Cardiovascular and Respiratory Effects of Increased Intra-abdominal Pressure with and without Dexmedetomidine in Anesthetized Dogs

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Posted Date: 9 August 2023

doi: 10.20944/preprints202308.0654.v1

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Article

Cardiovascular and Respiratory Effects of Increased Intra-Abdominal Pressure with and without Dexmedetomidine in Anesthetized Dogs

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Simple Summary: Endoscopic procedures have been gaining popularity in veterinary medicine due to the advantages of minimally invasive. However, as laparoscopic procedures performed by inducing capnoperitoneum, several studies have been conducted to evaluate the safety of capnoperitoneum and reported that high intra-abdominal pressure might have deleterious cardiorespiratory effects. Furthermore, there are no studies monitoring parameters with administration of dexmedetomidine which also has significant cardiovascular effects, even though this drug frequently used as adjuvants. Therefore, our study aimed to evaluate the effects of intra-abdominal pressure and dexmedetomidine on cardiorespiratory effects. Five healthy beagle dogs were enrolled in the study and conducted with crossover design. Our study revealed that no significant cardiorespiratory effects were observed until intra-abdominal pressure of 20 mmHg, moreover also during administration of dexmedetomidine. These findings have shown that dexmedetomidine infusion administration may be applicable in laparoscopic procedures in healthy dogs.

Abstract: Intra-abdominal pressure (IAP) elevation during capnoperitoneum can cause adverse cardiovascular and respiratory effects. This study aimed to determine if a sequentially increased IAP affects cardiovascular and respiratory variables in anesthetized dogs and evaluate the effects of constant rate infusion of dexmedetomidine (Dex) on cardiovascular and respiratory variables with increased IAP. Five dogs were anesthetized, instrumented, and the Veress needle was equipped to adjust the IAP using a carbonic anhydrase insufflator. Stabilization was conducted for 1 h, and physiological variables were measured in IAPs of 0, 5, 10, 15, and 20 mmHg and after desufflation. After the washout period, the dogs underwent similar procedures along with constant rate infusion of dexmedetomidine. Cardiovascular effects of increased IAP up to 20 mmHg were not significant in healthy beagle dogs and those administered with dexmedetomidine. When comparing the control and dexmedetomidine groups, overall significant effects of dexmedetomidine were noted on heart rate, cardiac output, and systemic vascular resistance during the experiment. Respiratory effects were not affected by abdominal insufflation when compared between different IAPs and between the two groups. Overall, an increased IAP of until 20 mmHg did not significantly affect cardiovascular and respiratory variables in both control and dexmedetomidine groups. This study suggests that dexmedetomidine infusion administration is applicable in laparoscopic procedures in healthy dogs.

Keywords: dog; intra-abdominal pressure; dexmedetomidine; hypercapnia; PulseCO

1. Introduction

The abdominal cavity is a relatively compliant confined space, which allows a relatively limited capacity to increasing intracavitary volume or reducing extracavitary compliance before a relative increase in intra-abdominal pressure (IAP) occurs [1]. Increased IAP occurs in several clinical situations, including bandaging, obesity, intra-abdominal visceral content distension, and

 doi:10.20944/preprints202308.0654.v1

capnoperitoneum for laparoscopy [1]. Among these, as carbon dioxide insufflation for the laparoscopy approach is gaining popularity, clinical and experimental studies have shown that capnoperitoneum can cause adverse cardiovascular and respiratory effects [2,3]. To minimize these effects, maintaining the insufflation pressure below 12 mmHg in dogs has been recommended [4].

Dexmedetomidine (Dex), an alpha-2 adrenergic agonist, is used to provide reliable sedation, analgesia, and chemical restraint in dogs [5]. Moreover, it has properties to reduce the doses of induction and maintenance drugs and dose-dependently decrease the minimum alveolar concentration (MAC) of isoflurane in dogs [6,7]. Furthermore, these alpha-2 agonists generally have significant effects on cardiovascular function, including bradycardia, increased systemic vascular resistance (SVR), and decreased cardiac output (CO) [8]. In human medicine, dexmedetomidine is frequently used as an adjuvant to general anesthesia in the laparoscopy procedure [9-11]. However, in veterinary medicine, the evaluation of the hemodynamic effects of dexmedetomidine during abdominal insufflation has not been studied.

CO is frequently measured during anesthesia or in critical care medicine, and CO measurement allows early detection of hemodynamic instability [12]. In veterinary medicine, several techniques have been evaluated to measure CO; among those, the pulmonary arterial catheter thermodilution (PAC-TD) method is the gold standard of CO measurement [13]. However, owing to the serious complications of these methods, including arrhythmias, thrombosis, and infections, less invasive methods have been studied to avoid them [14]. The PulseCO method, which calculates CO by pulse contour analysis, is one of these alternative methods. This method has an advantage for patients as it provides the CO in real-time; therefore, clinicians can immediately observe the response to treatment [15]. In the present study, we monitored CO by the PulseCO method using a LiDCO™ hemodynamic monitor (LiDCO Unity; Masimo, CA, USA).

Recently, minimally invasive surgeries using endoscopic procedures have been gaining popularity in veterinary medicine [16]. To investigate the cardiovascular and respiratory effects of abdominal insufflation in anesthetized dogs, several studies have been performed; however, to the author's knowledge, there are no studies monitoring parameters with constant rate infusion of dexmedetomidine during abdominal insufflation. Therefore, this study aimed to determine if a sequentially increased IAP affects cardiovascular and respiratory variables in anesthetized dogs and evaluate the effects of constant rate infusion of dexmedetomidine on cardiovascular and respiratory variables with an increased IAP.

2. Materials and Methods

2.1. Animals

Five young adult beagles (three females and two males) were included in this study after approval by the Institutional Animal Care and Use Committee of Kyungpook National University (approval number: KNU2022-0348). They were 10–14 months old with a median body weight of 10 (range, 9.4–10.5) kg. Physical examinations, blood analysis, and radiography were performed before the study, and all dogs were judged to be healthy. The body condition score was 5 (range, 4–6) points on a 9-point scale. The study had a prospective crossover design with a washout period between treatments of 7 days.

2.2. Anesthesia and Instrumentation

The dogs were fasted for 8 h, and water was available until the dogs were transferred to the examination room to be anesthetized. Subsequently, a 22-gauge catheter was placed into one of the cephalic veins, and crystalloid fluids (Hartmann solution; JW Pharmaceutical, Korea) were started at 5 mL/kg/h. The dogs were premedicated with tramadol (Maritrol; Jeil Pharmaceutical Co., Ltd., Korea) 4 mg/kg (intramuscularly [IM]) and cefazoline (Cefozol; Hankook Korus Pharm, Korea) 20 mg/kg (intravenously [IV]). Preoxygenation with 100% oxygen at 5 L/min for 5 min was followed by anesthetic induction with alfaxalone (Alfaxan Multidose; Jurox Pty, Australia) 2 mg/kg (IV) over 1 min. After endotracheal intubation, each dog was positioned in dorsal recumbency, and anesthesia was maintained with isoflurane (Isoflurane; Hana Pharm, Korea) through a circle rebreathing system (9100c NXT; GE Healthcare, China).

Each dog was instrumented to monitor physiological variables. A multiparameter monitor (CARESCAPE Monitor B650; GE Healthcare, Finland) was used to measure the heart rate (HR, by

 doi:10.20944/preprints202308.0654.v1

lead II electrocardiogram), oxygen saturation (SpO2, by pulse oximetry), esophageal temperature and systolic, diastolic, and mean arterial blood pressures (SAP, DAP, and MAP, respectively). An over-the-needle 22-gauge catheter was placed in the dorsal pedal artery for arterial blood pressure measurement and blood collection for arterial blood gas analysis. Arterial blood samples were collected and analyzed using a blood gas analyzer (ABL80 FLEX; Radiometer, Denmark) for determining potential hydrogen (pH), the partial pressure of oxygen in arterial blood (PaO2), the partial pressure of carbon dioxide in arterial blood (PaCO2), bicarbonate (HCO3−), and base excess (BE). Dorsal pedal artery catheters were connected to the pressure transducer (TruWave; Edwards Lifesciences, Germany), which was placed at the level of the heart base and connected to a catheter using noncompliant tubing system continuously flushed with heparinized saline (0.9% NaCl; JW Pharmaceutical, Korea). Arterial pulse pressure data, which are analyzed in the transducer, were transmitted from the multiparameter monitor to the LiDCO™ monitor. Stroke volume (SV), CO, and SVR data were continuously calculated in real-time using LiDCO. The end-expiratory partial pressure of carbon dioxide (ETCO2), respiratory rate (RR), and expired fraction of isoflurane (FE'Iso) were measured using a side-stream infrared gas analyzer (CARESCAPE Monitor B650; GE Healthcare, Finland), and tidal volume (TV) and minute volume (MV) were recorded using a volume monitor (9100c NXT; GE Healthcare, China). If ETCO2 was above 60 mmHg, intermittent positive pressure ventilation (IPPV) was started.

2.3. LiDCO™ Monitor Calibration

After stabilization, echocardiogram was performed using an ultrasound machine (Versana Active; GE Healthcare, Finland) to determine the CO and calibrate the LiDCO™ monitor. In the present study, the velocity–time integral method was applied to measure the CO. This method was performed by measuring the velocity of the left ventricular outflow tract in the left apical five-chamber view using pulsed-wave Doppler. Subsequently, the aortic diameter was measured in the right parasternal long-axis view. SV was calculated using the following formula: $\pi \times$ (aortic diameter/2)2, and HR was multiplied to obtain the CO. The CO of each dog was recorded as the mean of three consecutive measurements.

2.4. Veress Needle Placement

The ventral region of the abdomen was clipped and aseptically prepared. Subsequently, a skin incision was made 20 mm caudal to the umbilicus. A 14-gauge Veress needle (Veress needle, ADDLER, India) was inserted in the incision site, and the correct needle placement was checked by the injection of 5-mL normal saline, followed by aspiration. The Veress needle was connected to an automatic CO2 insufflator (UHI-3; Olympus Optical Inc., Japan), which can adjust the IAP.

2.5. Experimental Design

The instrumentation period was standardized to 60 min. Subsequently, FE'Iso was maintained at 1.73% (1.3 × MAC) [17], and the dogs were allowed to breathe spontaneously. Thereafter, baseline data of the following physiologic variables were recorded: HR, SpO2, esophageal temperature, SAP, DAP, MAP, EtCO2, RR, TV, MV, pH, PaO2, PaCO2, HCO3−, BE, SV, CO, and SVR. After data collection, the IAP was gradually increased to 5, 10, 15, and 20 mmHg by insufflating CO2; the dogs stabilized for 10 min between each of the IAP. Measurements of physiological variables were performed at the following six timepoints: T0 (baseline), right after the instrumentation period; T1, 10 min following IAP adjustment to 5 mmHg; T2, 10 min following IAP adjustment to 10 mmHg; T3, 10 min following IAP adjustment to 15 mmHg; T4, 10 min following IAP adjustment to 20 mmHg; and T5, 10 min following desufflation. Following desufflation, meloxicam (0.2 mg/kg, subcutaneously [SC]) was administered to all dogs before recovery from anesthesia. All catheters were removed and returned to their housing facility.

After a 1-week washout period, the dogs underwent similar procedures until 10 min before T0, and an IV loading dose of dexmedetomidine (3 μg/kg) was administered followed by an infusion at 3 μg/kg/hr of dexmedetomidine. Immediately following the administration, the FE'Iso was decreased to 0.72% based on a previous study [6] to maintain isoflurane–dexmedetomidine-administered dogs equipotent to 1.3 MAC isoflurane-administered-alone dogs. Subsequently, measurements of baseline variables at T0 were recorded, and a similar experimental protocol was performed with infusions of

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dexmedetomidine. The dogs that were not administered with dexmedetomidine were assigned as the control group and those administered with dexmedetomidine were assigned as the Dex group.

2.6. Statistical Analysis

Normal distribution of data was confirmed using the Shapiro–Wilk test before analysis. All values were presented as means and standard deviations. To compare each physiological variable within the two groups, a repeated measures one-way analysis of variance was performed. Additionally, the Kruskal–Wallis test was performed for nonnormally distributed data. If significant differences were identified, a post-hoc analysis was performed using the Bonferroni test. A Wilcoxon signed-rank test or Mann–Whitney U test was performed for each variable between the two groups at each time point. A p value of <0.05 was considered statistically significant. Analyses were performed using Statistical Package for the Social Sciences for Windows (version 25, IBM, Armonk, NY, USA).

3. Results

All five dogs completed the study without any complications. CO measurements using echocardiogram of five dogs ranged from 1.264 to 1.75 (mean, 1.47) L/min. No significant changes were observed in body temperature (35.1℃–37.9℃) and oxygen saturation (94%–100%) over time and between treatments.

Cardiovascular changes due to increased IAP with carbon dioxide and dexmedetomidine administration are summarized in Table 1. No significant differences were observed in variables between different IAPs in both groups. In the control group, HR, MAP, SAP, and DAP were increased in T3 and T4 compared with those in T0; however, the results were not statistically significant (HR, p = 1.000, p = 1.000; MAP, p = 1.000, p = 0.399; SAP, p = 1.000, p = 0.293; and DAP, p = 0.825, p = 0.141, statistical results of T3 and T4 compared with T0, respectively). Compared with the variables of the control group, overall significant effects of dexmedetomidine were observed on HR, CO, and SVR during the experiment. SAP, MAP, and DAP increased in T0, T1, T2, and T5 with dexmedetomidine treatment, whereas no differences were noted in T3 and T4 between the control and Dex groups. In the Dex group, the values for SV significantly decreased in T0, T1, and T2.

Table 1. Mean and standard deviation of cardiovascular variables across different IAPs between the control and dexmedetomidine groups.

		T0 (IAP 0 mmHg)		T1 (IAP 5 mmHg)		T2 (IAP 10 mmHg)		T3 (IAP 15 mmHg)		T4 (IAP 20 mmHg)		T5 (IAP 0 mmHg)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
HR (/min)	Control	103.0	13.7	111.2	13.5	110.8	15.9	114.8	11.9	116.0	14.4	117.0	14.6
	Dex	81.0†	11.0	71.2†	10.0	70.0†	17.7	69.8†	9.2	67.8†	9.09	64.8†	4.8
SAP (mmHg)	Control	97.0	18.9	94.4	9.0	91.2	7.6	112.6	16.4	122.6	24.6	95.0	14.3
	Dex	154.2†	14.0	146.0†	11.1	140.8†	10.4	134.6	8.7	135.2	11.0	131.4†	12.1
MAP (mmHg)	Control	68.4	17.5	68.0	8.2	66.6	7.7	84.8	15.1	90.8	22.2	67.4	13.9
	Dex	124.0†	11.8	114.0†	9.8	103.8†	14.7	98.4	8.6	97.8	13.1	96.8†	8.2
DAP (mmHg)	Control	53.6	17.6	53.6	9.8	53.0	8.6	72.6	16.0	80.2	20.6	56.4	12.9
	Dex	111.0†	11.1	98.4†	12.5	88.6†	13.2	84.2	9.2	84.0	14.1	83.0†	10.0
CO (L/min)	Control	1.5	0.4	1.5	0.4	1.5	0.4	1.3	0.3	1.2	0.4	1.6	0.3
	Dex	0.7†	0.2	0.6†	0.2	0.7†	0.2	0.8†	0.2	0.6†	0.2	0.7†	0.1
SV (mL)	Control	13.7	2.7	13.7	2.5	13.0	2.6	11.1	2.1	11.1	2.1	13.4	2.7
	Dex	7.8†	1.4	8.2†	1.6	9.1†	1.9	9.8	1.6	10.1	1.4	10.4	1.0
SVR	Control	3,621.1	1,416.7	3,467.5	1,272.1	3,563.2	1,267.3	5,043.2	1,972.0	5,962.9	2,286.7	3,436.2	1,302.9

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(Dynes/s/cm^{-5})	Dex	17,577.4†	5,397.8	18,031.9†	8,199.3	13,097.5†	3,921.4	10,535.2†	2,745.0	12,548.2†	4,613.3	11,417.9†	2,510.2

IAP, intra-abdominal pressure; HR, heart rate; SAP, systolic arterial blood pressure; MAP, mean arterial blood pressure; DAP, diastolic arterial blood pressure; CO, cardiac output; SV, stroke volume; SVR, systemic vascular resistance; SD, standard deviation. *Significantly different from the baseline (T0) within the group ($p < 0.05$); however, no differences are observed in cardiovascular variables between different time point. †Significantly different from the control group at the same time point in the dexmedetomidine group ($p < 0.05$).

The effects of intra-abdominal insufflation with carbon dioxide on respiratory and blood gas variables in the control and Dex groups are summarized in Table 2. No significant differences in EtCO2, RR, TV, MV, pH, PaO2, PaCO2, HCO3−, and BE were observed between different IAPs and between the two groups. In both treatments, PaCO2 was increased following carbon dioxide insufflation compared with that at baseline T0; however, the results were not statistically significant. Moderate-to-severe hypercapnia (EtCO2 > 50 mmHg) was detected in all five dogs. One dog showed an EtCO2 of >6 mmHg in both treatment periods, and IPPV could not resolve the hypercapnia.

Table 2. Mean and standard deviation of respiratory and blood gas variables across different IAPs between the control and dexmedetomidine groups.

		T0 (IAP 0 mmHg)		**T1 (IAP 5 mmHg)**		**T2 (IAP 10 mmHg)**		**T3 (IAP 15 mmHg)**		**T4 (IAP 20 mmHg)**		**T5 (IAP 0 mmHg)**	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
RR (/min)	Control	13.2	6.3	14.6	5.9	17.4	7.8	17.2	5.4	20.2	6.5	17.4	4.5
	Dex	12.4	2.7	14.2	4.2	18.8	6.8	21.0	7.2	21.0	6.9	15.4	2.1
$EtCO_2$ (mmHg)	Control	55.4	5.4	58.0	7.3	56.0	6.5	59.2	8.6	56.4	11.5	53.4	7.0
	Dex	51.0	4.5	50.6	5.0	51.0	5.2	53.6	7.0	53.2	8.4	44.8	3.0
TV (ml/min)	Control	139.6	27.8	136.0	24.9	136.8	32.4	129.6	14.5	125.6	17.5	155.6	32.1
	Dex	146.0	17.7	155.6	6.6	130.8	4.9	117.8	10.2	113.4	12.2	158.8	10.9
MV (L/min)	Control	1.9	0.7	1.7	0.8	2.2	0.6	2.0	0.8	2.5	1.1	2.7	0.5
	Dex	1.9	0.6	2.1	0.7	2.6	1.0	2.4	1.0	2.4	0.9	2.5	0.5
pH	Control	7.4	0.1	7.3	0.0	7.3	0.1	7.3	0.1	7.3	0.1	7.3	0.1
	Dex	7.4	0.1	7.3	0.1	7.3	0.1	7.3	0.1	7.3	0.1	7.4	0.1
PaO_2 (mmHg)	Control	447.4	60.9	472.6	70.5	477.2	113.4	503.6	73.7	516.8	93.6	508.6	81.1
	Dex	481.2	62.3	520.2	29.9	521.2	48.9	522.6	25.7	525.0	56.4	538.8	53.9
$PaCO_2$ (mmHg)	Control	53.6	6.8	56.9	9.0	56.3	8.5	59.9	9.5	57.4	7.0	54.7	8.2
	Dex	47.6	5.1	52.0	4.4	54.6	5.6	53.9	5.9	56.4	10.0	51.1	8.8
HCO_3^- (mmol/L)	Control	30.1	2.9	29.4	2.7	29.5	3.0	28.9	4.3	28.5	4.4	27.8	3.6
	Dex	26.5	4.9	26.8	4.9	27.4	5.4	27.0	5.8	28.1	6.0	28.2	5.0
BE (mmol/L)	Control	5.1	3.0	4.0	2.4	4.0	3.1	3.0	4.6	2.7	4.5	2.2	3.8
	Dex	1.6	5.6	1.5	5.6	1.9	6.0	1.5	6.4	2.4	6.4	3.1	5.3

IAP, intra-abdominal pressure; RR, respiratory rate; ETCO; $EtCO_2$, end-tidal carbon dioxide; TV, tidal volume; MV, minute volume; pH, potential hydrogen; PaO_2, partial pressure of oxygen; $PaCO_2$, partial pressure of carbon dioxide; HCO_3^-, bicarbonate; BE, base excess; SD, standard deviation. In respiratory and blood gas variables, no significant differences are observed between different IAPs and between the control and dexmedetomidine group.

 doi:10.20944/preprints202308.0654.v1

4. Discussion

This study compared cardiovascular and respiratory variables in anesthetized dogs between different sequentially increased IAPs and between groups administered with dexmedetomidine or not. Overall, an increased IAP of until 20 mmHg did not significantly affect cardiovascular and respiratory variables in the control group which were not premedicated. In the dexmedetomidine-administered group, the hemodynamic changes were compared with those in the control group however, during abdominal insufflation, cardiovascular and respiratory alterations were not observed.

The cardiovascular effects of increased IAP are induced by cranial disposition of the diaphragm and compression of the caudal vena cava, thereby resulting in direct compression to the heart and contributing to reduced preload and CO [1]. In the present study, in the control group, the HR and arterial pressure were slightly but statistically insignificantly increased following the IAP was increased to 15 and 20 mmHg. Furthermore, the slight increase in arterial pressure during insufflation may have been caused by an increase in SVR and a decrease in CO and SV. These results are consistent with those previously reported that decreased CO and increased SVR were observed following an increased IAP [18-20]. However, these studies were conducted using high pressure (>20 mmHg). Therefore, Duke et al. investigated using lower IAPs (15 mmHg), and the results showed increased SVR and maintained CO [21]. The maintenance in CO in that study seemed to be caused by a low IAP; however, in our study, slightly decreased CO was observed at an IAP of 15 mmHg. These results suggest that IAPs higher than 15 mmHg have contributed to the alteration in cardiovascular variables. The present study demonstrated the cardiovascular effects of dexmedetomidine, with the dexmedetomidine group showing increased arterial blood pressure and decreased HR. These results are consistent with those of other studies evaluating the cardiovascular effects of alpha-2 agonists, and these effects are believed to be because of the baroreceptor-mediated response, which is a widely accepted side effect of alpha-2 agonists [5,6,8].

As previously described, alpha-2 agonists and abdominal insufflation can both influence the cardiovascular system. In human medicine, several studies have been performed to evaluate the effects of dexmedetomidine on hemodynamic variables in patients undergoing laparoscopic procedures. The dexmedetomidine infusion rates applied in these studies were 0.1 and 0.2 μg/kg/h, respectively, and dexmedetomidine provided a stable hemodynamic status during laparoscopic surgery [9,10]. In the present study, the dexmedetomidine loading dose was 3 μg/kg, with an infusion rate of 3 μg/kg/h; the results showed that, in the dexmedetomidine-administered group, the cardiovascular variables following abdominal insufflation (T1–5) were not significantly different compared with those at baseline (T0). These findings suggest that hemodynamic stability was observed in anesthetized dogs administered with 3 μg/kg/h of dexmedetomidine with abdominal insufflation until 20 mmHg. However, our study was conducted with only one dexmedetomidine dose; therefore, to evaluate the effects of dexmedetomidine on hemodynamic status in laparoscopic procedures with different dexmedetomidine doses, further study is warranted.

In the present study, CO, SV, and SVR were measured using the LiDCO™ monitor, which calculates CO from the pulse contour analysis. Several studies were conducted to evaluate the PulseCO method and showed positive results, which are correlated with the PAC-TD method, the gold standard method [13,22,23]. As the PulseCO method is developed for measurements in human medicine, one-point calibration is necessary, and these studies calibrated the monitor with CO measured using the lithium-dilution or thermodilution method [13,22,23]. However, in veterinary clinical settings, there is no accepted method for measuring CO. Therefore, this study adapted a novel technique that calibration was conducted with CO measurement using echocardiogram to maximize the advantages of being less invasive, considering that lithium- and thermodilution methods are relatively invasive with risks of complications. Furthermore, the PulseCO method provides a significant advantage for patients by monitoring CO trends in real-time, which is clinically essential in determining patient care [13]. However, despite the advantages of the PulseCO method, it also has limitations. One study that compared CO between echocardiogram and thermodilution method reported that underestimated COs were measured in echocardiographic methods [24]. Moreover, the artifacts of echocardiogram have been described as causes of CO underestimation [24]. However, the CO measurements of our study obtained using the LiDCO™ monitor may have underestimated results; therefore, to determine the reliability of this novel technique, further studies are needed.

 doi:10.20944/preprints202308.0654.v1

Previous studies have shown that respiratory disturbances associated with increased IAPs can be observed in pediatric human patients and animals [20,25]. These effects are believed to be induced by cranial disposition of the diaphragm, which results in decreased functional residual capacity, residual volume, and total lung capacity [1]. Richardson et al. stated that significant respiratory effects were noted at IAPs above 25 mmHg, and our findings also showed that no significant differences were observed in respiratory variables until IAPs of 20 mmHg. However, although our results were not statistically significant, PaCO2 increased following abdominal insufflation, peaked at T3 and T4 in the control and Dex groups, respectively, and decreased following desufflation. These results suggest that IAPs below 20 mmHg induced by capnoperitoenum affect respiratory function but with a minor effect. Another respiratory disturbance identified in the present study was that all dogs exhibited moderate-to-severe hypercapnia (EtCO2 > 50 mmHg) throughout the anesthesia period. While the causes of hypercapnia can be diverse, in this study, it is believed that respiratory suppression due to anesthetics was the main factor leading to hypercapnia rather than increased IAP [26-28]. This is supported by a previous study on conscious dogs, which showed that hypercapnia was not observed throughout the experiments [29].

In one dog, in the current study, hypercapnia (EtCO2 > 50 mmHg) was observed before abdominal insufflation; after IAP insufflation of up to 15–20 mmHg, end-tidal carbon dioxide concentration reached more than 75 mmHg. Despite persistent manual positive pressure ventilation, severe hypercapnia did not resolve. After desufflation, EtCO2 levels decreased to the level before insufflation. However, why this dog exhibited severe hypercapnia remains unclear.

The present study had several limitations. First, this study was conducted with a small sample size. To minimize the influence of individual animal effects on physiological variables, the dogs used in this study were of the same breed and similar size and age, and a crossover design was applied. Second, the CO2 insufflator has an automatic relief function that automatically releases excess pressure when pressure exceeds 20 mmHg. Thus, our study could not evaluate the effects of IAPs over 20 mmHg. Third, plasma drug concentrations were not measured such that possible variations in concentration and their influence on circulation cannot be assessed within the present study. Lastly, the body positions of the dogs were not considered in the present study. Laparoscopic procedures are commonly performed in different positions, such as inverse or reverse Trendelenburg positions; as these positions influence cardiovascular variables, further study is needed to evaluate physiologic parameters in different positions.

5. Conclusions

Cardiovascular and respiratory effects of increased IAP up to 20 mmHg were not significant in healthy beagle dogs. Furthermore, the dogs administered with dexmedetomidine were not affected by abdominal insufflation. Therefore, this study has shown that dexmedetomidine infusion administration may be applicable in laparoscopic procedures in healthy dogs. However, further study is warranted as the present study was conducted in only one position and limited IAP.

Author Contributions: Conceptualization, Dongseok Kim and Min Jang.; methodology, Minjun Seo.; software, Geonho Choi.; validation, Sungin Lee., and Sang-Kwon Lee.; formal analysis, Won-Jae Lee.; resources, Young-Sam Kwon.; data curation, Sung-Ho Yun.; writing—original draft preparation, Dongseok Kim.; writing—review and editing, Min Jang.; supervision, Min Jang.; project administration, Min Jang.; funding acquisition, Min Jang. All authors have read and agreed to the published version of the manuscript.

Funding: This work was supported by a National Research Foundation of Korea (NRF) grant funded by the Korean government (Ministry of Science and ICT) (No. 2021R1G1A1013034).

Conflicts of Interest: The authors declare no conflict of interest.

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