Acta Scientiae Veterinariae, 2026. 54(Suppl 1): 1106.

CASE REPORT
Pub. 1106

ISSN 1679-9216

# Use of Detomidine Alone or Associated with Yohimbine in Horses: Cardiorespiratory Evaluation

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## ABSTRACT

***Background*:** Equine dentistry, an important veterinary field, saw significant advancements in the 1970s and 80s, allowing safer standing procedures with improved tools, techniques, and medications. The anesthetic protocol typically uses an α2-adrenergic agonist sedative like detomidine, which provides mild analgesia and sedation while keeping the horse standing, thus avoiding risks linked to general anesthesia. However, detomidine can alter physiological parameters during procedures. Yohimbine, an α2-adrenergic antagonist, functions as a reversing agent for α2-adrenergic agonists, reducing both their sedative effects and associated adverse outcomes.

***Cases*:** The study evaluated 24 horses (12 males & 12 females) from Viamão city and the southern zone of Porto Alegre, Brazil, weighing between 340 and 580 kg, undergoing elective or therapeutic dental procedures including routine odontoplasty without extractions or local blocks. The animals fasted for 6 h before the procedure and were advised to consume only fibrous foods post-treatment to avoid gastrointestinal issues. Detomidine [0.02 mg/kg IV], an α2-adrenergic agonist inducing sedation, analgesia, and muscle relaxation, was administered to all horses, and yohimbine [0.1 mg/kg IV], an α2-adrenergic antagonist, to half of them, with evaluations of vital signs (heart rate, respiratory rate, body temperature) at baseline, 20, and 50 min. Results showed that heart rate was the only parameter with statistically significant changes, while other parameters remained stable. Environmental factors, like exposure to open, unshaded areas, influenced the animals' physiological parameters, potentially affecting results due to the limited sample size. The limited sample size was noted as a constraint, as random effects could disproportionately affect results. Statistical analysis used a two-tailed test with an alpha of 0.05 and 11 degrees of freedom, reflecting the sample size per group. These findings highlight the importance of controlled conditions and adequate sample sizes for future studies.

***Discussion*:** This study confirms that detomidine is a safe sedative for cardiorespiratory evaluation in equine dental procedures, provided that vital signs are monitored before, during, and after its use. The benefits of combining detomidine with an α2-adrenergic antagonist like yohimbine, which effectively reduces adverse effects, such as gastrointestinal hypomotility and bradycardia. Detomidine acts by binding to α2-adrenergic receptors, decreasing catecholamine release and central nervous system excitation. This mechanism explains the physiological responses, including decreased heart rate (HR), respiratory rate (RR), and motor activity. The study observed significant heart rate changes in both groups at 20 min post-detomidine administration, with further changes in the yohimbine group at 50 min due to the antagonist's rapid reversal of sedation and bradycardia. While detomidine affects thermoregulation, no body temperature changes were noted, a known side effect, underscores the benefit of using an antagonist to mitigate such risks. Detomidine, with an action lasting 60-120 min, is safe at low doses (0.002 - 0.02 mg/kg IV or IM). Gastrointestinal effects, including mild hypomotility, were noted but did not worsen post-procedure. The results indicate that while detomidine is generally safe for equine dental procedures, proper preparation and continuous monitoring of vital parameters are essential. Additionally, yohimbine demonstrates significant benefits as a reversing agent by mitigating adverse effects such as bradycardia and gastrointestinal hypomotility.

**Keywords:** equine, α2-agonist, reverser, sedative, equine dentistry.

DOI: 10.22456/1679-9216.150445

Received: 22 December2025 Accepted: 12 June 2026 Published: 7 July 2026

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## INTRODUCTION

Equine dentistry is a significant field in veterinary medicine [8]. The 1970s and 1980s brought considerable advancements in instrumentation, methodology, and pharmacological agents, enabling safer dental procedures to be performed on horses in a standing position [8,9]. The anesthetic protocols for these procedures typically involve α2-adrenergic agonists, which provide mild analgesia, sedation, and muscle relaxation while maintaining the animal standing [5,12]. General anesthesia, in contrast, is associated with complications such as trauma, hypotension, hypoventilation, edema, nerve paralysis, and colic [1]. Standing sedation therefore eliminates many of these risks, in addition to being more economical and faster [7], making it the preferred approach for equine dental procedures.

Detomidine, an α2-adrenergic agonist, has been widely used as an analgesic and sedative agent in equine practice due to its efficacy and compatibility with other drugs [13]. However, despite its advantages, detomidine can also induce changes in physiological parameters during procedures [5].

Yohimbine, an α2-adrenergic antagonist, reverses the sedative effects of α2-adrenergic agonists. It has been used in cases of intoxication or prolonged sedation caused by xylazine, detomidine, amitraz, and romifidine, effectively mitigating both sedative and adverse physiological effects [2].

This study aimed to evaluate the main cardiorespiratory changes caused by detomidine during and after equine dental procedures and to assess the effectiveness of yohimbine as a reversal agent.

## CASES

The study evaluated 24 horses (12 males & 12 females) originating from Viamão city and the southern region of Porto Alegre, Rio Grande do Sul, Brazil. All animals were healthy and scheduled for elective or therapeutic dental procedures, with body weights ranging from 340 to 580 kg. Horses underwent a 6 h solid food fast prior to treatment, and after the procedure, they were fed only fiber, avoiding concentrated feeds to reduce the risk of gastrointestinal complications.

Before treatment, all horses were weighed and received detomidine[1] [Dettovet® - 0.02 mg/kg, IV], an α2-adrenergic agonist with sedative, analgesic, and muscle relaxant properties. Within 5 min, a satisfactory level of sedation with visible relaxation was observed (Figure 1). In 12 horses (50%), yohimbine[2] [Reset® - 0.1 mg/kg, IV], an α2-adrenergic antagonist, was subsequently administered.

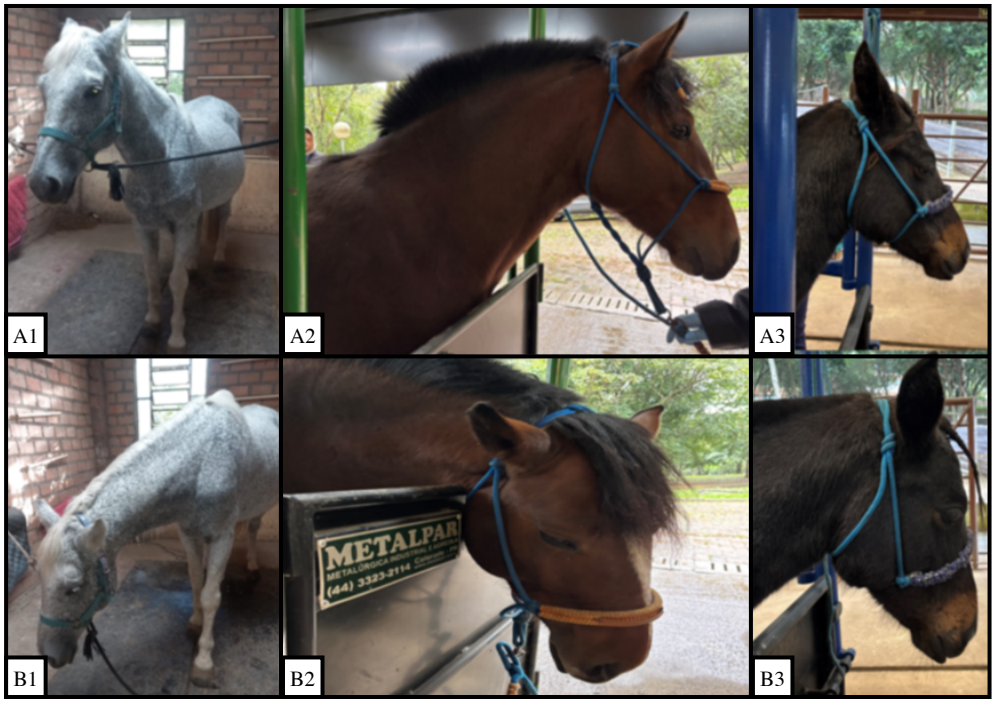


**Figure 1.** A1, A2 & A3- Alert horses without the use of medication. B1, B2 & B3- Horses 5 min after the use of the sedative detomidine [0.02 mg/kg, IV].

**Table 1.** Variables evaluated in 24 equines studied: the effects of detomidine and yohimbine, at moments 0, 20 and 50 min.

| # | Weight (kg) | Yohimbine | Temp (°C) | HR | RR | Temp (°C) | HR | RR | Temp (°C) | HR | RR |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 0 min | | | 20 min | | | 50 min | | |
| 1 | 390 | Yes | 37.00 | 48 | 24 | 38.40 | 28 | 20 | 36.70 | 40 | 24 |
| 2 | 480 | Yes | 37.00 | 28 | 20 | 38.40 | 20 | 28 | 37.60 | 24 | 24 |
| 3 | 480 | Yes | 37.80 | 44 | 16 | 37.70 | 28 | 20 | 37.50 | 28 | 16 |
| 4 | 420 | Yes | 37.90 | 48 | 32 | 38.00 | 28 | 24 | 38.10 | 36 | 24 |
| 5 | 380 | Yes | 38.30 | 56 | 40 | 37.70 | 28 | 22 | 37.70 | 40 | 20 |
| 6 | 435 | Yes | 38.60 | 52 | 44 | 38.30 | 40 | 20 | 38.10 | 48 | 20 |
| 7 | 420 | Yes | 38.90 | 56 | 24 | 36.90 | 28 | 20 | 38.20 | 36 | 20 |
| 8 | 370 | Yes | 36.40 | 40 | 16 | 38.20 | 32 | 16 | 37.40 | 32 | 16 |
| 9 | 490 | Yes | 37.60 | 36 | 34 | 37.70 | 32 | 16 | 37.30 | 36 | 16 |
| 10 | 460 | Yes | 37.80 | 32 | 16 | 37.40 | 36 | 28 | 37.70 | 36 | 16 |
| 11 | 340 | Yes | 36.70 | 40 | 16 | 36.00 | 28 | 16 | 35.00 | 44 | 20 |
| 12 | 485 | Yes | 36.90 | 44 | 20 | 36.90 | 32 | 16 | 36.70 | 36 | 20 |
| 13 | 435 | No | 38.50 | 36 | 32 | 39.00 | 28 | 20 | 37.70 | 36 | 20 |
| 14 | 413 | No | 38.30 | 40 | 35 | 37.80 | 32 | 24 | 38.80 | 28 | 20 |
| 15 | 460 | No | 37.30 | 40 | 16 | 38.00 | 32 | 20 | 38.20 | 28 | 24 |
| 16 | 490 | No | 37.80 | 28 | 20 | 38.20 | 24 | 20 | 37.80 | 20 | 20 |
| 17 | 405 | No | 38.30 | 32 | 20 | 38.30 | 28 | 20 | 37.70 | 32 | 20 |
| 18 | 345 | No | 37.30 | 44 | 20 | 37.40 | 36 | 16 | 37.20 | 39 | 16 |
| 19 | 390 | No | 37.80 | 38 | 24 | 37.20 | 44 | 20 | 37.20 | 51 | 16 |
| 20 | 500 | No | 37.30 | 32 | 28 | 37.60 | 28 | 16 | 37.40 | 28 | 20 |
| 21 | 510 | No | 37.30 | 56 | 40 | 38.10 | 36 | 20 | 38.00 | 32 | 20 |
| 22 | 410 | No | 36.90 | 52 | 24 | 37.00 | 28 | 24 | 36.20 | 28 | 20 |
| 23 | 580 | No | 36.90 | 32 | 24 | 37.40 | 28 | 16 | 37.40 | 32 | 16 |
| 24 | 570 | No | 3670 | 36 | 16 | 36.80 | 28 | 16 | 37.30 | 28 | 16 |

Physiological parameters, including heart rate (HR), respiratory rate (RR), and body temperature (BT), were measured before, during, and after detomidine administration (Table 1). All animals underwent routine odontoplasty without extractions or local nerve blocks. Evaluations were conducted at 3 time points: Time 0 (baseline, before drug administration), Time 20 (20 min after detomidine and yohimbine administration), and Time 50 (50 min after detomidine and 30 min after yohimbine). Gastrointestinal motility was also assessed by auscultation at the ileocecal valve on the right flank. A transient reduction in bowel sounds was noted after detomidine administration, but recovery occurred more rapidly in horses that received yohimbine.

Considerable variation was observed in the collected data. A major limitation of the study was the difficulty in maintaining uniform management conditions prior to treatment. For instance, animals were often kept outdoors without shade, which directly affected physiological parameters such as body temperature. In

**Table 2.** Statistical result of 24 horses under the effects of detomidine alone or associated with yoimbine.

| | ΔT 20 | ΔT 50 | ΔHR 20 | ΔHR 50 | ΔRR 20 | ΔRR 50 |
|---|---|---|---|---|---|---|
| Mean with yohimbine | 0.06 | -0.30 | -13.67 | 6.33 | -4.67 | -0.83 |
| Mean without yohimbine | 0.20 | -0.16 | -7.83 | 0.83 | -5.58 | -0.33 |
| Std. Dev. With yohimbine | 1.06 | 0.75 | 9.41 | 5.25 | 10.87 | 4.47 |
| Std. Dev. Without yohimbine | 0.43 | 0.60 | 7.74 | 4.37 | 7.05 | 2.67 |
| T-value with yohimbine | 0.19 | -1.39 | -5.03 | 4.18 | -1.49 | -0.65 |
| T-value without yohimbine | 1.61 | -0.92 | -3.50 | 0.66 | -2.74 | -0.43 |

Note: Critical *t*-value used = 2.201.

larger samples, random effects are expected to balance out, but in this study of 24 horses, random variation may have influenced the results.

Based on the parameters obtained (Table 2), HR was the only variable to show statistically significant differences. Other parameters did not present significant changes. The critical *t*-value was calculated using a two-tailed paired *t*-test with $\alpha = 0.05$ and 11 degrees of freedom, determined by the sample size of 12 per group.

## DISCUSSION

Based on the results of this study, detomidine is a safe agent for sedation in equine dental procedures, with minimal cardiorespiratory effects when used at recommended doses. Nevertheless, careful preparation of the animal and monitoring of vital parameters before, during, and after the procedure are essential. The use of an α2-adrenergic antagonist was shown to be beneficial in reducing adverse effects such as gastrointestinal hypomotility and bradycardia.

The α2-adrenergic agonists are widely used in equine pre-anesthetic protocols [5]. They bind to α2-receptors, inhibiting adenylate cyclase activity via Gi proteins and reducing intracellular cyclic adenosine monophosphate (cAMP) [17]. This leads to decreased sympathetic outflow, reduced release of catecholamines such as norepinephrine, and central nervous system depression.

The principal physiological effects of detomidine include bradycardia, reduced respiratory rate (RR), and decreased motor activity [10]. In this study, a transient change in RR was observed at Time 20; however, no corresponding change occurred in the yohimbine group, suggesting random variation. Heart rate (HR) showed the most significant alterations, with both groups presenting changes at Time 20, while the yohimbine group also exhibited changes at Time 50. These later changes are consistent with the rapid reversal of detomidine's effects by intravenous yohimbine, particularly the attenuation of sedation and bradycardia [4]. Although α2-agonists are known to affect thermoregulation [14], no significant alterations in body temperature (BT) were observed, likely due to environmental influences on this parameter.

Gastrointestinal effects are clinically relevant, as α2-agonists reduce motility for several hours after administration [16]. Detomidine has a longer duration of action than xylazine [approximately 60-120 min, depending on the dose], but when administered at low doses [0.002 - 0.02 mg/kg IV or IM], it remains a safe option [11,15]. In this study, mild hypomotility was noted on abdominal auscultation at Time 50, but no animal showed clinical deterioration after the procedure.

Yohimbine, administered at 0.1 mg/kg IV as recommended in the package insert [18], was effective in mitigating detomidine-induced hypomotility and bradycardia. These findings suggest that α2-adrenergic antagonists may represent a valuable strategy to enhance the safety and tolerability of detomidine sedation in equine dental practice.

These findings confirm that detomidine is a safe sedative for equine dental procedures, with minimal impact on cardiovascular and respiratory systems.

MANUFACTURERS

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***Declaration of interest.*** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of paper.

## REFERENCES


1 **Almeida M.R. & Almeida R.M. 2008.** Complicações anestésicas em equinos – revisão de literatura. *PUBVET.* 2(27): 1-32.

2 **Andrade S.F. 2004.** Estudo clínico e laboratorial da intoxicação experimental por amitraz em gatos e comparação entre ioimbina e atipamezole como tratamento. 175p. Tese (Doutorado em Medicina Veterinária). Universidade Estadual Paulista, São Paulo.

3 **Bednarski R.M. 2017.** Cães e Gatos. In: Tranquilli W.J., Thurmon J.C. & Grimm K.A. (Eds). *Lumb & Jones' Veterinary Anesthesia and Analgesia.* 5.ed. São Paulo: Roca, pp.2387-2412.

4 **Brunton L.L. 2007.** *Goodman & Gilman's Manual of Pharmacology and Therapeutics.* New York: McGraw-Hill, pp.148-187.

5 **Carter J.E. 2015.** Anesthesia and sedation in thefield. In: Cole C. (Ed). *Equine Pharmacology.* Hoboken: John Wiley & Sons, pp.44-62.

6 **Costa F.A. 2010.** Avaliação comparativa entre as associações de tiletamina e zolazepam a romifidina ou detomidina, em doses calculadas por extrapolação alométrica, para anestesia de campo em cavalos (*Equus caballus*). *A Hora Veterinária*. 29(174): 34-40.

7 **Doherty T. & Schumacher J. 2011.** Dental restraint and anesthesia. In: Easley J. (Ed). *Equine Dentistry.* 3rd edn. Amsterdam: Elsevier, pp.241-244. DOI: 10.1016/B978-0-7020-2980-6.00015-5

8 **Easley J., Baker G.J. & Easley J. 2005.** Dental and oral examination. In: Baker G.J. & Easley J. (Eds). *Equine Dentistry.* 2nd edn. London: Saunders, pp.151-169. DOI: 10.1016/B0-70-202724-3/50016-5

9 **Easley J. & Hatzel J. 2011.** The history of equine dentistry. In: Easley J. (Ed). *Equine Dentistry.* 3rd edn. Amsterdam: Elsevier, pp.11-25. DOI: 10.1016/B978-0-7020-2980-6.00002-7

10 **Fernandes V. 2016.** Utilização da associação de cetamina, diazepam e detomidina na contenção farmacológica de equídeos (*Equus* sp.) para procedimentos de orquiectomia em campo. *Arquivos de Ciências Veterinárias e Zoologia da UNIPAR*. 19(1): 1-5. DOI:10.25110/arqvet.v19i1.2016.5793

11 **Forney B.D. 2007.** *Understanding Equine Medications: Your Guide to Horse Health Care and Management.* Lexington: Eclipse Press, pp.96-99.

12 **Hubbell J.A.E. 2017.** Equinos. In: Tranquilli W.J., Thurmon J.C. & Grimm K.A. (Eds). *Lumb & Jones' Veterinary Anesthesia and Analgesia.* 4.ed. São Paulo: Roca. pp.585-597.

13 **Lamont L.A. & Martinez E.A. 2006.** Pharmacology of drugs used in equine anesthesia. In: Doherty T. & Valverde A. (Eds). *Manual of Equine Anesthesia and Analgesia.* Oxford: Blackwell Publishing, pp.128-174. DOI: 10.1002/9780470753248

14 **Madden C.J., Tupone D., Cano G. & Morrison S.F. 2013.** α2-adrenergic receptor-mediated inhibition of thermogenesis. *Journal of Neuroscience.* 33: 2017-2028. DOI: 10.1523/JNEUROSCI.4701-12.2013

15 **Mair T.S.2017.** Medical managementof gastrointestinal diseases. In: Blikslager A.T. (Ed). *The Equine Acute Abdomen.* 3rd edn. Wiley Blackwell, pp.313-330. DOI: 10.1002/9781119063254.ch27

16 **Mair T.S. 2002.** Medical therapies for colic. In: Mair T., Divers T. & Ducharme N. (Eds). *Manual of Equine Gastroenterology.* Philadelphia: W.B. Saunders, pp.119-125.

17 **McMurphy R.M. Davis E.G. Rankin A.J. Frese D.A. Lutjemeier B.J & Kenney M.J. 2018.** Adrenergic receptor agonists and antagonists. In: Riviere J.E. & Papich M.G. (Eds). *Veterinary Pharmacology and Therapeutics*. 10th edn. Hoboken: John Wiley & Sons, pp.131-150.

18 **Reset®.** Botucatu: Botupharma Indústria e Comércio de Produtos Veterinários Ltda. Available at: https://botupharma.com/download/01-BULA-RESET.pdf