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RESEARCH ARTICLE

Induction and Intubation Effects on Hemodynamic Response Using Slow Propofol or Ketamine- Slow Propofol Infusion in Dogs: An Experimental Study

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ABSTRACT

Propofol is widely used in anesthetic induction in both human and veterinary medicine. However, it has hemodynamic adverse effects such as hypotension and bradycardia, particularly at rapid infusion rate. This study aimed to attenuate the hemodynamic changes during induction and investigate the hemodynamic response to tracheal intubation using slow propofol or ketamine- slow propofol infusion in dogs. Eight dogs were assigned to two groups: Group1 received intravenous slow propofol 1 mg/kg/min for anesthesia induction and Group2 received intravenous ketamine bolus 2 mg/kg prior to slow propofol 1 mg/kg/min for anesthetic induction. The propofol infusion rate continued until achieving the appropriate conditions for intubation. Heart rate and arterial blood pressure were measured 30 minutes after premedication (baseline), one minute after induction and intubation. The results showed that the propofol dose required for induction and intubation was significantly lower in Group 2 (1.55±0.37 mg/kg) than Group1 (3.56±0.44 mg/kg), with P = 0.01. After induction, Group 2 exhibited a sharp increase in heart rate (96.2 ±2.72a) compared to Group1 (60 ± 3.14c) with P < 0.001. Both groups showed non-significant changes in arterial blood pressure after induction. Meanwhile, Group 1 showed more variability and less stability in response to intubation. Also, it exhibited more significant fluctuations in systolic, diastolic and mean arterial blood pressure levels after intubation. Group 2 maintained greater stability in arterial blood pressure in response to tracheal intubation. In conclusion and from the obtained results, slow propofol or ketamine- slow propofol combination were effective in mitigating hemodynamic fluctuations following induction. However, the ketamine-slow propofol combination achieved better stability in arterial pressure post-intubation than slow propofol alone.

Keywords: Induction, Intubation, Slow Propofol, Ketamine-Slow Propofol, General Anesthesia.

Introduction

Hemodynamic fluctuations are indeed a critical concern during anesthetic induction and intubation process. These fluctuations often arise from the stress caused by the anesthetic agents and the mechanical act of intubation [1]. Apnea and decreased mean arterial pressure are common side effects of anesthetic induction, which can be particularly risky in sick and debilitated animals, where

maintaining steady cardiovascular and respiratory function is essential [2].

Propofol is a hypnotic agent commonly used for induction and maintenance of general anesthesia in dogs [3-4]. Propofol produces rapid and smooth induction, however it induces hypotension, respiratory depression and bradycardia in a dose- and rate- dependent manner [2,3,5-9]. Propofol is associated with post-induction apnea at rapid rates, depending on the concentration of propofol in the brain. Postinduction apnea (PIA) and decreased mean arterial pressure commonly occur after propofol induction, are associated with the total dose needed for anesthetic induction and the rate of administration [3,10-12]. Slower propofol administration compared to precalculated dose can produce a decrease in the required amount of propofol for induction of anesthesia and endotracheal intubation in dogs [2, 7, 11].

Ketamine, a dissociative agent, increases cardiovascular activity in contrast to many other anesthetics. This is indicated by increased heart rate, arterial blood pressure and cardiac output. It induces a sympathetic response that can counteract the depressant effects of anesthetic drugs, such as propofol and xylazine when combined with these anesthetics and maintains cardiovascular and respiratory stability during anesthesia [13-15]. The reduction in heart rate was less pronounced when ketamine-propofol co-administrated in separate syringes compared to propofol alone at anesthesia induction [13].

Like humans, endotracheal intubation is a common procedure in veterinary practice and can produce significant sympathetic stimulation resulting in increased heart rate, arterial blood pressure and serum concentrations of catecholamines which can be dangerous in patients with cardiovascular diseases or hypertensive patients [16-18]. Although propofol is widely used for induction in veterinary anesthesia, there is a lack of research on the effects of slow propofol or ketamine-slow propofol on the hemodynamic response during induction and intubation.

Therefore, the aim of the current study is to mitigate the hemodynamic changes during anesthesia induction and evaluate the hemodynamic response to tracheal intubation using slow propofol or ketamine-slow propofol infusion.

Materials and Methods

The study was conducted at the Department of Surgery, Anesthesiology, and Radiology, Faculty of Veterinary Medicine, Zagazig University, Egypt after getting an approval (ZU-IACUC/2/F/332/2023) from Zagazig University Institutional Animal Care & Use Committee. and performed.

Animals

The experiment was conducted on eight male mongrel dogs (9-12 months old) and weighed (20-25kg). All the examined dogs arrived 1 week prior to the procedure, to acclimate the environment and were housed in separate cages. These dogs received an anthelmintic treatment (1ml/ 50kg S/C) (Dectomax®, Zoetis, USA) to ensure they were free of internal and external parasites prior to the experiment. These dogs were categorized as class I (healthy without disease) based on physical examination, chest radiography and abdominal ultrasonography according to the American Society of Anesthesiologists (ASA) physical status classification system (ASAPS). Dogs were fasted 12

hours and had free access to water till premedication.

Anesthetic procedure

All dogs received an intramuscular injection of xylazine (0.7 mg/kg, Xylaject®, Adwia Co., Egypt) combined with nalbuphine (0.5 mg/kg, Nalufin®, Amoun Pharmaceuticals Co., Egypt) in the same syringe, along with meloxicam (0.2 mg/kg, Mobitil®, Medical Union Pharma, Egypt). Twenty minutes later, a 20-gauge intravenous catheter was inserted in the cephalic vein for the administration of the anesthetic agents.

Preoxygenation with 3 L/min of oxygen for 5 minutes was done in all examined dogs using facemask prior to the induction of general anesthesia. Dogs under study were randomly allocated into 2 groups: Group 1 (G1) (n=4) received slow propofol at a rate of 1 mg/kg/min (Propofol® 1% Fresenius; Fresenius Kabi Co LTD., Germany) as the control group, Group 2 (G2) (n=4) received an intravenous bolus of ketamine (2 mg/kg, Ketam®; Egyptian International Pharmaceutical Industries Co., EPICO., Egypt), administrated manually over 15 seconds, 5 minutes before slow propofol administration at 1 mg/kg/min. The propofol infusion continued until the patient met the criteria for intubation, which include absence of palpebral reflex, jaw tone, swallow reflex, and tongue movement in response to traction or the placement of the laryngoscope blade. Intubation was performed in all dogs by positioning a miller's laryngoscope blade at the tongue base, applying gentle pressure while pulling the tongue out of the mouth. This maneuver lowered the epiglottis, exposed the entrance of trachea, allowing the placement of an appropriately sized cuffed endotracheal tube (KRUUSE PVC Endotracheal Tubus, China) into the trachea. The correct size of endotracheal tube was chosen by palpating the outer diameter of the trachea at the mid-neck region, selecting either an internal diameter (I.D) of 9.0 mm or 10.0 mm). The cuffs of endotracheal tubes were inflated with air until no noise was heard, and the adjustable pressure-limiting valve was closed at a pressure of 20 mm H2O to ensure a proper seal. The propofol rate was programmed into a syringe pump (injectomate Agilia®; Fresenius Kabi Co., Germany) and set in ml/h for the induction of anesthesia in both groups. The propofol dose (mg/kg) and the volume of propofol (ml/kg) for intubation were recorded in all animals.

Heart rate (HR) was measured by electrocardiography (ECG) lead II displayed on a multiparametric monitor. Electrocardiography was conducted using four electrodes attached to the skin at the levels of elbow and stifle. The red electrode was placed on the right forelimb, the black electrode on the right hindlimb, the yellow electrode on the left forelimb, and the green electrode on the left hindlimb. ECG measurements were monitored with the animals positioned in right lateral recumbency. Non-invasive blood pressure (NIBP) was measured by placing a cuff (NIBP Cuff Neonate, Dräger®, Drägerwerk AG & Co. Lübeck, Germany) was placed above the hock joint and its width was approximately about 40% of the limb's circumference. All measurements including HR and arterial blood pressure were done, using a multiparametric monitor (Vista 120, Dräger®, Drägerwerk AG & Co., Lübeck, Germany). Incidence of post-induction apnea (PIA) (defined as an absence of spontaneous breathing for longer than 30 seconds), was recorded in both groups. If post-induction apnea occurred, manual ventilation was administrated using a

rebreathing bag. A positive pressure breath to 10-15 cmH_2O was delivered every 15 seconds till spontaneous breathing resumed [19]. In addition, bradycardia (HR less than 60 beats/min) and hypotension (MAP less than 60 mmHg) were recorded during the procedure.

Data collection

Baseline values of heart rate (HR), systolic arterial pressure (SAP), diastolic arterial pressure (DAP), and mean arterial pressure (MAP) were recorded 30 minutes after premedication and prior to propofol induction. These values were measured 1 min immediately after induction, following tracheal intubation.

Statistical analysis

Data were analyzed using SPSS version 25. Data were described as mean ±SD. Data were screened for normality using the Shapiro-Wilk test and Levene's test for assessing homogeneity of variance. Repeated measures ANOVA was performed after checking the sphericity assumption and correcting violations using Green House Geiser [20]. The significant interaction results were followed up by Duncan's multiple comparison post hoc test to investigate differences over time points of operation. The significance level is considered at $P < 0.05$.

Results

All dogs exhibited bradycardia following premedication. The propofol dose required to allow endotracheal intubation in G2 (1.55±0.37 mg/kg), was lower than that for G1 (3.56±0.44 mg/kg), with P = 0.01. Meanwhile the volume of propofol was (0.36±0.05 ml/kg) and (0.15±0.04 ml/kg) for G1 and G2, respectively, with P=0.01.

Baseline values of HR, SAP, DAP, and MAP did not significantly differ between both groups. G2 showed more variability in HR at induction and intubation compared to G1. Both groups recorded a significant increase in HR levels from their baseline values; however, G2 showed a sharp rise in HR level (96.2 $\pm 2.72^{a}$) than G1 (60 ± 3.14^{c}). After tracheal intubation, G1 showed a significant increase in HR level (86.7± 3.14^{ab}), while G2 exhibited a slight decrease (82.8 $\pm 2.73^{b}$) compared to the HR value at induction that did not significantly differ from G1's HR level. Overall, G1 showed more variability and less stability in response to intubation (Table 1 and Figure 1).

At induction, there were non-significant differences in SAP, DAP, MAP levels in G1 and G2 compared to their baseline values. However, G1 showed a more stable SAP level (133 ± 4.43 ^{c}) than G2 (146.5 ± 3.83 ^{abc}) compared to baseline values, although the results were not statistically significant (Table1 and Figure 2A). Neither group experienced hypotension at induction in both groups. While there was a slight (non-significant) decrease in DAP level in G1 compared to the baseline, G2 maintained stable DAP level (93.2 ± 5.74^{ab}) at induction, as shown in (Table 1 and Figure 2B).

After tracheal intubation, significant increases were observed in SAP, DAP, MAP values compared to their induction values in G1, while G2 maintained stable levels of these parameters in response to tracheal intubation compared to their values at induction. Overall, G1 exhibited more significant fluctuations in SAP, DAP and MAP levels after intubation, whereas G2 maintained more hemodynamic stability in response to tracheal intubation than G1 (Table1 and Figures2A, B, C). Post-induction of apnea was not observed in both groups.

Table 1. Hemodynamic changes in heart rate (beats/min), systolic arterial pressure (SAP) mmHg, diastolic arterial pressure (DAP) mmHg and mean arterial pressure (MAP) mmHg at baseline (30 minutes after premedication), at induction, and post-tracheal intubation.

^{abc} means with different superscript are statistically different P<0.05.*significant difference P< 0.05; ** highly significant difference P< 0.001.

	Time	G1	G2	*P. value*
HR (beats/min)	**Baseline**	**43.2 ± 2.72 ^{d}**	**53.2 ± 2.72 ^{d}**	**< 0.001****
	At induction	**60 ± 3.14 ^{c}**	**96.2 ±2.72^{a}**	
	Tracheal Intubation	**86.7± 3.14^{ab}**	**82.8 ±2.73 ^{b}**	
SAP (mmHg)	**Baseline**	**132 ± 3.81 ^{c}**	**138 ± 3.83 ^{bc}**	**< 0.001****
	At induction	**133 ± 4.43 ^{c}**	**146.5 ± 3.83 ^{abc}**	
	Tracheal Intubation	**153 ± 4.43 ^{ab}**	**158 ± 3.83 ^{a}**	
DAP (mmHg)	**Baseline**	**79.8 ± 5.74 ^{b}**	**93.2 ± 5.8 ^{ab}**	**0.02***
	At induction	**77.7 ± 6.62 ^{b}**	**93.2 ± 5.74^{ab}**	
	Tracheal Intubation	**104.3 ± 6.62^{a}**	**105.8 ± 5.74^{a}**	
MAP (mmHg)	**Baseline**	**102.00 ± 3.33^{bc}**	**108.5 ± 3.33 ^{abc}**	**0.04***
	At induction	**98.7 ± 3.84 ^{c}**	**111.9 ± 3.33 ^{ab}**	
	Tracheal Intubation	**111.7 ± 3.84 ^{ab}**	**113.2 ± 3.33^{a}**	

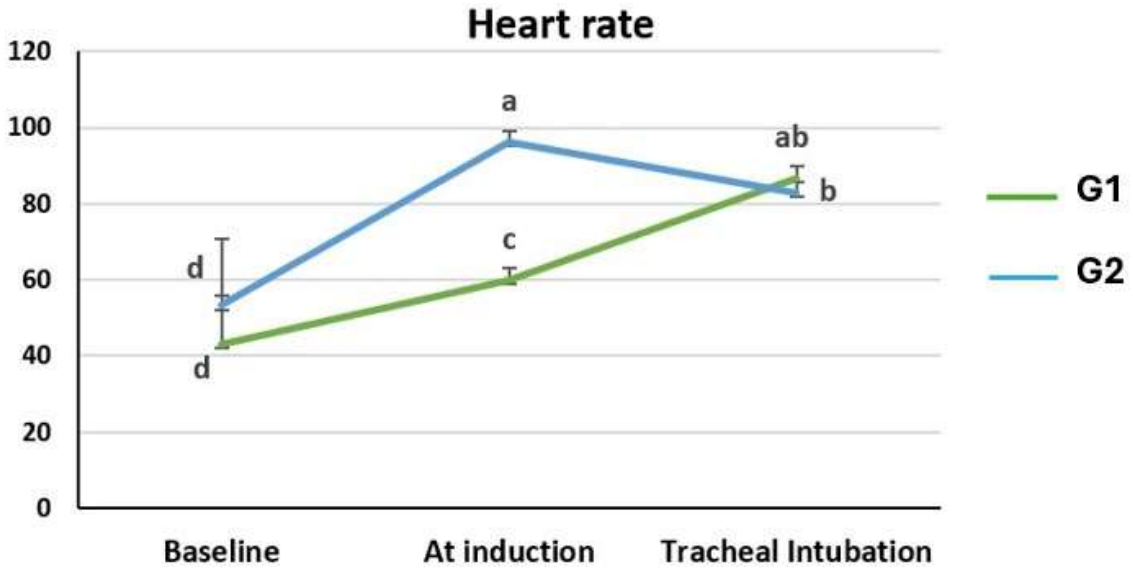

Figure 1. Shows heart rate changes at baseline (30 min after premedication), at induction, and after tracheal intubation in G1 and G2. Note that groups not sharing the same letter (a, b, c) indicate statistically significant differences

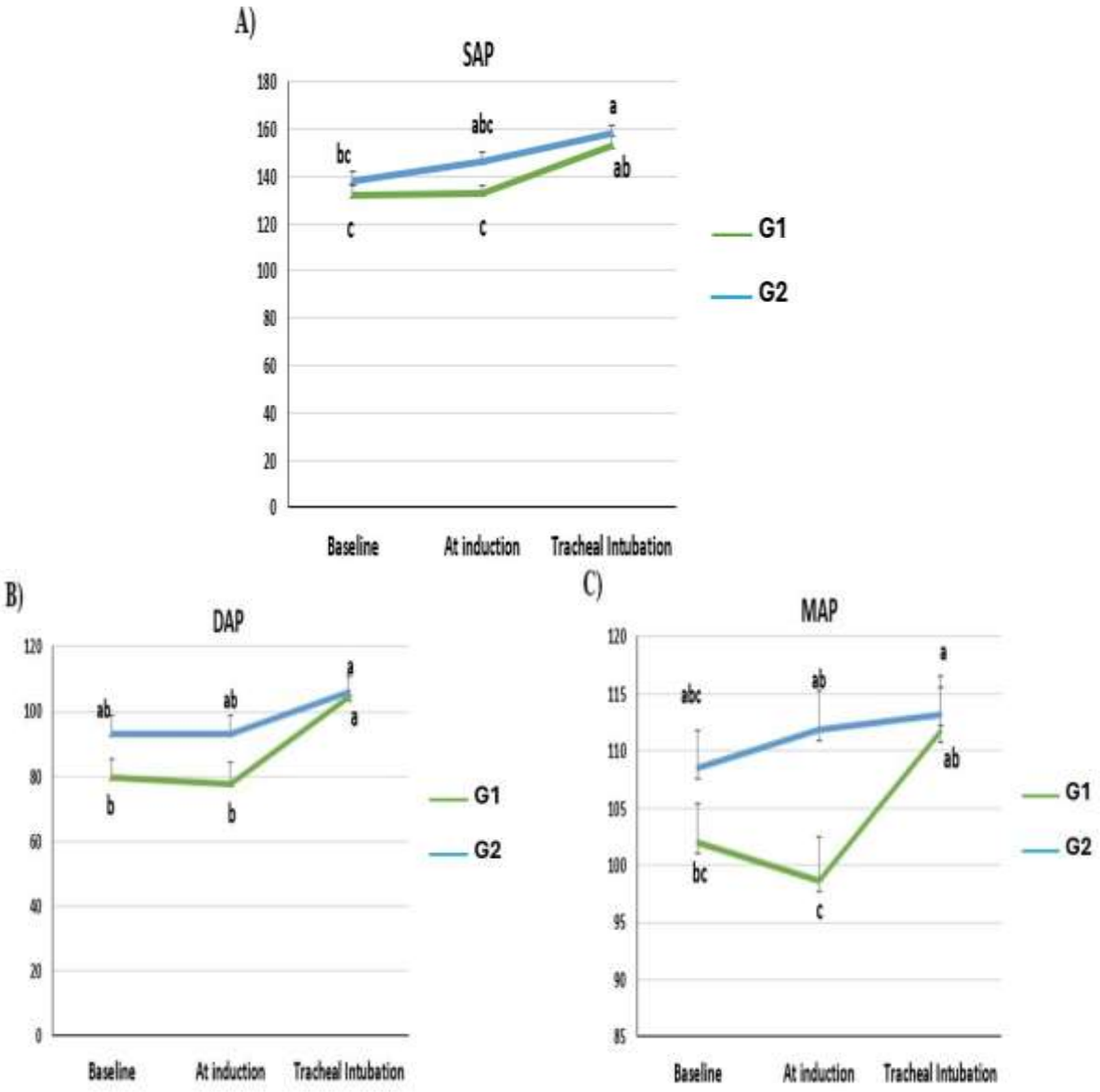

Figure 2.A: Shows systolic arterial pressure (SAP) (mmHg) changes, B: Showing diastolic arterial pressure (DAP) (mmHg) changes, C: Showing mean arterial pressure (MAP) (mmHg) changes, at baseline (30 min after premedication), at induction, and after tracheal intubation in G1 and G2. the groups not sharing the same letter (a, b, c) indicate statistically significant differences

Discussion

Bradycardia observed after premedication could be attributed to xylazine, an α2- agonist that decreases sympathetic outflow from the central nervous system (CNS) and mediate vagal activity, producing further slowing of the conduction system of the heart [21]. In the present study, the nalbuphine-xylazine combination effectively decreased perioperative stress, excitement and sympathetic stimulation from restraining and injections that improved anesthesia safety by decreased anesthetic requirement for induction. This finding aligns with Lester *et al.* [22], who found that nalbuphine-xylazine decreased stress and provided greater sedation, analgesia in dogs with discomfort compared to xylazine alone.

The present study showed that slow propofol administration was associated with decreased propofol requirement and with fewer adverse effects when compared to fast propofol infusion rate of 4 mg/kg/min and rapid bolus administration of 4 mg/kg over 60 seconds, as reported by [2, 11, 19, 23]. Moreover, the propofol doses required for induction and intubation in aforenamed studies were (4.1±0.7, 5.0 ± 1.0, 3.9 ±1.3, and 4±0.0 mg/kg), respectively, which were higher than the propofol dose (3.56±0.44 mg/kg) used in the current study.

The propofol dose resulting from slow propofol administration in the present study is consistent with the findings of Hristova *et al.* [24]**,** who demonstrated that the same administration rate resulted in a propofol dose of (3.3 ± 1.0 mg/kg).

In contrast, another study found that propofol was used at a dose of (1.8 ± 0.9 mg/ kg) for intubation [17]. They used propofol-ketamine mixture (1:1) in the same syringe delivered at a rate of 1.8 mg/kg/min of each for induction. This dose was slightly higher than our propofol doses (1.55±0.37 mg/kg) when ketamine and propofol were administered separately.

However, the propofol dose used for induction in the present study was higher than the (1.8 ± 0.6 mg/kg) as reported by Bigby *et al.* [11] following slow propofol at 1 mg/kg/min, this may be attributed to the deep sedation achieved by using a high dose of methadone (0.5 mg/kg) combined with dexmedetomidine (5 µg/kg) in the aforementioned protocol, compared to xylazine and nalbuphine used in the current study. Our propofol dose was similar to that 3.7 ± 1.1 mg/kg studied by Raillard *et al.* [2] following 1.3 mg/kg/min of propofol.

Similarly, in cats a study demonstrated that administrating propofol at a slower rate (1mg/kg/min) resulted in reduced propofol induction doses of (5.1 ± 1.5 mg/kg) compared to the faster propofol administration (4mg/kg/min), which required doses of (9.1 ± 1.8 mg/kg) in cats premedicated with tramadol [25]. Additionally, fast propofol administration at 8 mg/kg/min was associated with a higher propofol requirement of (5±0.9 mg/kg) compared to slow propofol 2 mg/kg/min, which required (3.8±0.7 mg/kg) [26]. These findings supported the benefits of slow propofol administration in reducing the total propofol dose required for induction and minimizing the adverse effects, such as apnea, hypotension, and bradycardia. The cause of higher doses observed with fast propofol can be attributed to the delayed time to achieve equilibrium between

plasma and cerebral concentrations. This delay consequently postpones the desired clinical effect, resulting in overshooting the necessary dose for anesthesia induction [27].

On the other hand, a previous study revealed that excessively prolonged propofol administration caused disequilibrium between blood and brain concentrations in sheep, potentially preventing the achievement of anesthesia [28].

Some investigators demonstrated that a bolus of propofol (5 mg/kg) infused over 30 seconds was associated with a rapid drop in mean arterial pressure [27]. This reduction might be due to the vasodilatory effects of propofol by decreasing sympathetic tone on vasculature and systemic vascular resistance in a dose-dependent manner at induction [29-31]. This finding was in contrary to our study, where MAP remained stable after a slow propofol, either alone or in combination with ketamine. Slower propofol administration may cause slower onset vasodilation, permitting the body more time to compensate and maintain blood pressure. As in humans, titration of propofol to the desired clinical effect is preferred over bolus administration to mitigate changes in blood pressure and reduce the induction dose requirement [32].

Furthermore, the addition of ketamine improved MAP by mitigating the vasodilatory effects of propofol and reducing its dose. This observation was consistent with the findings of [31]**,** who found that a ketamine – propofol combination, administrated at 2 mg/kg/min of each, effectively preserved MAP after induction. According to this study, maintaining stable MAP was attributed to an increase in cardiac output, which resulted from increased HR caused by ketamine's sympathetic stimulation. These results align with the present study in which the administration of ketamine as co-induction agent (2 mg/kg IV) prior to slow propofol maintained stability in MAP post-induction.

A study implied that endotracheal intubation triggers an initial increase in heart rate and arterial blood pressure in humans within 30 seconds [33]. They observed a further increase around 60 seconds post-intubation. Based on these findings, we followed up any hemodynamic change after 1 min post-intubation in the current study.

Authors observed a significant reduction in SAP, DAP, MAP in dogs after intubation with slow propofol [24]. However, our study found a significant increase in these parameters. This difference can be attributed to sympathetic stimulation triggered by laryngoscopy and tracheal intubation [32]**.**

Moreover, a previous study reported that a lower dose of propofol (4.0 to 5.6 mg/kg) resulted in a sharp rise in arterial blood pressure post-induction, compared to higher doses of propofol (6.6 to 8.3 mg/kg) administrated prior to intubation [34]. This higher dose was associated with a non-significant increase in heart rate and blood pressure. This suggests that the lower propofol dose (3.56±0.44 mg/kg) may not be sufficient to adequately attenuate the hemodynamic response to intubation inhibiting sympathetic stimulation in the current study.

Here in, the ketamine- slow propofol combination resulted in better stability in HR and blood pressure post-intubation. This finding aligned with the results of [35] in humans**,** which reported that the administration ketamine (0.5 mg/kg IV) 1 min prior to anesthetic induction with

propofol attenuated arterial blood pressure changes postinduction and maintained hemodynamic stability compared to using propofol alone. In this context, a study reported that propofol-ketamine combination provided better hemodynamic stability to laryngoscopy and intubation compared to thiopental-ketamine combination and etomidate [36].

However, in the present study, ketamine was administrated 5 min prior to propofol to allow it sufficient time to exert its full effect. This time of administration helped inhibit hypertension post-intubation from ketamine's sympathomimetic properties. Meloxicam, a nonsteroidal anti-inflammatory drug (NSAID), was administrated preoperatively to provide preventive analgesia for minimizing postoperative pain associated with intubation. This approach aligns with findings of Wang *et al.* [37], which reported that NSAIDs are effective in reducing postoperative pharyngeal pain related to intubation. The limitations in our study, invasive blood pressure technique was not used, which shows blood pressure changes with each heartbeat.

Conclusion

Both slow propofol and the ketamine-slow propofol combination can blunt hemodynamic fluctuations post-induction. However, using ketamine as a co-induction agent at a dose 2 mg/kg IV prior to slow propofol (1 mg/kg/min) is more effective in attenuating hemodynamic changes in response to intubation compared to slow propofol alone. Therefore, ketamine- slow propofol is particularly beneficial for dogs with high risk of hypotension, such as those with preexisting cardiovascular disease or trauma patients and elderly canine patients to maintain hemodynamic stability during anesthetic induction and tracheal intubation.

Conflict of interest

Authors declares no conflict of interest.

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الملخص العربي

تأثيرات التخدير و التنبيب علي الإستجابة الديناميكية الدموية باستخدام البروبوفول ببطء أو الكيتامين مع البروبوفول ببطء فى الكلاب :دراسة تجريبية.

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تهدف هذه الدراسة إلي ملاحظة التغيرات الديناميكية الدموية عند بدء التخدير و التنبيب بإستخدام البروبوفول ببطء أوالكيتامين مع البروبوفول ببطء.حيث تم تقسيم ثمانية كلاب إلي مجموعتين كل منهما تحتوي علي 4 كلاب : وقد تم حقن المجموعة الأولي البروبوفول عن طريق الحقن الوريدي ببطء بمعدل 1 ملغم/كغم/دقيقة، وتم حقن المجموعة الثانية جرعة من الكيتامين بمعدل2ملغم/كغم بالوريد قبل حقن البروبوفول عن طريق الحقن الوريدي ببطء بمعدل 1 ملغم/كغم/دقيقة لإحداث التخدير.استمر حقن البربوفول حتي تحقيق الظروف المناسبة للتنبيب. وتم قياس معدل ضربات القلب و ضغط الدم بعد 30 دقيقة قبل بدء التخدير، وبعد دقيقة واحدة من أحداث التخدير والتنبيب. و لقد أظهرت النتائج ان جرعة البربوفول اللازمة لحدوث التخدير والتنبيب في المجموعة الثانية وهي (1.55±0.37 ملغم/كغم) أقل بشكل كبير من المجموعة الأولي (3.56±0.44 ملغم/كغم) مع قيمة p تساوي0.01. كما أظهرت المجموعة الثانية ارتفاع حاد في معدل ضربات القلب التي كانت 96.2 a2.72± مقارنة بالمجموعة الأولي والتي كانت قيمتها 60 c3.13± عند حدوث التخديرمع قيمة p تساوي اقل من 0.001 . بينما في كلا المجموعتين لم تظهرتغيرات معنوية في ضغط الدم الشرياني عند حدوث التخدير، حافظت المجموعة الثانية علي استقرار أكبر في ضغط الدم الشرياني استجابة للتنبيب. في الختام نستنتج أن كل من البربوفول ببطء أو مزيج الكيتامين مع البربوفول ببطء ذو فاعلية في تخفيف التقلبات الديناميكية الدموية بعد حدوث التخدير. ولكن مزيج الكيتامين مع البربوفول ببطء حقق أكثر استقرارا في ضغط الدم بعد التنبيب مقارنة بالبربوفول ببطء بمفرده.