DOI: 10.5433/1679-0359.2025v46n6p1999

Effect of intranasal detomidine in horses undergoing odontoplasty

Efeito da detomidina intranasal em cavalos submetidos à odontoplastia

Alline Morgana Silva Leite[1]*; Camille Alexandra Carvalho e Silva[2]; Ianna Lins Teodoro Napoleão[3]; Laura Cecília Bernardo Lima[4]; Ana Beatriz Oliveira Lara[4]; Cândice Mara Bertonha[5]; Joana Zafalon Ferreira[6]

Highlights

Intranasal detomidine (30 µg kg^{-1}) induces mild sedation in horses.
Sedation was insufficient for effective periodontal sensitivity blockade.
Delayed sedation peak (90 min) is crucial for clinical procedure planning.

Abstract

This study aimed to evaluate the effects of intranasal administration of detomidine at a dose of 30 µg kg^{-1} in horses undergoing odontoplasty. Thirteen healthy, crossbred horses were used, and the following parameters were measured: heart rate (HR), respiratory rate (*f*), systolic arterial pressure (SAP), rectal temperature (RT), head height relative to the ground (HH), blood glucose (BG), intestinal motility (GI motility), sedation score, and the presence of upper eyelid and lip ptosis. Measurements were taken at baseline (M0) and at 10, 30, 60, 90, and 120 min after intranasal administration. Hematocrit (Hct) and leukogram (LEU) were evaluated at M30, M60, M90, and M120. Periodontal sensitivity was assessed at M30 and during the odontoplasty procedure (upper, lower, and incisor teeth), as well as at its conclusion. Statistically significant differences ($p \leq 0.05$) were observed for HR, *f*, RT, Hct, WBC, HH, and GI motility. No significant changes were found in SAP or BG. All animals exhibited more pronounced sedation at M90. Periodontal sensitivity was detected in 69.23% of the animals, all of which responded to dental

[1] Master's student, Postgraduate Degree in Animal Bioscience, Faculdade de Zootecnia e Engenharia de Alimentos, Universidade de São Paulo, FZEA-USP, Pirassununga, SP, Brazil. E-mail: allinemorgana@usp.br
[2] Self-employed Veterinarian, Uberlândia, MG, Brazil. E-mail: camillecarvalhos.92@gmail.com
[3] Resident, Health Surveillance Program, Universidade de Brasília, UnB, Brasília, DF, Brazil. E-mail: iannanapoleao@gmail.com
[4] Undergraduate Students, Veterinary Medicine, Instituto Federal de Educação, Ciência e Tecnologia de Minas Gerais, IFMG – Campus Bambuí, Bambuí, MG, Brazil. E-mail: lauraceciliabernardo@gmail.com; laraanabeatriz23@gmail.com
[5] Profª. Drª., Instituto Federal de Educação, Ciência e Tecnologia de Minas Gerais, IFMG – Campus Bambuí, Bambuí, MG, Brazil. E-mail: candice.bertonha@ifmg.edu.br
[6] Profª. Drª., Universidade Federal de Juiz de Fora (UFJF), Juiz de Fora, MG, Brazil. E-mail: joana.zafalon@ufjf.br
* Author for correspondence

Received: Nov. 03, 2025 Approved: Fev. 02, 2026

floating of the lower incisors. Intranasal detomidine at a dose of 30 µg kg^{-1} promotes mild sedation in horses, with a late onset and without clinically relevant cardiovascular changes, but is insufficient to allow periodontal procedures when used alone.
Key words: Alpha-2 adrenergic agonists. Equines. Odontoplasty. Sedation.

Resumo

Objetivou-se avaliar os efeitos da detomidina na dose de 30 µg kg^{-1} pela via intranasal em equinos submetidos à odontoplastia. Em 13 equinos hígidos e mestiços, os parâmetros frequência cardíaca (FC), frequência respiratória (*f*), pressão arterial sistólica (PAS), temperatura retal (TR), glicemia (Glu), altura de cabeça em relação ao solo (AC), motilidade intestinal (MI), grau de sedação e presença de ptose labial e palpebral foram mensurados no momento basal (M0) e nos minutos 10, 30, 60, 90 e 120 após administração intranasal. O hematócrito (Hct) e leucograma (LEU) foram avaliados nos momentos M30, M60, M90 e M120. A sensibilidade periodontal foi avaliada em M30 e a sensibilidade dentária durante à odontoplastia (dentes superiores, inferiores e incisivos) e ao término do procedimento. Obteve-se diferença estatística ($p \leq 0,05$) entre os momentos avaliados, em comparação com os valores basais na FC, *f*, TR, Hct, LEU, AC, grau de sedação e MI. Não houve alteração significativa da PAS e BG. Todos os animais apresentaram sedação mais pronunciada em M90. A sensibilidade periodontal foi detectada em 69,23% dos animaise todos responderam ao desgaste dentário nos incisivos inferiores. A detomidina intranasal na dose de 30 µg kg^{-1} promove sedação leve em equinos, com início tardio e sem alterações cardiovasculares clinicamente relevantes, porém insuficiente para permitir a realização de procedimentos periodontais quando utilizada isoladamente.
Palavras-chave: Agonistas alfa-2 adrenérgicos. Equinos. Odontoplastia. Sedação.

Introduction

Equine veterinary dentistry is essential for maintaining health, enhancing athletic performance, and promoting quality of life. However, fewer than 1% of horses receive preventive or therapeutic dental care (Trigueiro et al., 2010). Dental procedures are commonly performed with the horse in a standing position to facilitate access to the oral cavity and to avoid physiological complications associated with recumbency (Dugdale et al., 2016).

In this context, sedation is commonly performed using detomidine, as it provides more effective analgesia and sedation compared to equipotent doses of xylazine (Lamont & Martinez, 2006). Nevertheless, adverse effects such as respiratory depression, bradycardia, ataxia, and prolonged sedation (Creighton & Lamont, 2024) require caution when selecting the dose and route of administration.

Intravenous (IV) and intramuscular (IM) routes of administration offer predictable effects but are associated with a higher incidence of adverse reactions and require more invasive restraint (Grimsrud et al., 2009). In this context, the intranasal (IN) route emerges as a less invasive alternative, with reduced hemodynamic impact, and is particularly useful in patients with difficult venous access (Weber et al., 2004). The high vascularization of the nasal mucosa favors rapid systemic absorption and may result in an onset of action similar to that

of the intravenous route (Fortuna et al., 2014). Intranasal administration of sedative drugs has demonstrated efficacy in other species, such as dogs (Santangelo et al., 2019), cats (Hommuang et al., 2022), birds (Adão et al., 2024), and swine (Rabelo et al., 2024), highlighting its potential for clinical sedation; therefore, intranasal administration of detomidine may be used for sedation of horses undergoing periodontal procedures.

Therefore, investigating the efficacy of intranasal detomidine administration in horses is relevant to expanding available therapeutic strategies and promoting less invasive and safer sedation during clinical and dental procedures. The dose of 30 μg kg^{-1} was selected based on previous studies that demonstrated the clinical sedative effects of detomidine administered intravenously and intramuscularly in horses (Mama et al., 2009). The choice of the intranasal route was based on its potential to expand therapeutic options and promote safer and more effective sedation during clinical and dental procedures.

Materials and Methods

The study was approved by the Animal Ethics Committee (Protocol 07/2020) of the Federal Institute of Education, Science and Technology of Minas Gerais – Bambuí campus, where the experiment was conducted. Thirteen clinically healthy horses (based on physical, hematological, and biochemical examinations) were used, including eight castrated males and five non-pregnant females, all crossbred, with a mean weight of 464.6 ± 85 kg and a mean age of 11.2 ± 7 years (mean ± SD).

After 8 h of feed fasting and 2 h of water fasting, the animals were restrained for jugular vein catheterization, which was performed to allow blood sample collection without further punctures and to ensure immediate venous access in case of emergency. After a 15-min acclimation period, baseline parameters were measured and recorded. Detomidine was administered intranasally at a dose of 30 μg kg^{-1}, diluted in 0.9% NaCl solution, with a standardized final volume of 2 mL, using a size 10 nasogastric tube for instillation of the drug into the ventral nasal meatus. After administration, the animals' heads were kept elevated using a dental headgear for approximately 1 min to promote absorption of the drug by the nasal mucosa, which was inferred based on the observation of clinical sedative effects throughout the experimental period. Evaluations were performed at 10 (M10), 30 (M30), 60 (M60), 90 (M90), and 120 (M120) min after administration.

At all time points, the following parameters were evaluated: heart rate (HR) by auscultation with a stethoscope; respiratory rate (f) by observation of thoracic movements; systolic arterial pressure (SAP) using a Doppler ultrasound device with cuff placement at the base of the tail (approximately 40% of tail circumference); rectal temperature (RT) using a digital thermometer; head height (HH) measured as the distance between the mandibular region and the ground; sedation score based on behavior and ataxia; presence of lip and eyelid ptosis; intestinal motility (GI motility) as described by Teixeira et al. (2004); blood glucose (BG) using a handheld glucometer; and hematocrit (Hct) and leukogram (WBC) measured at a private clinical laboratory, with the latter two not assessed at M10.

Periodontal sensitivity was assessed at M30 by a single evaluator through the insertion of a sterile, disposable 25 × 7 hypodermic needle into the gingival sulcus, parallel to the upper and lower incisor teeth. The animals' responses were classified as absent (0), mild (1), or present (2), as described by Roscoe (2007). Dental sensitivity was also evaluated by the same professional during the use of an electric rasp on the upper, lower, and incisor teeth, as well as at the end of the procedure, using the same scale: absent (0), mild (1), or present (2).

Statistical analyses were performed using GraphPad Prism 10 (GraphPad Software Inc., CA, USA). Data distribution symmetry was verified using the Shapiro-Wilk test. For the analysis of variables in relation to M0, one-way repeated-measures ANOVA followed by Sidak's multiple comparisons test was used for normally distributed data. Non-normally distributed data were analyzed using the Friedman test followed by Dunn's multiple comparisons test. A significance level of 5% was adopted for all analyses.

Results and Discussion

Heart rate showed a significant reduction from M60 onward ($p = 0.044$) compared to baseline (M0), with the greatest decrease observed at M90 ($p = 0.008$) (Table 1), although not indicative of bradycardia for the species (Mendes Netto, 2014). This finding may be related to both the dose used and the route of administration, as bradycardia results from the activation of presynaptic alpha-2 receptors in the central nervous system, leading to reduced norepinephrine release and consequent HR decrease, even at relatively low doses (Muir, 2009).

Table 1
Heart rate (HR); respiratory rate (*f*); systolic arterial pressure (SAP); rectal temperature (RT); blood glucose (BG); hematocrit (Hct); leukogram (WBC); head height relative to the ground (HH); and sedation score in horses treated with 30 µg kg^{-1} of intranasal detomidine. Parametric data are reported as mean ± standard deviation, and non-parametric data as median (interquartile range)

Variable	Time point (minutes)					
	M0	M10	M30	M60	M90	M120
HR (bpm)	36 ± 7	35± 5	31 ± 5	31 ± 6*	29 ± 5*	31 ± 6
f (mpm)	29 ± 8	30 ± 8	20 (16–40)	20 ± 6*	16 (12-24)*	15 ± 3*
SAP (mmHg)	124 ± 12	124 ± 13	119 ± 14	120 (110-160)	117 (103-158)	111 (100-160.0)
RT (ºC)	37.3 ± 0.4	37.3 ± 0.3	37.3 ± 0.4	37.4 (35.0-38.0)	37.1 (35.3-37.7)	37.0 (35.7-37.6)*
BG (mg dL^{-1})	81.0 ± 7.3	78.0 ± 6.7	84.0 ± 6.7	91.2 ± 10.8	88.0 (79.0-118.0)	92.2 ± 11.7
Hct (%)	32.7 ± 3.7	N/A	27.1 ± 2.6*	27.3 ± 2.3*	26.4 ± 2.3*	26.4 ± 2.3*
WBC (10 µL^{-3})	9.3 ± 1.8	N/A	4.9 ± 1.3*	4.7 ± 1.2*	4.2 (3.1-7.6)*	4.6 ± 1.1*
Degree of sedation	2 (1-2)	2 (1-3)	2 (2-3)	3 (2-4)*	3 (2-5)*	2.5 ± 0.8*

* Significant difference compared to baseline (M0) ($p \leq 0.05$).

A significant and progressive reduction in f was observed compared to baseline (M0), culminating in the lowest value at M120 ($p < 0.0001$) (Table 1), although not indicative of bradypnea for the species. Mama et al. (2009) did not report significant changes in gas exchange after sedation with detomidine at 30 μg kg^{-1} (IV and IM) in horses. However, arterial blood gas analysis is a more accurate tool to assess ventilation in animals, as demonstrated by Nyman et al. (2009), who observed elevated arterial carbon dioxide pressure in horses sedated with detomidine, even in the absence of changes in respiratory rate.

Alpha-2 adrenergic agonists are known to induce hemodynamic side effects, such as transient hypertension followed by hypotension (Medeiros et al., 2017). However, in the present study, SAP remained stable across time points (Table 1), which may be related to the route of administration. Studies in humans have shown that intranasal dexmedetomidine, even at doses equivalent to those administered intravenously, results in lower peak plasma concentrations (Yoo et al., 2015), which may attenuate abrupt hemodynamic effects. Moreover, intranasal administration allows for dual absorption pathways: systemic absorption through the nasal mucosa and direct access to the central nervous system via the olfactory and trigeminal nerves (Yoo et al., 2015; Crowe et al., 2018). This mechanism may result in a more gradual drug distribution, contributing to the hemodynamic stability observed.

A significant reduction in RT was observed at M120 ($p = 0.0376$) compared to M0 (Table 1), as also reported in another study involving horses treated with detomidine (Elfenbein et al., 2009). This variation may be attributed to the central redistribution of blood flow resulting from peripheral vasoconstriction induced by the drug (Creighton & Lamont, 2024), a pharmacological effect that is expected and well described in the literature.

Another well-established pharmacological effect of alpha-2 adrenergic agonists is the induction of hyperglycemia, due to suppression of insulin release (Fantoni & Cortopassi, 2010). In horses, BG concentrations above 110 mg dL^{-1} are considered indicative of hyperglycemia (Frank, 2011), and only two animals (15.3%) displayed values consistent with this condition, one at M90 and another at M120, both with 118 mg dL^{-1} (Table 1). This finding suggests a low incidence of the expected hyperglycemic effect; however, it should be noted that the fasting period imposed on the animals may have contributed to the attenuation of hyperglycemia by lowering baseline plasma glucose levels.

Both Hct and WBC values decreased significantly at all evaluated time points in comparison to baseline, with adjusted p-values ranging from 0.0001 to 0.0010 for Hct and from < 0.0001 to 0.0291 for WBC, respectively. The reduction in Hct can be mainly attributed to splenic vasodilation, which promotes sequestration of red blood cells by the spleen, and to fluid translocation into the intravascular space as a compensatory mechanism to maintain cardiac output (Luna et al., 2014). In parallel, leukopenia was observed, which may result from both splenic sequestration and suppression of lymphocyte proliferation (Bao et al., 2008).

A significant reduction in HH was observed from M30 onward (p = 0.0002), with the most pronounced decrease occurring at M90 ($p < 0.0001$) relative to baseline (Table 1). The occurrence of lip and eyelid ptosis was documented in 15.38%, 84.61%, 100%, 100%, and 61.53% of subjects at M10, M30, M60, M90, and M120, respectively.

Sedation in horses is characterized by head lowering, ataxia, and lip and eyelid ptosis (Valverde, 2010), with head height being one of the most relevant clinical parameters for sedation assessment (Kaukinen et al., 2011). A significant difference in sedation score was observed from M60 onward ($p = 0.0010$), with the highest intensity recorded at M90 ($p < 0.0001$) compared to baseline. These findings contrast with those described by Mama et al. (2009), who observed the greatest reduction in head height relative to the ground as early as 10 min following intravenous administration and at 60 min following intramuscular administration of detomidine at a dose of 30 μg kg^{-1} in horses. This pattern reinforces that, although intravenous administration of detomidine results in a faster and more predictable onset of sedation, the intranasal route presents slower absorption and a more gradual sedative effect, which explains the late onset of sedation observed in this study. On the other hand, this slower absorption rate may contribute to greater hemodynamic stability, making the intranasal route a less invasive alternative to the intravenous route in specific clinical situations.

GI motility scores decreased significantly from M60 ($p = 0.0012$) and remained reduced through M120 ($p < 0.0001$) (Figure 1). This effect is attributed to the action of detomidine on enteric neural pathways responsible for motility regulation (Creighton & Lamont, 2024), inhibiting propulsive motility by directly interfering with the cyclic activity of small intestinal electrical waves, thereby reducing intestinal transit rate (Zullian et al., 2011; Elfenbein et al., 2009). In addition, α2-adrenergic agonists reduce cardiac output, which activates presynaptic fibers and stimulates the vagal system, resulting in decreased splanchnic blood flow (Valverde, 2010). In the present study, ileocecal valve discharge scores did not return to baseline levels by M120, suggesting a persistent inhibitory effect on GI motility in this segment.

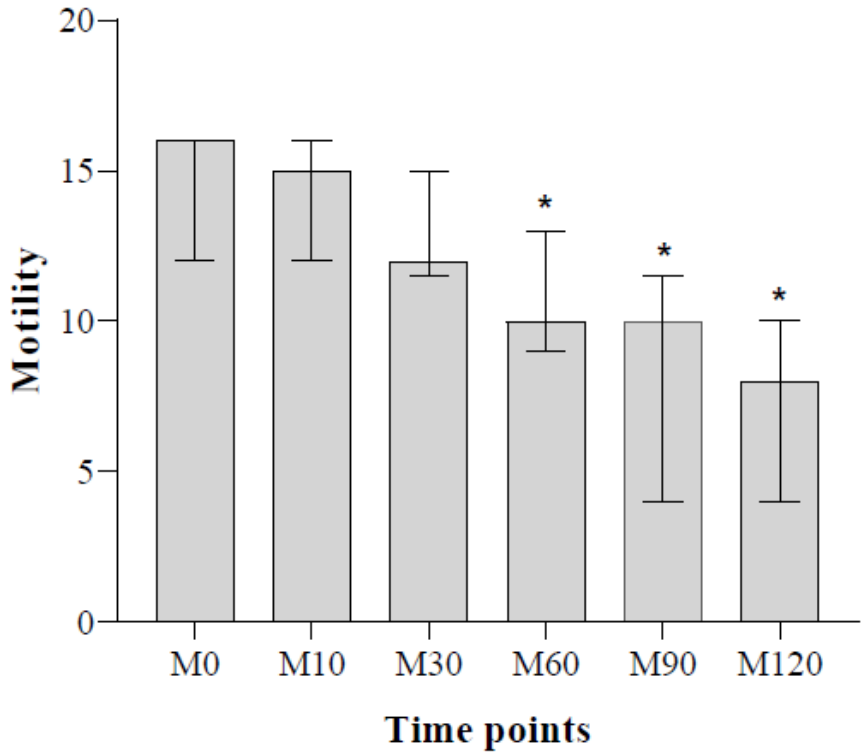

Figure 1. Variation in intestinal motility in horses treated with detomidine (30 µg kg^{-1}, intranasal route), expressed as median and interquartile range at the different time points analyzed
* Significant difference ($p < 0.05$) between each analysis time point and the baseline time point (M0).

Periodontal sensitivity was observed in 69.23% of the animals, rendering the procedure unfeasible in two cases. Among the animals that tolerated full (15.3%) or partial (69.2%) odontoplasty, sensitivity was noted primarily in the lower incisors. Although the procedure time was shorter than usual, sensitivity persisted at the end of the procedure in 76.9% of the cases. These findings indicate that intranasal detomidine, when used alone, provides limited analgesic and sedative effects for dental floating. In addition to pain associated with tooth grinding, other factors contribute to discomfort, including prolonged mouth opening with a speculum (an unnatural position for the species) and continuous manipulation of oral structures, all of which compromise sedation stability (Gozalo-Marcilla et al., 2019). Therefore, it is recommended to combine detomidine with adjuvant drugs, such as opioids (butorphanol or methadone), and the use of locoregional anesthesia techniques to ensure animal welfare and allow the procedure to be completed.

The main limitation of this study was the absence of a control group, which prevents direct comparisons between routes of administration. The evaluated variables were compared only to baseline values, which may limit the strength of

the conclusions. Additionally, the use of subjective sedation scores may introduce bias in the assessment, making it difficult to detect subtle differences over time. The lack of precise measurement of total sedation duration and the persistence of reduced motility also limited a more comprehensive analysis of the pharmacological effects.

Conclusion

Intranasal administration of detomidine at a dose of 30 μg kg^{-1} induces mild sedation in horses, with a delayed onset of action and an effect lasting up to 120 min after administration, without causing clinically relevant cardiovascular changes. However, when used alone, this approach is insufficient to provide adequate analgesia and immobilization for periodontal procedures, and the use of local anesthetic blocks and/or sedative or analgesic drugs should be considered to improve clinical efficacy and horse comfort during these treatments.

Acknowledgments

We thank the Federal Institute of Minas Gerais (IFMG – Bambuí campus) for institutional and financial support, including the undergraduate research fellowship and full funding of resources through Calls 14/2022 and 25/2022. We also extend our gratitude to the Equine-Assisted Therapy Unit staff for providing the animals used in this study.

References

Adão, F. M., Martins, I. D., Passos, Á. A. M. S. dos, Moraes, R. F. F. de, Balthazar, D. A., & Carvalho, E. B. de. (2024). Anesthetic effects of the ketamine and midazolam association by intranasal or intramuscular route in domestic chickens: prospective, blinded, randomized and crossover study. *Brazilian Journal of Veterinary Medicine, 46*(4), e005953. doi: 10.29374/2527-2179.bjvm005923

Bao, J. Y., Huang, Y., Wang, F., Peng, Y. P., & Qiu, Y. H. (2008). Expression of alpha-AR subtypes in T lymphocytes and role of the alpha-ARs in mediating modulation of T cell function. *Neuroimmunomodulation, 14*(6), 344-353. doi: 10.1159/000129670

Creighton, C. M., & Lamont, L. A. (2024). Sedatives and tranquilizers. In L. Lamont, W. W. Muir, S. M. Grimm, & D. R. Hodgson (Eds.), *Lumb & Jones' veterinary anaesthesia and analgesia* (6nd ed., pp. 333-354). Hoboken, NJ: Wiley-Blackwell.

Crowe, T. P., Greenlee, M. H. W., Kanthasamy, A. G., & Hsu, W. H. (2018). Mechanism of intranasal drug delivery directly to the brain. *Life Sciences, 195,* 44-52. doi: 10.1016/j.lfs.2017.12.025

Dugdale, A. H., Obhrai, J., & Cripps, P. J. (2016). Twenty years later: a single-centre, repeat retrospective analysis of equine perioperative mortality and investigation of recovery quality. *Veterinary Anaesthesia and Analgesia, 43*(2), 171-178. doi: 10.1111/vaa.12285

Elfenbein, J. R., Sanchez, L. C., Robertson, S. A., Cole, C. A., & Sams, R. (2009). Effect of detomidine on visceral and

somatic nociception and duodenal motility in conscious adult horses. *Veterinary Anaesthesia and Analgesia, 36*(2), 162-172. doi: 10.1111/j.1467-2995.2008.00441.x

Fantoni, D. T., & Cortopassi, S. R. G. (2010). Medicação pré-anestésica. In D. T. Fantoni, & S. R. G. Cortopassi (Eds.), *Anestesia em cães e gatos* (2a ed., cap. 13, pp. 217-227). São Paulo: Roca.

Fortuna, A., Alves, G., Serralheiro, A., Sousa, J., & Falcão, A. (2014). Intranasal delivery of systemic-acting drugs: small-molecules and biomacromolecules. *European Journal of Pharmaceutics and Biopharmaceutics, 88*(1), 8-27. doi: 10.1016/j.ejpb.2014.03.004

Frank, N. (2011). Equine metabolic syndrome. *Veterinary Clinics of North America: Equine Practice, 27*(1), 73-92. doi: 10.1016/j.cveq.2010.12.004

Gozalo-Marcilla, M., Luna, S. P. L., Gasthuys, F., Pollaris, E., Vlaminck, L., Martens, A., Haspeslagh, M., & Schauvliege, S. (2019). Clinical applicability of detomidine and methadone constant rate infusions for surgery in standing horses. *Veterinary Anaesthesia and Analgesia, 46*(3), 325-334. doi: 10.1016/j.vaa.2019.01.005

Grimsrud, K. N., Mama, K. R., Thomasy, S. M., & Stanley, S. D. (2009). Pharmacokinetics of detomidine and its metabolites following intravenous and intramuscular administration in horses. *Equine Veterinary Journal, 41*(4), 361-365. doi: 10.2746/042516409x370900

Hommuang, K., Sattasathuchana, P., & Thengchaisri, N. (2022). Effects of intranasal and intramuscular dexmedetomidine in cats receiving total intravenous propofol anesthesia. *Veterinary World, 15*(7), 1706-1713. doi: 10.14202/vetworld.2022.1706-1713

Kaukinen, H., Aspegrén, J., Hyyppa, S., Tamm, L., & Salonen, J. (2011). Bioavailability of detomidine administered sublingually to horses as an oromucosal gel. *Journal of Veterinary Pharmacology and Therapeutics, 34*(1), 76-81. doi: 10.1111/j.1365-2885.2010.01193.x

Lamont, L., & Martinez, E. A. (2006). Pharmacology of drugs used in equine anesthesia. In T. Doherty, & J. C. Valverde (Eds.), *Manual of equine anesthesia and analgesia* (Cap. 11, pp. 128-174). Oxford, UK: Blackwell Publishing.

Luna, S. P. L., Taffarel, M. O., Outeda, N. C., Oliveira, F. A. D., Matsubara, L. M., Albernaz, R. M., Carneiro, R., Rosa, S. C., & Rizzi, V. G. (2014). Avaliação da eficácia e parâmetros fisiológicos de equinos sedados com uma formulação à base de detomidina 1%. *Revista Brasileira de Medicina Equina, 10*(28), 28-30.

Mama, K. R., Grimsrud, K., Snell, T., & Stanley, S. (2009). Plasma concentrations, behavioural and physiological effects following intravenous and intramuscular detomidine in horses. *Equine Veterinary Journal, 41*(8), 772-777. doi: 10.2746/042516409x421624

Medeiros, L. Q., Luna, S. P. L., Castro, G. B. de, & de Oliveira, F. A. (2017). Sedative and cardiopulmonary effects of dexmedetomidine infusions randomly receiving, or not, butorphanol in standing horses. *Veterinary Record, 181*(15), 402. doi: 10.1136/vr.104359

Mendes Netto, D. M. (2014). Seção A - Semiologia do sistema circulatório de equinos e ruminantes. In F. L. F. Feitosa (Ed.), *Semiologia veterinária: a arte do diagnóstico* (3a ed., pp. 472-543). São Paulo: Roca.

Muir, W. W. (2009). Anxiolytics, nonopioid sedative-analgesics, and opioid analgesics. In W. W. Muir, & J. A. E. Hubbell (Eds.), *Equine anesthesia: monitoring and emergency therapy* (2a ed., pp. 185-209). St. Louis, Missouri: Elsevier.

Nyman, G., Marntell, S., Edner, A., Funkquist, P., & Morgan, K. (2009). Effect of sedation with detomidine and butorphanol on pulmonary gas exchange in the horse. *Acta Veterinaria Scandinavica, 51*(1), 1-9. doi: 10.1186/1751-0147-51-22

Rabelo, I. P., Luna, S. P. L., Rocha, T. L., & Minto, B. W. (2024). Intranasal vs. intramuscular administration of azaperone, midazolam and ketamine in pigs. *Frontiers in Veterinary Science, 11*, 1408103. doi: 10.3389/fvets.2024.1408103

Roscoe, M. P. (2007). *Evaluation of six sedation protocols for dental procedures in horses.* Dissertação de mestrado, Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brasil. https://repositorio.ufmg.br/handle/1843/BUOS-8TFHNG

Santangelo, B., Alampi, D., Navas, L., Breghi, G., & Briganti, A. (2019). Intranasal dexmedetomidine in healthy beagles: an echocardiographic and pharmacokinetic/pharmacodynamic study. *The Veterinary Journal, 251*, 105346. doi: 10.1016/j.tvjl.2019.105346

Teixeira, F. J., Neto, Natalini, C. C., & Driessen, B. (2004). Effects of glycopyrrolate on cardiorespiratory function in horses anesthetized with halothane and xylazine. *American Journal of Veterinary Research, 65*(4), 456-463. doi: 10.2460/ajvr.2004.65.456

Trigueiro, P. H. C., Urbano, S. A., Lima, S. M., & Costa, I. C. C. (2010). Alterações morfodentárias que influenciam a saúde dos equinos. *Revista Verde de Agroecologia e Desenvolvimento Sustentável, 5*(4), 1-10.

Valverde, A. (2010). Alpha-2 agonists as pain therapy in horses. *Veterinary Clinics: Equine Practice, 26*(3), 515-532. doi: 10.1016/j.cveq.2010.07.003

Weber, F., Wulf, H., & El Saeidi, G. (2004). S-ketamine and s-norketamine plasma concentrations after nasal and i.v. administration in anesthetized children. *Pediatric Anesthesia, 14*(12), 983-988. doi: 10.1111/j.1460-9592.2004.01358.x

Yoo, H., Chung, S. J., & Lee, H. J. (2015). Mechanism-based population pharmacokinetic and pharmacodynamic modeling of intravenous and intranasal dexmedetomidine in healthy subjects. *European Journal of Clinical Pharmacology, 71*(10), 1197-1207. doi: 10.1007/s00228-015-1913-0

Zullian, C., Menozzi, A., Pozzoli, C., Poli, E., & Bertini, S. (2011). Effects of α2-adrenergic drugs on small intestinal motility in the horse: an *in vitro* study. *Veterinary Journal, 187*(3), 342-346. doi: 10.1016/j.tvjl.2009.12.015