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EFEITOS HEMODINÂMICOS DA LIDOCAÍNA INTRAVENOSA EM GATOS DOMÉSTICOS SUBMETIDOS À ANESTESIA TOTAL INTRAVENOSA

HEMODYNAMIC EFFECTS OF INTRAVENOUS LIDOCAINE IN DOMESTIC CATS UNDER TOTAL INTRAVENOUS ANESTHESIA

EFECTOS HEMODINÁMICOS DE LA LIDOCAÍNA INTRAVENOSA EN GATOS DOMÉSTICOS SOMETIDOS A ANESTESIA TOTAL INTRAVENOSA

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RESUMO: A administração intravenosa de lidocaína em gatos é limitada pelo potencial de toxicidade e depressão cardiovascular. Objetivou-se avaliar os efeitos hemodinâmicos e ecocardiográficos de duas velocidades de administração intravenosa de lidocaína em gatos hígidos sob anestesia total intravenosa. Dez gatos receberam gabapentina, acepromazina e butorfanol, foram induzidos com cetamina e propofol e mantidos com propofol e dexmedetomidina. Os animais foram distribuídos aleatoriamente em dois grupos (n = 5/grupo), recebendo bolus de lidocaína (2 mg/kg) durante dois (grupo A) ou cinco minutos (grupo B), seguido de infusão de 3 mg/kg/h. As avaliações ocorreram durante a infusão de propofol (MP), após dexmedetomidina (MD) e aos 5, 30 e 60 minutos após lidocaína (ML5, ML30 e ML60). Volume sistólico, débito cardíaco, índice cardíaco, pressão arterial média e distância de ejeção não apresentaram alterações significativas. No grupo A, o diâmetro interno ventricular esquerdo ao final da sístole aumentou e a fração de encurtamento diminuiu significativamente entre MD e ML30. Fração de ejeção e frequência cardíaca variaram durante a anestesia, sem diferenças significativas entre MD e os momentos posteriores à lidocaína. Não se observaram alterações hemodinâmicas clinicamente relevantes, embora os achados ecocardiográficos exijam interpretação considerando os efeitos dos anestésicos e as condições de carga ventricular.

Palavras-chave: Lidocaína intravenosa. Função sistólica ventricular esquerda. Variáveis hemodinâmicas. Débito cardíaco. Anestesia felina.

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ABSTRACT: Intravenous lidocaine administration in cats is limited by concerns regarding systemic toxicity and cardiovascular depression. This study aimed to evaluate the hemodynamic and echocardiographic effects of two intravenous lidocaine bolus administration rates in healthy cats undergoing total intravenous anesthesia. Ten cats were premedicated with gabapentin, acepromazine, and butorphanol, induced with ketamine and propofol, and maintained under anesthesia with propofol and dexmedetomidine. Cats were randomly allocated to two groups (n = 5 per group) and received lidocaine (2 mg/kg IV) over two (group A) or five minutes (group B), followed by a continuous infusion at 3 mg/kg/h. Hemodynamic and echocardiographic variables were assessed during propofol infusion (MP), after dexmedetomidine administration (MD), and at 5, 30, and 60 minutes after lidocaine administration (ML5, ML30, and ML60). Stroke volume, cardiac output, cardiac index, mean arterial pressure, and stroke distance did not change significantly. In group A, left ventricular internal diameter at end-systole increased significantly, whereas fractional shortening decreased significantly between MD and ML30. Ejection fraction and heart rate varied throughout anesthesia, without significant differences between MD and subsequent lidocaine assessment periods. Intravenous lidocaine was not associated with clinically relevant impairment of global hemodynamic performance; however, echocardiographic findings should be interpreted considering the combined cardiovascular effects of anesthetic agents and ventricular loading conditions.

Keywords: Intravenous lidocaine. Left ventricular systolic function. Hemodynamic variables. Cardiac output. Feline anesthesia.

RESUMEN: La administración intravenosa de lidocaína en gatos está limitada por el riesgo de toxicidad sistémica y depresión cardiovascular. El objetivo fue evaluar los efectos hemodinámicos y ecocardiográficos de dos velocidades de administración intravenosa de lidocaína en gatos sanos sometidos a anestesia total intravenosa. Diez gatos recibieron gabapentina, acepromazina y butorfanol como premedicación, fueron inducidos con ketamina y propofol y mantenidos bajo anestesia con propofol y dexmedetomidina. Los animales fueron distribuidos aleatoriamente en dos grupos (n = 5 por grupo) y recibieron un bolo intravenoso de lidocaína (2 mg/kg) durante dos (grupo A) o cinco minutos (grupo B), seguido de una infusión continua de 3 mg/kg/h. Las variables hemodinámicas y ecocardiográficas se evaluaron durante la infusión de propofol (MP), después de la administración de dexmedetomidina (MD) y a los 5, 30 y 60 minutos posteriores a la administración de lidocaína (ML5, ML30 y ML60). El volumen sistólico, el gasto cardíaco, el índice cardíaco, la presión arterial media y la distancia de eyección no presentaron cambios significativos. En el grupo A, el diámetro interno del ventrículo izquierdo al final de la sístole aumentó significativamente, mientras que la fracción de acortamiento disminuyó entre MD y ML30. La fracción de eyección y la frecuencia cardíaca variaron durante la anestesia, sin diferencias significativas entre MD y los períodos posteriores a la administración de lidocaína. La lidocaína intravenosa no se asoció con un deterioro hemodinámico global clínicamente relevante; sin embargo, los hallazgos ecocardiográficos deben interpretarse considerando los efectos cardiovasculares combinados de los anestésicos y las condiciones de carga ventricular.

Palabras clave: Lidocaína intravenosa. Función sistólica ventricular izquierda. Variables hemodinámicas. Gasto cardíaco. Anestesia felina.

1 INTRODUCTION

Feline anesthesia presents particular challenges owing to species-specific physiological and pharmacokinetic characteristics. In a large prospective study, anesthesia-related mortality was higher in cats than in dogs, with reported rates of 0.24% and 0.17%, respectively, highlighting the importance of appropriate anesthetic management and perioperative monitoring (BRODBELT DC, et al., 2007; BRODBELT DC, et al., 2008). Differences in drug metabolism, sensitivity to anesthetic agents, autonomic responses to stress, and a relatively high body surface area-to-mass ratio, which increases susceptibility to hypothermia, may further complicate anesthetic management in this species (COURT MH, 2013; GRIMM KA, et al., 2015; LITTLE S, 2015).

Balanced anesthetic techniques are commonly employed to optimize analgesia while minimizing the dose-dependent adverse effects of individual anesthetic agents. In this context, intravenous (IV) lidocaine has been investigated as an adjunct to multimodal analgesia, with evidence of perioperative analgesic effects and reduced inhalant anesthetic requirements in different species (PYPENDOP BH & ILKIW JE, 2005b; DOHERTY T, et al., 2007; CORRÊA JMX, et al., 2021). Nevertheless, its use in cats has historically been restricted because of concerns regarding systemic toxicity and cardiovascular depression (TILLEY LP & WEITZ J, 1977; PYPENDOP BH & ILKIW JE, 2005a). Previous experimental findings in isoflurane-anesthetized cats demonstrated that IV lidocaine administration was associated with reductions in cardiac index and stroke index, particularly at higher plasma concentrations (PYPENDOP BH & ILKIW JE, 2005a).

More recent clinical studies have reported that IV lidocaine, administered as an initial bolus of 1.5–2 mg/kg followed by a continuous infusion of 3 mg/kg/h, may provide perioperative analgesic benefits without clinically detectable signs of systemic toxicity in healthy cats undergoing surgery (CORRÊA JMX, et al., 2021; COMASSETTO F, et al., 2025). Additionally, a retrospective study evaluating lidocaine as part of a multimodal anesthetic protocol reported reduced isoflurane requirements without a significant association between lidocaine administration and hypotension or heart rate alterations (KIM M, et al., 2025). Despite these findings, the hemodynamic effects of IV lidocaine during total intravenous anesthesia (TIVA), particularly when combined with propofol and dexmedetomidine, remain insufficiently characterized. Furthermore, the influence of the initial lidocaine bolus administration rate on cardiovascular variables, including cardiac output (CO) and stroke volume (SV), has not been adequately investigated.

Therefore, the primary objective of this study was to evaluate the effects of IV lidocaine administration, consisting of a 2 mg/kg bolus followed by a continuous infusion at 3 mg/kg/h, on CO and SV in healthy cats undergoing TIVA with propofol and dexmedetomidine. The secondary objective was to determine whether bolus administration over two or five minutes influenced these cardiovascular variables.

2 MATERIALS AND METHODS

2.1 Animals and experimental design

Ten clinically healthy mixed-breed domestic cats scheduled for elective orchiectomy or ovariohysterectomy were enrolled in this study. Eligible cats were aged between 8 months and 8 years and classified as American Society of Anesthesiologists (ASA) physical status I or II based on physical examination, preoperative laboratory testing, and echocardiographic assessment.

An *a priori* sample size calculation was performed based on the primary outcome, cardiac output (CO), assuming a two-sided significance level of 5%, statistical power of 80%, an expected difference of 15%, and a standard deviation of 0.07 L/min. The calculated sample size was five cats per group. The experimental design considered the principles of replacement, reduction, and refinement (3Rs), aiming to minimize animal use while maintaining adequate statistical power for the primary outcome. Alternative methods were considered unsuitable for reproducing the integrated cardiovascular responses to anesthesia and drug administration. Refinement measures included standardized anesthetic and analgesic protocols, continuous perioperative monitoring, postoperative care, and predefined humane endpoints.

2.2 Anesthetic protocol

All cats received gabapentin (200 mg/cat, Drogavet®) orally 2 hours before intramuscular (IM) premedication with acepromazine (0.05 mg/kg, Acepran®, Vetnil) and butorphanol (0.4 mg/kg, Butorfin®, Vetnil). Following premedication, a 22-gauge intravenous catheter (Safalet®, Nipro) was placed in a cephalic vein. Anesthesia was induced with intravenous (IV) ketamine (2 mg/kg, Cetamin®, Syntec) and propofol (Fresenius Kabi) administered using a target-controlled infusion (TCI) system (CATTAI A, et al., 2016). The initial target plasma concentration of propofol was set at 7 μg/mL and subsequently increased in increments of approximately 1 μg/mL every minute until adequate anesthetic depth for

induction was achieved. Anesthetic induction was defined by the absence of the palpebral reflex and loss of jaw tone, assessed at 10-second intervals.

Following induction, cats were endotracheally intubated and connected to a circle breathing system delivering 100% oxygen. Volume-controlled mechanical ventilation was initiated with a tidal volume of 8 mL/kg, an inspiratory-to-expiratory ratio of 1:2, positive end-expiratory pressure of 3 cmH_2O, and respiratory rate of 15 breaths/min. Ventilator settings were adjusted to maintain a driving pressure of 7–8 cmH_2O and an end-tidal carbon dioxide partial pressure ($PE'CO_2$) of 35–45 mmHg (4.66–5.99 kPa).

Anesthesia was maintained with propofol TCI, and the predicted plasma concentration was adjusted at 5-minute intervals to maintain a consistent anesthetic depth throughout the experimental period. Anesthetic depth was assessed based on a centrally positioned eye, presence of the medial palpebral reflex, and absence of swallowing or tongue movements.

Thirty minutes after anesthetic induction, dexmedetomidine (1 μg/kg IV, Dexdomitor®, Zoetis) was administered as a bolus, followed by a continuous rate infusion (CRI) of 1 μg/kg/h, which was maintained throughout the experimental period.

2.3 Experimental groups and lidocaine administration

Sixty minutes after initiation of the dexmedetomidine infusion, corresponding to the baseline assessment (MD), cats were randomly allocated to two experimental groups (n = 5 per group). Both groups received an IV bolus of lidocaine (2 mg/kg, Xylestesin®, Cristália), followed immediately by a CRI of 3 mg/kg/h. In group A, the lidocaine bolus was administered over 2 minutes, whereas in group B, it was administered over 5 minutes. Data analysts were blinded to treatment allocation.

To preserve blinding and standardize the assessment schedule, a nominal bolus administration period of 5 minutes was considered for both groups, irrespective of the actual administration duration. Post-bolus assessments were standardized relative to initiation of the lidocaine CRI, with the first assessment performed 5 minutes after infusion onset. The lidocaine infusion was maintained for 60 minutes. Following completion of the experimental period, cats underwent their scheduled elective surgical procedures.

2.4 Hemodynamic monitoring and data collection

Hemodynamic and echocardiographic assessments were performed at five predefined time points: immediately after anesthetic induction, before dexmedetomidine administration

(MP); 60 minutes after initiation of the dexmedetomidine infusion, immediately before lidocaine administration (MD); and at 5 (ML5), 30 (ML30), and 60 minutes (ML60) after initiation of the lidocaine CRI.

Physiological variables were monitored at 10-minute intervals throughout the experimental period using a multiparameter monitor (Life Window Lite+®, Digicare). These included heart rate (HR), respiratory rate (fR), peripheral oxygen saturation (SpO_2), end-tidal carbon dioxide partial pressure ($P_E'CO_2$), rectal temperature, and noninvasive systolic, diastolic, and mean arterial pressures (MAP), measured using the oscillometric method. Stroke distance (StD) was assessed using an esophageal Doppler monitor (CardioQ_ODM+®, Deltex Medical).

2.5 Echocardiographic assessment

Echocardiographic examinations were performed at all predefined assessment time points (MP, MD, ML5, ML30, and ML60) using the right parasternal acoustic window. Measurements were obtained in triplicate and included left ventricular internal diameter at end-diastole (LVIDd) and end-systole (LVIDs).

Left ventricular end-diastolic volume (EDV) and end-systolic volume (ESV) were calculated and used to derive stroke volume (SV = EDV – ESV). Cardiac output was calculated as CO = (SV × HR)/1000, with SV expressed in milliliters and HR in beats/min, yielding CO in L/min. Cardiac index (CI) was calculated by dividing CO by body surface area (CI = CO/BSA). Left ventricular ejection fraction (EF) was determined using the Simpson method, whereas fractional shortening (FS) was calculated as FS = [(LVIDd – LVIDs)/LVIDd] × 100%.

2.6 Statistical analysis

Statistical analyses were performed using Statistical Analysis System software (SAS, version 9.3; SAS Institute Inc., Cary, NC, USA). Data normality was assessed using the Shapiro–Wilk test. Comparisons were performed within each group across the predefined assessment time points (MP, MD, ML5, ML30, and ML60) and between groups at corresponding time points.

The effects of treatment group, assessment time, and their interaction were evaluated, and multiple comparisons of means were performed using Tukey's test. Statistical significance was set at $p < 0.05$. Results are presented as mean ± standard deviation (SD), unless otherwise stated.

3 RESULTS

All ten cats completed the experimental protocol, and their demographic characteristics are summarized in Table 1. Predicted plasma propofol concentrations were 9.4 ± 0.7 μg/mL at MP, 4.1 ± 0.7 μg/mL at MD, 3.9 ± 0.5 μg/mL at ML5, 3.2 ± 0.4 μg/mL at ML30, and 2.7 ± 0.5 μg/mL at ML60. All monitored physiological variables remained within clinically acceptable ranges throughout the experimental period, and all cats recovered uneventfully.

Table 1 – Demographic characteristics of the ten cats included in the study

Demographic	Group A (2')	Group B (5')
Cats (n)	5	5
Body weight (kg)	3.5 ± 1.2	3.6 ± 0.8
Age (years)	1.5 ± 0.5	1.6 ± 1.3
Sex (F/M)	3F + 2M	3F + 2M
Body Condition Score (1-9)	5 (4 - 5)	5 (4 - 6)

Source: JULIA N, et. Al, 2026.

No significant differences were observed in left ventricular internal diameter at end-diastole (LVIDd), stroke volume (SV), cardiac output (CO), cardiac index (CI), mean arterial pressure (MAP), or stroke distance (StD) between the pre-lidocaine assessment (MD) and subsequent assessment time points (ML5, ML30, and ML60) within either group (Table 2; $p > 0.05$). Similarly, no significant differences were detected between groups at corresponding time points for these variables. For LVIDd and MAP, significant differences were restricted to comparisons between MP, before dexmedetomidine administration, and subsequent assessment time points, with no significant differences between MD and the post-lidocaine assessments.

Table 2 – Echocardiographic variables and hemodynamic parameters of healthy cats anesthetized with dexmedetomidine–propofol (1 μg kg-1 h-1 and 7 μg ml-1 TCI, respectively) in time: before dexmedetomidine (MP); before lidocaine (MD); 5 min after initiation of lidocaine infusion (ML5); 30 min after initiation of lidocaine infusion (ML30); and 60 min after initiation of lidocaine infusion (ML60)

	Group		Moment							Pr > F		
	A	B	MP	MD	ML5	ML30	ML60	Mean	SEM	Group	Moment	GxM
LVIDd (cm)	1,30a	1,54a	1,27b	1,48a	1,45a	1,46a	1,44a	1,425	0,035	0,097	<0,0001	0,336
SF (%)	38,87b	50,98a	52,15a	46,48b	42,61b	41,77c	41,62c	44,928	1,610	0,039	<0,0001	0,055
EF (%)	63,57a	68,23a	70,39a	66,12ab	68,33ab	61,94b	62,73b	65,907	1,720	0,530	0,011	0,940
SV (ml/kg)	0,43a	0,63a	0,50a	0,56a	0,57a	0,50a	0,52a	0,533	0,027	0,064	0,201	0,701
CO (l/min)	0,12a	0,17a	0,15a	0,15a	0,15a	0,13a	0,12a	0,146	0,011	0,263	0,085	0,746
CI (l/min/m²)	0,51a	0,71a	0,65a	0,65a	0,66a	0,54a	0,55a	0,615	0,036	0,156	0,115	0,760

	Group		Moment							Pr > F		
	A	B	MP	MD	ML5	ML30	ML60	Mean	SEM	Group	Moment	GxM
MAP (mmHg)	84,56a	87,28a	65,10b	84,00a	92,40a	93,70a	94,40a	85,920	2,275	0,640	<0,0001	0,685
SD (cm/bpm)	7,90a	8,94a	8,18a	7,95a	8,43a	8,67a	8,90a	8,426	0,474	0,558	0,914	0,297
HR (bpm)	79,36a	76,80a	93,20a	82,10ab	75,10b	70,40b	69,60b	78,080	2,631	0,768	0,001	0,135

Note: Pr > F – probability; SEM – standard error of the mean; different lower-case letters in the row differ at 5% by Tukey test ($p < 0.05$).
Source: JULIA N, et. Al, 2026.

For left ventricular internal diameter at end-systole (LVIDs), no significant overall effect of group was detected ($p = 0.760$). However, significant effects of time ($p < 0.0001$) and the group × time interaction ($p = 0.035$) were observed (Table 3). Within group A, LVIDs was significantly higher at ML30 than at MD ($p = 0.029$) and MP ($p < 0.0001$). Values at ML5 and ML60 were also significantly higher than those at MP ($p < 0.0001$ for both comparisons). In group B, LVIDs was significantly lower at MP than at MD ($p = 0.004$), ML5 ($p = 0.001$), ML30 ($p = 0.003$), and ML60 ($p = 0.0006$).

Table 3 – Difference of LVIDs in groups A (lidocaine bolus in 2 minutes) and B (lidocaine bolus in 5 minutes) in time: before dexmedetomidine (MP); before lidocaine (MD); 5 min after initiation of lidocaine infusion (ML5); 30 min after initiation of lidocaine infusion (ML30); and 60 min after initiation of lidocaine infusion (ML60)

LVIDs (cm)	Group			
Moment	A	B	Mean	SEM
MP	0.58Ca	0.63Ba	0.61	0.053
MD	0.79Ba	0.78Aa	0.79	0.050
ML5	0.87ABa	0.80Aa	0.83	0.071
ML30	0.91Aa	0.79Aa	0.85	0.072
ML60	0.85ABa	0.81Aa	0.83	0.055

Note: SEM – standard error of the mean; different letters in the row differ at 5% by Tukey test ($p < 0.05$).
Source: JULIA N, et. Al, 2026.

Fractional shortening (FS) was significantly affected by group ($p = 0.039$) and time ($p < 0.0001$), whereas the group × time interaction was not significant ($p = 0.055$). The estimated mean FS was significantly higher in group B (50.98%) than in group A (38.87%; $p = 0.039$). Comparisons between assessment time points revealed significantly higher FS at MD than at ML30 ($p = 0.046$). No other significant differences were identified following adjustment for multiple comparisons.

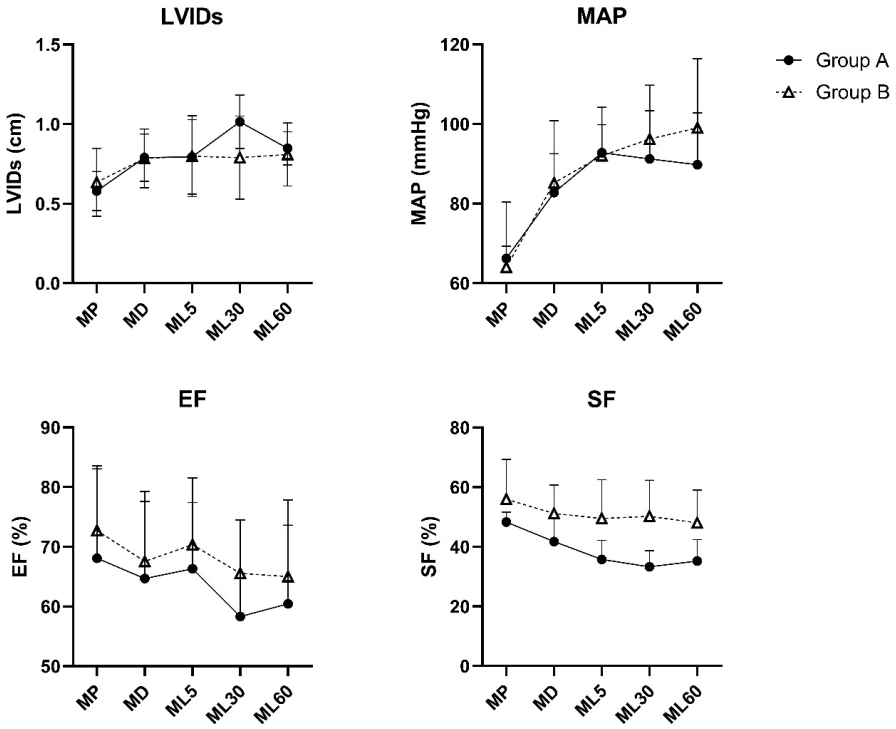
For left ventricular ejection fraction (EF), neither the effect of group ($p = 0.530$) nor the group × time interaction was significant. However, a significant effect of time was observed ($p = 0.011$). Ejection fraction, determined using the Simpson method, was significantly lower at

ML30 (p = 0.019) and ML60 (p = 0.040) than at MP. No other significant differences were observed between assessment time points.

Heart rate (HR) was not significantly affected by group (p = 0.768) or the group × time interaction (p = 0.135). However, a significant effect of time was detected (p < 0.001), with HR significantly higher at MP than at ML5 (p = 0.015), ML30 (p = 0.001), and ML60 (p < 0.001).

Changes in LVIDs, MAP, EF, and FS throughout the experimental period in both groups are illustrated in Figure 1.

Figure 1 – Changes in left ventricular internal diameter at end-systole (LVIDs), mean arterial pressure (MAP), ejection fraction (EF), and fractional shortening (SF) in cats undergoing total intravenous anesthesia with propofol and dexmedetomidine, followed by intravenous lidocaine administration. Group A (solid lines, filled circles) received a lidocaine bolus of 2 mg kg^{-1} over two minutes, whereas Group B (dashed lines, open triangles) received the same bolus over five minutes. Both groups subsequently received a continuous lidocaine infusion of 3 mg kg^{-1} h^{-1}. Measurements were obtained during propofol infusion alone (MP), during propofol and dexmedetomidine administration immediately before lidocaine administration (MD), and at ML5, ML30, and ML60 during lidocaine infusion. Data are presented as mean ± standard deviation (SD)



Source: JULIA N, et. Al, 2026.

4 DISCUSSION

Intravenous lidocaine administration was not associated with statistically significant changes in cardiac output (CO), stroke volume (SV), cardiac index (CI), stroke distance (StD), or mean arterial pressure (MAP), regardless of whether the initial bolus was administered over

two or five minutes. These findings suggest that lidocaine administration at the doses evaluated was not associated with detectable impairment of global hemodynamic performance under the experimental conditions. Similarly, a previous clinical study using a 2 mg/kg intravenous lidocaine bolus followed by a continuous infusion of 3 mg/kg/h in cats undergoing ovariohysterectomy reported no clinically detectable signs of lidocaine toxicity, although direct measurements of cardiac function were not performed (COMASSETTO F, et al., 2025).

The increase in left ventricular internal diameter at end-systole (LVIDs) observed at selected assessment time points following lidocaine administration may indicate changes in left ventricular systolic emptying. However, this parameter is influenced by myocardial contractility, preload, and afterload. Previous investigations involving dexmedetomidine-based sedation in cats demonstrated an approximately 31% increase in LVIDs, highlighting the influence of this drug on ventricular dimensions and systolic function (CASTRO ML, et al., 2022). Furthermore, propofol administered at induction rates of 2 and 8 mg/kg/min has been associated with changes in echocardiographic variables, including fractional shortening (FS), in healthy cats (MARANGONI S, et al., 2023). Therefore, the increase in LVIDs observed in the present study may reflect the combined cardiovascular effects of lidocaine, dexmedetomidine, and propofol, as well as changes in ventricular loading conditions, rather than a direct negative inotropic effect of lidocaine alone.

Nevertheless, the significant increase in LVIDs between MD and ML30 in group A deserves particular consideration, as dexmedetomidine was already being administered at MD. Although a significant group × time interaction was detected, no significant differences were observed between groups at individual assessment time points. These findings indicate differences in the temporal response of LVIDs but do not establish a causal relationship with lidocaine administration rate. Furthermore, the absence of a time-matched control group receiving propofol and dexmedetomidine without lidocaine prevents the effects of lidocaine from being distinguished from those associated with the concomitant anesthetic agents and the duration of anesthesia.

Changes in MAP and heart rate (HR) were particularly evident when comparing assessments performed before and after dexmedetomidine administration (MP and MD, respectively). The increase in MAP following dexmedetomidine administration is consistent with peripheral α_2-adrenoceptor-mediated vasoconstriction, which increases systemic vascular resistance and may induce reflex bradycardia. In addition, the central sympatholytic effects of dexmedetomidine contribute to reductions in HR (BLOOR BC, et al., 1992; PYPENDOP BH,

et al., 2011; CARVALHO ER, et al., 2019). In dogs anesthetized with sevoflurane, a continuous infusion of lidocaine at 6 mg/kg/h did not produce significant cardiovascular alterations. Conversely, dexmedetomidine administered at 2 μg/kg/h, either alone or in combination with lidocaine, resulted in reductions in HR and CO, accompanied by increases in MAP and systemic vascular resistance. The addition of lidocaine did not substantially modify the cardiovascular effects associated with dexmedetomidine (MORAN-MUÑOZ R, et al., 2017). Although these findings were obtained in dogs under inhalational anesthesia, they support the importance of considering the cardiovascular effects of α_2-adrenoceptor agonists when interpreting hemodynamic changes during multimodal anesthetic protocols.

Regarding left ventricular ejection fraction (EF), significant reductions were observed at ML30 and ML60 compared with MP, when cats had not yet received dexmedetomidine or lidocaine. However, no significant differences were detected between MD, immediately before lidocaine administration, and subsequent assessments during lidocaine infusion. Cardiovascular depression has previously been reported following intravenous lidocaine administration in isoflurane-anesthetized cats, with reductions in cardiac index and stroke index, particularly at target plasma concentrations of 7 μg/mL or higher (PYPENDOP BH & ILKIW JE, 2005a). In cougars (*Puma concolor*) undergoing total intravenous anesthesia, transient alterations in hemodynamic and echocardiographic variables were also observed following intravenous lidocaine administration, without clinically relevant cardiovascular complications (RIBEIRO RN, 2025). Nevertheless, differences in anesthetic protocols, species, and experimental conditions should be considered when comparing these findings with those of the present study.

In cats, lidocaine undergoes hepatic metabolism, producing the pharmacologically active metabolite monoethylglycinexylidide (MEGX). The pharmacokinetics of lidocaine and MEGX are influenced by anesthetic conditions, with isoflurane anesthesia associated with changes in lidocaine clearance and an increase in the time required to reach the maximum plasma concentration of MEGX (THOMASY SM, et al., 2005). The reduction in FS observed at ML30 in the present study suggests a temporal change in an echocardiographic index of left ventricular systolic function. However, FS is calculated from end-diastolic and end-systolic ventricular dimensions and is strongly influenced by ventricular loading conditions. An increase in afterload may reduce FS even in the absence of primary myocardial contractile dysfunction. Reductions in FS of approximately 28–30% have previously been observed following dexmedetomidine-based sedation in cats, supporting the importance of considering

the cardiovascular effects of this agent when interpreting changes in echocardiographic indices of systolic function (CASTRO ML, et al., 2022).

The significant difference in estimated mean FS between groups should also be interpreted cautiously. Group A received the lidocaine bolus over two minutes, whereas group B received it over five minutes. Although post-bolus assessments were standardized relative to the initiation of the continuous infusion, differences in bolus administration rates may have influenced the early plasma concentration profiles of lidocaine. Nevertheless, the group × time interaction for FS was not statistically significant, indicating that the observed differences do not provide sufficient evidence of distinct temporal responses between administration rates. Furthermore, FS is influenced by changes in ventricular loading conditions, including arterial pressure and systemic vascular resistance. Consequently, the observed differences may have been influenced by the cardiovascular effects of dexmedetomidine. The estimated mean FS values of approximately 39% and 51% were comparable to values reported in healthy cats, although reference values obtained under different physiological conditions should be interpreted cautiously (HÄGGSTRÖM J, et al., 2016). Importantly, these differences were not accompanied by statistically significant reductions in SV or CI.

Gabapentin was administered before anesthetic management to minimize fear- and stress-related responses during handling. Its use is consistent with feline-friendly handling recommendations aimed at reducing anxiety and facilitating veterinary procedures (RODAN I, et al., 2022). Previous investigations have demonstrated that oral gabapentin reduces fear-related aggressive behaviors and facilitates physical examination in cats (KRUSZKA M, et al., 2021). In the present study, gabapentin was administered to reduce stress associated with handling and potentially minimize fluctuations in sympathetic activity. However, gabapentin may also influence cardiovascular and echocardiographic measurements. Previous findings demonstrated modest reductions in selected echocardiographic indices of left ventricular systolic function following oral gabapentin administration in healthy cats, although the affected variables remained within established reference ranges (ALLEN ME, et al., 2021).

Body temperature decreased throughout the anesthetic period, which lasted approximately 2 hours and 45 minutes. Maintaining normothermia during prolonged anesthesia is particularly challenging in cats because of their relatively small body size and increased susceptibility to heat loss (GRIMM KA, et al., 2015; LITTLE S, 2015). To minimize heat loss, the animals were covered with blankets and provided with heated thermal packs.

Nevertheless, all cats required additional warming during anesthetic recovery until an appropriate body temperature was restored.

Another challenge encountered during the experimental period was related to the simultaneous use of multiple monitoring devices. Cats were positioned on a table adapted for echocardiographic examinations while receiving thermal support and being connected to a multiparameter monitor and an esophageal Doppler device (CardioQ). The esophageal Doppler device was developed for human use, and its application in smaller patients, such as cats, required careful positioning to obtain reliable measurements. Minor movements occasionally resulted in signal loss, requiring frequent repositioning of the probe. Furthermore, the device had to be removed during echocardiographic examinations to avoid interference with image acquisition. The relationship between transesophageal Doppler measurements and cardiac output obtained using reference methods has previously been investigated in anesthetized cats, highlighting methodological considerations regarding the interpretation of Doppler-derived measurements in this species (PYPENDOP BH, et al., 2024).

The present study has several limitations. Although an *a priori* sample size calculation was performed, the small sample size (n = 5 per group) may have limited the ability to detect differences between treatments. The absence of a control group without lidocaine and the concomitant administration of propofol and dexmedetomidine limit the attribution of cardiovascular changes specifically to lidocaine. Echocardiographic assessment before gabapentin administration could have provided additional baseline information, although handling unsedated cats might have increased stress. Additionally, behavioral or sedation scores were not assessed, arterial pressure was measured noninvasively, and predicted propofol concentrations decreased throughout anesthesia. Finally, the use of total intravenous anesthesia limits direct comparisons with previous studies conducted under inhalational anesthesia (PYPENDOP BH & ILKIW JE, 2005a; PYPENDOP BH & ILKIW JE, 2005b; KIM M, et al., 2025).

5 CONCLUSION

Intravenous lidocaine administration at 3 mg/kg/h, preceded by a 2 mg/kg bolus, was not associated with statistically significant changes in key hemodynamic parameters, including left ventricular internal diameter at end-diastole (LVIDd), stroke volume (SV), cardiac output (CO), cardiac index (CI), mean arterial pressure (MAP), and stroke distance (StD), in healthy cats undergoing total intravenous anesthesia with propofol and dexmedetomidine. No

significant differences in these parameters were observed between bolus administration durations of two and five minutes.

Nevertheless, changes in selected echocardiographic indices of left ventricular systolic function were observed, particularly at ML30. Fractional shortening decreased significantly between MD and ML30, whereas ejection fraction decreased at ML30 and ML60 compared with MP, but not with MD. These findings suggest that the observed cardiovascular changes may reflect the combined effects of the anesthetic agents and variations in ventricular loading conditions rather than the isolated effects of lidocaine. Given the small sample size and absence of a control group without lidocaine, these results should be interpreted cautiously.

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AUTHOR CONTRIBUTIONS

Conceptualization and study design: Ricardo Guilherme D'Otaviano de Castro Vilani and all co-authors. Methodology, investigation, project administration, data curation, data analysis, and writing – original draft: Júlia de Noronha. Anesthetic management and writing – original draft: Henrique Zem Chequin Sprengel and Júlia Milczewski Vilani. Echocardiographic examinations and data collection: Mariangela Endrighi. Statistical analysis: Vanessa Peripolli. Supervision and writing – review and editing: Ricardo Guilherme D'Otaviano de Castro Vilani. All authors contributed to the preparation of the manuscript, critically reviewed its content, and approved the final version for publication.

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INSTITUTIONAL REVIEW BOARD STATEMENT

The study was approved by the Animal Use Ethics Committee of the Agricultural Sciences Sector, Federal University of Paraná (UFPR), Brazil (protocol numbers 003/2025 and 031/2025).

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon request.

CONFLICTS OF INTEREST

The authors declare no financial or non-financial conflicts of interest.

USE OF GENERATIVE ARTIFICIAL INTELLIGENCE

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