bpm)	125.40 ± 24.08	140.00 ± 19.91[a)]	139.80 ± 25.13	136.40 ± 20.74	136.60 ± 22.61	132.00 ± 14.96
CVP (mmHg)	1.20 ± 1.09	5.60 ± 3.20[a)]	2.00 ± 2.34	7.00 ± 5.70[a)]	-1.00 ± 2.23	4.40 ± 3.78[a)]
FVP (mmHg)	2.20 ± 1.92	15.00 ± 3.53[a)]	1.20 ± 1.64	10.60 ± 3.50[a)]	5.80 ± 3.34	18.00 ± 3.39[a,b)]
CO (*l*/min)	1.79 ± 0.84	2.33 ± 0.63[a)]	2.51 ± 1.01	2.22 ± 0.53	1.89 ± 0.48	2.43 ± 0.59[a)]
MAP (mmHg)	78.60 ± 11.71	103.60 ± 8.41[a)]	95.40 ± 7.98	102.73 ± 8.38	85.20 ± 15.70	96.93 ± 9.53
MPAP (mmHg)	9.06 ± 1.03	13.00 ± 2.89[a)]	12.13 ± 4.22	13.59 ± 2.47	6.26 ± 1.49	11.73 ± 4.24[c)]
SVRI (dyn/sec/cm^{-5}/m^2)	1,527.31 ± 576.45	1,584.56 ± 503.74	1,433.66 ± 531.07	1,609.27 ± 367.94	1,561.19 ± 338.27	1,368.12 ± 284.91
PVRI (dyn/sec/cm^{-5}/m^2)	178.07 ± 77.58	196.46 ± 69.17	190.11 ± 123.10	213.26 ± 59.73	133.76 ± 59.72	164.68 ± 62.43

Data are shown as the mean ± SD. Before: before pneumoperitoneum, After: after pneumoperitoneum, HR: heart rate, CVP: central venous pressure, FVP: femoral venous pressure, CO: cardiac output, MAP: mean aortic pressure, MPAP: mean pulmonary artery pressure, SVRI: systemic vascular resistance index, PVRI: pulmonary vascular resistance index, a) Significant difference vs before pneumoperitoneum (P<0.01), † (P<0.05), b) Significant difference vs after pneumoperitoneum in Trendelenbulg's position (P<0.01).

Table 2. Fluctuation of respiratory parameters

Position	Horizontal		Trendelenburg		Inverse Trendelenburg	
	Before	After	Before	After	Before	After
PAW (mmHg)	12.4 ± 2.07	18.8 ± 4.38[a)]	12.4 ± 1.67	19.6 ± 4.33[a)]	13.2 ± 1.09	19.0 ± 4.00[a)]
$PaCO_2$ (mmHg)	36.46 ± 3.88	55.98 ± 2.37[a)]	44.28 ± 2.84	56.34 ± 3.75[a)]	46.8 ± 3.82	61.4 ± 5.92[a)]
pH	7.42 ± 0.04	7.27 ± 0.03[a)]	7.35 ± 0.02	7.25 ± 0.02[a)]	7.33 ± 0.02	7.23 ± 0.02[a)]
BE (mmol/*l*)	–1.18 ± 1.6	–1.72 ± 1.5	–1.74 ± 1.2	–2.64 ± 1.7	–1.84 ± 0.7	–2.5 ± 1.4
HCO_3^- (mmol/*l*)	23.05 ± 1.19	24.77 ± 1.43	23.8 ± 1.43	24.53 ± 1.4	23.88 ± 0.99	24.82 ± 1.53

Data are shown as the mean ± SD. Before: before pneumoperitoneum, After: after pneumoperitoneum PAW: airway pressure, $PaCO_2$: partial pressure of arterial carbon dioxide, pH: potential hydrogen, BE: base excess, HCO_3^-: bicarbonate ion, *a) Significant difference vs before pneumoperitoneum (P<0.01).

an increase in the intrathoracic pressure, changes in venous return and changes in heart function. In the Trendelenburg position, the pulmonary arterial pressure increased before pneumoperitoneum, and post-pneumoperitoneum changes were less marked than the inverse Trendelenburg position. This was possibly associated with postural change-related alterations in venous return and the pressure reflex [11]. Several studies indicated the influence of the pneumoperitoneum operation and postural change on cardiac output. The post-pneumoperitoneum values varied among these studies. Cardiac output depends on myocardial contractility and venous return as a preload. However, the depth of anesthesia, transfusion volume, sympathetic stimulation, vagal nerve stimulation, type of pneumoperitoneum gas and pneumoperitoneum pressure are involved in these factors. Smith and Motew *et al.* reported that cardiac output increased at an intraperitoneal pressure of 20 mmHg or less, whereas it decreased at 30 mmHg [10]. Concerning the mechanism of the dimorphous change, an increase in $PaCO_2$ may stimulate the sympathetic nervous system under a low abdominal pressure and increase venous return and cardiac output by shifting blood from the intraperitoneal organs to the posterior vena cava via the positive inotropic actions of the cardiac muscle and contraction of peripheral blood vessels. On the other hand, under a high abdominal pressure, venous return may decrease via compression of the posterior vena cava, reducing cardiac output. James *et al.* reported the safety of pneumoperitoneum with carbon dioxide with regard to the cardiovascular/respiratory systems under the following conditions: intraperitoneal pressure, 15 mmHg or less, and tilting angle in the Trendelenburg or inverse Trendelenburg positions, 15°C or smaller [2]. In the present experiment, the intraperitoneal pressure was established as 15 mmHg, and the tilting angle on postural change was 15°C; the inhibitory effects of excessive pneumoperitoneum/postural tilting on the cardiovascular/respiratory systems may be ruled out. Cardiac output increased after pneumoperitoneum in the horizontal and inverse Trendelenburg positions. This was possibly related to enhancement of venous return flowing in the heart through the outflow of blood pooled in the abdominal cavity with a pneumoperitoneum-associated increase in intraperitoneal pressure, as well as excitation of the sympathetic nervous system associated with pneumoperitoneum with carbon dioxide/abdominal wall extension. In the Trendelenburg position, the cardiac output prior to pneumoperitoneum was greater than in the horizontal position, and there were no marked changes after pneumoperitoneum. This was possibly associated with a shift in blood flow to the cephalic side under low-level head positioning and the pressure reflex. Carbon dioxide pneumoperitoneum performed with the anesthesia protocol employed in this experiment at an intraperitoneal pressure of 15 mmHg and postural tilting angle of 15°C may have enhanced venous return via an increase in the blood carbon dioxide level, abdominal wall extension-

related excitation of the sympathetic nervous system and a rise in the abdominal pressure, showing marked positive effects on the cardiovascular system rather than inhibitory effects on the circulatory system via a decrease in venous return, vagal nerve stimulation and a diaphragmatic elevation-related increase in the intrathoracic pressure. However, even at a postural tilting angle of 15°C, there was an increase in cardiac output in the pre-pneumoperitoneum phase in the Trendelenburg position, suggesting the influence of a shift in blood to the cephalic side in the Trendelenburg position. The central/femoral venous pressures are dependent on the presence of pneumoperitoneum and changes in posture. In this experiment, there was a significant increase in femoral venous pressure and a slight increase in central venous pressure after pneumoperitoneum, regardless of the posture. This may have resulted from an increase in intraperitoneal pressure, a rise in venous pressure associated with abdominal pressure elevation-related compression and a postural change-related shift of blood in the direction of gravity. It seems that intraperitoneal pressure caused enhancement of venous return through the outflow of blood pooled in the abdominal cavity and also caused blockage of venous return from the hind legs. The results of this experiment suggest that appropriate anesthesia control, such as increasing the ventilation frequency after pneumoperitoneum, is required for laparoscopic surgery under inhalation anesthesia with isoflurane, since the pneumoperitoneum operation and postural changes influence the cardiovascular and respiratory systems.

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