Article

Evaluation of acepromazine-induced hemodynamic alterations and reversal with norepinephrine infusion in standing horses

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Abstract — The effects of norepinephrine (NOR) infusion on hemodynamic alterations induced with sedative doses of acepromazine (ACP) were evaluated. Infusion of NOR at 1 μg/kg body weight (BW)/minute for 15 min was administered to 5 standing horses 45 min (T_{45}) after intravenous injection of ACP at 0.1 mg/kg BW. Systolic arterial blood pressure (SAP) and hemodynamic parameters were evaluated on the median artery. Parameters were evaluated every 5 min from 45 to 65 min (T_{65}) at 75 (T_{75}), 90 (T_{90}), and 105 (T_{105}) minutes after ACP administration, and the vessel's surface (SURF), diameter (DIAM), circumference (CIRC), peak systolic velocity (PSV), end diastolic velocity (EDV), mean velocity (MV), volumetric flow (VF) and resistivity index (RI) of the flow were calculated. Acepromazine induced hypotension and vasodilation with a significant rise in SURF, DIAM, CIRC, PSV, EDV, MV, and VF and a reduction in RI and SAP, which were significantly counteracted from T_{50} to T_{60} for EDV, VF, MV and RI, and to T_{65} for SAP, from T_{50} to T_{90} for CIRC and SURF and to T_{60} for DIAM. These findings demonstrate that a 1 μg/kg BW/minute NOR infusion can reverse ACP's vasodilatory effects, restoring hemodynamic parameters and blood pressure in horses.

Résumé — Évaluation d'altérations hémodynamiques induites par l'acépromazine et inversion par une infusion de norépinéphrine chez des chevaux debout. Les effets d'une infusion de norépinéphrine (NOR) sur les altérations hémodynamiques induites avec des doses sédatives d'acépromazine (ACP) ont été évalués. Une infusion de NOR à 1 μg/kg poids corporel (PC)/minute pendant 15 minutes a été administrée à 5 chevaux debout 45 minutes (T_{45}) après une injection intraveineuse d'ACP à 0,1 mg/kg PC. La tension artérielle systolique (TAS) et les paramètres hémodynamiques ont été évalués sur l'artère médiane. Les paramètres ont été évalués toutes les 5 minutes, de 45 à 65 minutes (T_{65}), puis 75 (T_{75}), 90 (T_{90}) et 105 (T_{105}) minutes après l'administration d'ACP et la surface (SURF), le diamètre (DIAM), la circonférence (CIRC), le pic de vélocité systolique (PVS), la vélocité en fin de diastole (VFD), la vélocité moyenne (VM) et l'écoulement volumétrique (EV) du vaisseau ainsi que l'indice de résistivité (IR) du débit ont été calculés. L'hypotension et la vasodilatation induites par l'acépromazine causant une hausse significative de SURF, de DIAM, de CIRC, de PVS, d'EV, de VM et de EV ainsi qu'une réduction d'IR et de TAS ont été significativement compensées de T_{50} à T_{60} pour EDV, VF, MV et RI, à T_{65} pour SAP, de T_{50} à T_{90} pour CIRC et SURF et à T_{60} pour DIAM. Ces constatations démontrent qu'une infusion de 1 μg/kg PC/minute NOR peut inverser les effets vasodilatoires d'ACP, rétablissant les paramètres hémodynamiques et la tension artérielle chez les chevaux.

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Introduction

Acepromazine (ACP), a phenothiazine (PHE) commonly used in horses as a sedative agent in preanaesthetic protocols (1), or in the treatment of laminitis (2) has peripheral hemodynamic effects in horses: it induces an increase in the blood flow and has a concomitant vasodilatory effect on the digital vasculature (3,4), metatarsal artery (5), and microcirculation in the coronary band and laminae (4). The peripheral vasodilation, evident after intramuscular injection of 0.05 and 0.055 mg/kg body weight (BW) is the major side effect (5,6). Undesirable effects of ACP are particularly prominent and may become life-threatening when horses suffer from hypotension, anemia, or dehydration (1). In normovolemic and hemodynamically stable horses, the drop in blood pressure is without major consequences, but the risk of hypotensive crisis and subsequent collapse is high if the patient shows volume depletion (7). This low blood pressure results from blockade of the α_1-adrenergic receptors (8) or from depression of the central vasomotor center (9).

Other pharmacological properties of ACP are anti-inflammatory and antioxidant effects (2). Acepromazine diminishes monocyte TNF-α production, inhibits the differentiation of monocytes into macrophages (10,11), and decreases the production of reactive oxygen species (2,12). These properties account for the therapeutic value of ACP in equine patients with inflammatory diseases. However, in order to safely administer ACP to high-risk patients, it is essential that a hypotensive crisis be avoided.

Vasoconstrictor drugs, including α-sympathomimetics, such as norepinephrine (NOR) may help to modulate the vasodilatory effects of ACP. Norepinephrine is an endogenous catecholamine, acting as a sympathetic neural and humoral transmitter in most mammalian species (13), having a potent agonist effect on α_1- and α_2-adrenergic receptors, a lesser effect on β_1-adrenergic receptors, and a minor effect on β_2-adrenergic receptors (14). Overall, NOR action results in an intense vasopressor effect that induces an increase in systolic and diastolic arterial blood pressures, which translates into positive inotropic and chronotropic effects (13). Therefore, NOR is primarily used for its intense vasoconstrictor effect in the hemodynamic management of horses under anesthesia, specifically when low arterial blood pressure is refractory to dobutamine (15) or when horses suffer from hyperdynamic shock, in which intravascular fluid resuscitation fails to reverse a low mean arterial blood pressure and systemic vascular resistance is decreased (14).

There is controversy regarding the side effects of NOR. Some authors have noted a decrease in hepatosplanchnic (16) and renal (17) perfusion in patients under normal circulatory conditions; however, others have observed no alterations in renal function in normotensive foals, at a dose of 0.1 µg/kg BW/min (18). In addition, improved mean arterial blood pressure and renal function have been described in hyperdynamic shock in septic humans (19), sheep (20), dogs (17), and foals, in doses ranging from 0.1 to 1.5 µg/kg BW/min (21). The authors are not aware of any pharmacological antagonists to counteract acepromazine side effects in horses. Thus, the objective of this study was to determine if the hemodynamic effects induced by the intravenous administration of ACP at 0.1 mg/kg BW can be reversed by a continuous infusion of NOR at 1 µg/kg BW/min, which may allow a safer application of ACP to patients whose cardiovascular systems are compromised.

Materials and methods

The experimental protocol used in this study was approved by the animal ethical committee of the University of Liège. Five healthy resident research mares, 12 to 27 years old [20 ± 6 y, mean ± standard deviation (SD)] with weight between 420 and 620 kg (478 ± 81 kg) were used. All mares were housed in stocks, to which they were accustomed, and had the same management, health care, and follow-up. They were all acclimatized to the techniques employed in this study before it started. The mares had not been used in any other research for 2 wk prior to the study.

The mares were clipped before leaving the stocks. The trials took place in a quiet examination room for the setup and duration of the experiments. The setup of the experiments took about 15 min and immediately afterwards baseline measurements were done, followed directly by the administration of ACP.

Peripheral hemodynamic variables were measured ultrasonographically, with horses fully weight-bearing on all 4 limbs. Images were taken from the median artery of the left forelimb, immediately below the chestnut, after shaving the medial aspect of the region, using a Phased Array 7 MHz probe coupled to the ultrasound machine (model RT 6800; General Electric, Brussels, Belgium). No offset pad was used but a copious amount of coupling gel was applied. The artery was initially examined in B-mode, in transverse and then in longitudinal planes. The Doppler sample volume was placed centrally within the vessel in order to obtain the velocity waveforms. The angle between the probe and the vessel was always below 55° and the velocity waveforms chosen were those that presented the clearest visual and acoustic signal, and were the most homogenous. For each parameter evaluated, the mean of 3 successive measurements (throughout at least 10 cardiac cycles) was the final value retained.

From a B-mode ultrasonography, the hemodynamic parameters measured included the diameter (DIAM), circumference (CIRC), and surface (SURF) of the vessel. From the Doppler images, the peak systolic velocity (PSV) and the end diastolic velocity (EDV) were measured by placing the cursor at the apex of the maximal upward motion of blood flow, during systole, and at the minimal velocity of blood flow, during the end of diastole, respectively. The area under the velocity waveform (VTI) was measured by tracing the modal velocity envelope, represented by the brightest line in the spectral Doppler waveform. The heart rate (HR) was calculated by counting the number of Doppler curves per minute and the mean velocity (MV), volumetric flow (VF), and resistivity index (RI) were calculated using the following formulae:

$$MV = VTI \times HR \quad (5)$$

$$VF = SURF \times MV \quad (22)$$

$$RI = (PSV - EDV)/PSV \quad (22)$$
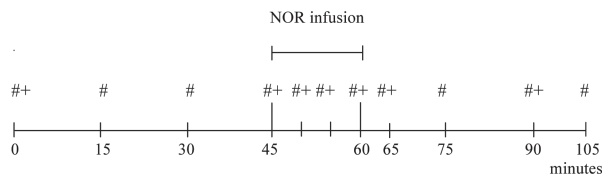
Figure 1. Chronology of the study. Baseline measures were done, immediately before the intravenous administration of acepromazine at 0.1 mg/kg BW, at 0 minutes; from 45 to 60 minutes the norepinephrine infusion was administered at 1 μg/kg BW/minute.
# SAP measure; + ultrasonographic images.

The arterial velocity waveforms were also morphologically analyzed for shape of the systolic peak and amount and direction of blood flow during diastole.

Heart rate, cardiac rhythm, and a continuous base-apex ECG were continuously recorded by a Holter monitor (model Vista; Verimed Medical Supply, Wetteren, Belgium) during the study. Systolic arterial blood pressure was indirectly and manually measured at the tail, using an ultrasonic Doppler flow detector (model 811-B; Park's Medical Electronics, 9.5 MHz probe) together with a 10 cm width cuff and a manometer. The SAP values were the mean of 3 consecutive measurements.

Before the beginning of the study, a 16 G catheter was aseptically inserted in the left jugular vein, in a clipped site, previously anesthetized with lidocaine 2%. Each mare received an intravenous bolus of ACP (Combistress; Kela Laboratoria, Hoogstraten, Belgium), 0.1 mg/kg BW and 45 min later an intravenous infusion of NOR (Levophed; Abbot N.V., Wavre, Belgium), 1 μg/kg BW/min for 15 min. Norepinephrine infusion would be interrupted if 1 of the following situations was presented: a HR higher than 60 beats/min, an increase in the SAP over 180 mmHg, abnormalities in cardiac rhythm, or excitement. The choice of the dose of NOR was based on a preliminary protocol, in which the mares received only NOR without ACP premedication.

The SAP was recorded immediately before administration of ACP (T_0) and every 15 min for 45 min after administration. Then the infusion of NOR was immediately started and maintained for 15 min while the SAP was measured every 5 min and ultrasound images were taken. At the end of the NOR infusion SAP continued to be regularly measured until T_{105} and ultrasound images continued to be taken until T_{90}. Overall SAP was measured at T_0, T_{15}, T_{30}, T_{45}, T_{50}, T_{55}, T_{60}, T_{65}, T_{75}, T_{90}, and T_{105} and ultrasonographic images of the median artery of the left forelimb were taken at T_0, T_{45}, T_{50}, T_{60}, T_{65}, and T_{90} (Figure 1).

Data were analyzed using Statistical Analysis System software (SAS Institute, Cary, North Carolina, USA) with a mixed model (PROC MIXED). This model evaluates the variations over time induced by ACP, from T_0 to T_{45}, and by NOR, from T_{50} to T_{60}, and takes into consideration repeated measures on different individuals. The Shapiro-Wilk test was used to evaluate normality. When normality failed, a Friedman's test was conducted, as an alternative to analysis of variance (ANOVA), to evaluate the differences between repeated measures. Statistical differences were denoted at $P < 0.05$ with values expressed as least square means ± standard deviation (SD).
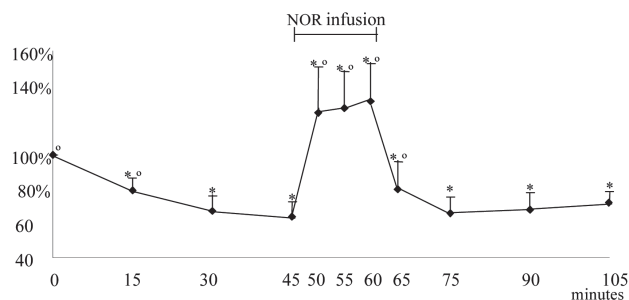
Figure 2. Mean (± SD) of the variation percentage of the systolic arterial pressure before, during, and after norepinephrine (NOR) infusion. At 0 minutes acepromazine at 0.1 mg/kg BW was administered intravenously; from 45 to 60 minutes the norepinephrine (NOR) infusion was administered at 1 μg/kg BW/minute.
* significantly different from T_0. ° significantly different from T_{45}.

Results

Acepromazine induced a significant drop in the SAP from T_{15} to T_{45}. However, during the NOR infusion, the SAP significantly increased at T_{50}, T_{55} and T_{60}, compared with T_0 and T_{45}, respectively. At T_{65} the SAP rapidly decreased, but remained statistically higher compared to T_{45}. From T_{75} to T_{105}, SAP was no longer statistically higher than at T_{45}. From T_{90} onwards only the effect of ACP was present (Figure 2, Table 1).

Acepromazine induced a significant increase in the DIAM, CIRC and SURF, at T_{45}, in comparison with T_0. However, during the NOR infusion, those parameters significantly decreased from T_{45}, and became similar to the values measured at T_0. At T_{65} and T_{90}; the CIRC and SURF, although numerically increased, stayed statistically smaller than at T_{45}, while the DIAM, at T_{65} and T_{90}, increased again, to values similar to those at T_{45} (Figure 3, Table 2).

At T_{45}, the PSV, EDV, MV, and VF significantly increased, while RI significantly decreased, compared with T_0 values. During the NOR infusion, at T_{50} and T_{60}, the EDV and VF significantly diminished with regard to T_{45}, and the RI increased, with these parameters becoming similar to the values recorded at T_0. From T_{45}, PSV continuously increased compared with T_0; however, the difference wasn't significant and during this period it did not statistically change with respect to T_{45}. The MV had a tendency to decrease at T_{50} ($P < 0.093$) and T_{60} ($P < 0.052$), compared to T_{45}. When the NOR infusion ended, all hemodynamic parameters approached values previously recorded at T_{45} (Figures 4 and 5; Table 2).

The Doppler waveforms observed at T_0 were biphasic, with a small spectral window and no reverse flow, which is characteristic of an intermediate resistance flow pattern. During systole, there was a sharp rise in flow velocity and a rapid decline toward baseline, and during diastole an oscillatory portion with alternating acceleration and deceleration of the antegrade blood flow. At T_{45}, the waveforms matched a low resistance flow pattern, with a diastolic velocity higher than at T_0, and a smaller Doppler shift on the initial alternating acceleration-deceleration phase. During the NOR infusion the Doppler curves indicated a smaller diastolic velocity, comparable to those from T_0.

Table 1. Values for systolic arterial pressure (SAP) before (T_0) and 45 minutes after (T_{45}) administration of acepromazine (ACP), during continuous infusion of norepinephrine (NOR) between T_{45} and T_{60} and after NOR infusion, from T_{60}. Results are given as mean ± SD

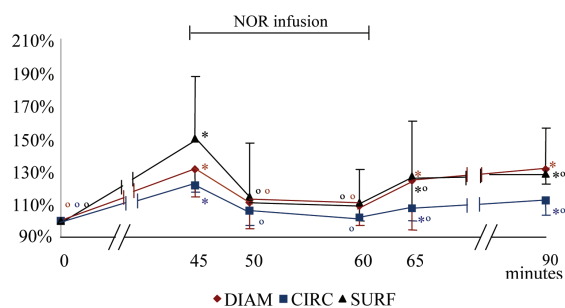| Time ACP/NOR	SAP (mmHg)
T_0	108.9º ± 11.24
T_{15}	85.3*º ± 4.31
T_{30}	72.4* ± 6.68
T_{45}	68.7* ± 7.39
T_{50}	133.7*º ± 24.98
T_{55}	137.3*º ± 20.77
T_{60}	141.0*º ± 19.53
T_{65}	86.3*º ± 11.27
T_{75}	71.1* ± 9.31
T_{90}	73.9* ± 9.52
T_{105}	78.3* ± 5.97

Each value of SAP is significantly different ($P < 0.05$) from T_0 if marked with * and from T_{45} if marked with º.

Figure 3. Mean (± SD) of the variation percentage of the diameter (DIAM), circumference (CIRC) and surface (SURF) of the median artery before, during, and after norepinephrine infusion (NOR). At 0 minutes acepromazine at 0.1 mg/kg BW was administered intravenously; from 45 to 60 minutes the norepinephrine (NOR) infusion was administered at 1 µg/kg BW/minute.
* significantly different from T_0. º significantly different from T_{45}.

During the study, horses appeared sedated after ACP administration; those signs were absent during NOR infusion and reappeared after it, with less magnitude. None of the mares had excessively high arterial blood pressures or arrhythmias and all recovered uneventfully after completing the experimental protocol.

Discussion

The present study confirms the hemodynamic effects of intravenous ACP administration and reveals that NOR infusion at 1 µg/kg BW/min can reverse ACP's vasodilatory effects, restoring hemodynamic parameters and blood pressure in a group of healthy adult horses. Throughout the study, the horses were tranquilized; however, as evaluation of sedation was not an objective, the study only evaluated the hemodynamic effect of ACP and NOR at the given doses.

Acepromazine's vasodilatory properties can be expressed through a significant decrease in SAP and RI, and a significant increase in DIAM, CIRC, SURF, VF, PSV, EDV, and MV. In this study, such responses were observed during the period in which horses were exclusively under the effect of ACP, before, from T_0 to T_{45}, and after, from T_{65} onwards, NOR infusion. Results from our study are in agreement with those previously published and confirm a decrease in SAP (23,24), and an increase in DIAM (5,25) after ACP administration. The augmentation of CIRC and SURF, from T_0 to T_{45}, observed in the current study confirms results from previous reports, demonstrating that ACP administration induces a common vasodilatory effect (3). As well there was augmentation of VF, at T_{45} in the current study, previously reported in healthy horses after administration of ACP (3,5,24). The augmentation of VF has been associated with increases in the vessel diameter and the maximal flow velocity and a decrease in the peripheral resistance (22), which are compatible with the augmentation in PSV and MV and the diminution of RI. Additionally, the present study demonstrates that the morphology of Doppler waveforms varies in parallel, as it is consistent with vasodilation observed after ACP administration, when waveforms turn from an initial intermediate resistance flow pattern to a low resistance one. When compared with an intermediate resistance flow, the low resistance pattern is characterized by a higher dia-

stolic velocity, and a greater amount of diastolic flow, which is consistent with peripheral vasodilation (26) and diminution of the Doppler shift following the systolic peak and lower pulsatility waveform (27).

Forty-five minutes after ACP administration, when all the signs of vasodilation were present in the horses, a 15-minute NOR infusion progressively reversed ACP's effect on the majority of parameters studied. Indeed, between T_{50} and T_{65}, the SAP not only significantly increased after NOR infusion, but became higher than the values recorded at T_0, which agrees with results previously reported in foals (15,18,28), dogs (17,29), sheep (20), and humans (30,31). The NOR infusion induced a significant increase in SAP for 15 min and then was progressively attenuated from T_{65} onwards. Based on clinical experience, similar reports have been cited with adult horses (15), and are in agreement with our results. As the values of SAP became higher during NOR infusion than at T_0, we could question if the doses of NOR should be lower. To determine this, further studies could investigate the effect of different doses of NOR administered to horses under ACP premedication. During the period of NOR infusion, after T_{45}, the CIRC and the SURF significantly decreased, from T_{65} to T_{90} and the DIAM from T_{45} to T_{65}, proving there was an induced vasoconstriction and being in agreement with the significant diminution of EDV and the VF (32), while the increase in peripheral resistance was supported by an increase in RI (33). Besides, during NOR infusion, the PSV and the MV did not statistically change, with respect to T_{45}; only MV had a tendency to decrease, from T_{50} to T_{60}, which is also consistent with vasoconstriction (32). When analyzing changes in the morphology of Doppler waveforms during NOR infusion, with respect to T_{45}, these are compatible with an augmentation in the vascular resistance, resulting in vasoconstriction. This effect can occur through augmentation of the oscillations, and consequent higher pulsatility (27), or through a smaller diastolic velocity (32), as observed.

Overall, once NOR infusion was completed, at T_{60}, apart from CIRC and SURF, the rest of the parameters that varied during NOR infusion, returned to values similar to those at T_{45}, when the only effect was vasodilation from ACP's action.

Table 2. Values for surface (SURF), circumference (CIRC), diameter (DIAM), peak systolic velocity (PSV), end diastolic velocity (EDV), mean velocity (MV), volumetric flow (VF) and resistivity index (RI) of the left median artery with time, before (T_0) and 45 minutes after (T_{45}) administration of acepromazine (ACP), during continuous infusion of norepinephrine (NOR) between T_{45} and T_{60} and after NOR infusion, from T_{60}. Results are given as mean ± SD

	Time					
ACP/NOR	T_0	T_{45}	T_{50}	T_{60}	T_{65}	T_{90}
DIAM (cm)	0.30° ± 0	0.393* ± 0.04	0.333° ± 0.04	0.327° ± 0.03	0.373* ± 0.07	0.393* ± 0.03
CIRC (cm)	1.113° ± 0.04	1.347* ± 0.05	1.18° ± 0.08	1.133° ± 0.06	1.193*° ± 0.14	1.247*° ± 0.09
SURF (cm)	0.085° ± 0.01	0.127* ± 0.03	0.095° ± 0.02	0.093° ± 0.02	0.107*° ± 0.03	0.107*° ± 0.02
PSV (m/s)	0.294° ± 0.06	0.427* ± 0.15	0.456* ± 0.24	0.440* ± 0.17	0.473* ± 0.14	0.482* ± 0.14
EDV (m/s)	0.041° ± 0.02	0.165* ± 0.07	0.095° ± 0.11	0.078° ± 0.08	0.148* ± 0.08	0.172* ± 0.09
MV (m/s)	0.095° ± 0.03	0.265* ± 0.15	0.159 ± 0.13	0.141 ± 0.08	0.330* ± 0.24	0.261* ± 0.09
VF (mL/min)	47.51° ± 14.93	202.47* ± 139.35	87.76° ± 61.43	76.86° ± 37.31	207.13* ± 152.92	166.02* ± 60.82
RI	0.864° ± 0.04	0.618* ± 0.04	0.825° ± 0.09	0.845° ± 0.08	0.699* ± 0.09	0.655* ± 0.12

Each value is significantly different ($P < 0.05$) from T_0 if marked with * and from T_{45} if marked with °.
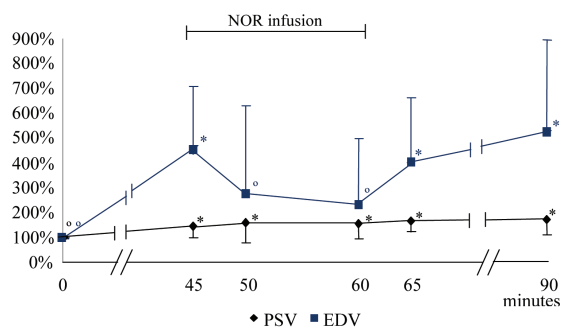
Figure 4. Mean (± SD) of the variation percentage of the peak systolic velocity (PSV) and the end diastolic velocity (EDV) of the median artery before, during, and after norepinephrine infusion (NOR). At 0 minutes acepromazine at 0.1 mg/kg BW was administered intravenously; from 45 to 60 minutes the norepinephrine (NOR) infusion was administered at 1 µg/kg BW/minute.
* different from T_0. ° significantly different from T_{45}.
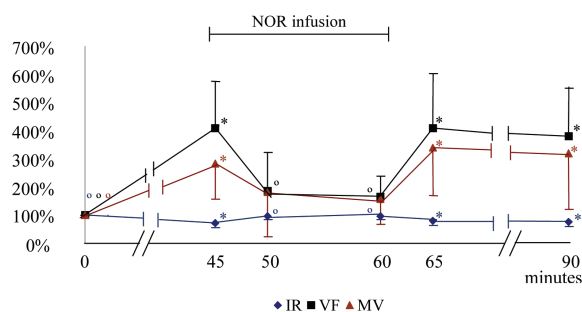
Figure 5. Mean (± SD) of the variation percentage of the resistivity index (RI), volumetric flow (VF), and mean velocity (MV) of the median artery before, during, and after norepinephrine infusion (NOR). At 0 minutes acepromazine at 0.1 mg/kg BW was administered intravenously; from 45 to 60 minutes the norepinephrine (NOR) infusion was administered at 1 µg/kg BW/minute.
* significantly different from T_0. ° significantly different from T_{45}.

Consequently throughout a NOR infusion of 1 µg/kg BW/min the hypotension induced by ACP can be reversed.

Practically, the use of NOR to counteract hemodynamic effects of ACP may seem extreme, but results from our study provide equine clinicians and anesthesiologists a new clinical approach to counteract those effects, in particular in patients intolerant to ACP. To the authors' knowledge the hemodynamic response on standing adult hypotensive horses to a NOR infusion has not been reported previously. As a consequence of α-adrenergic activity of NOR, which primarily causes vasoconstriction (13), the effects observed in the present study are those expected. Furthermore, the fact that no sign of epinephrine reversal was observed, following the concomitant administration of ACP and NOR, confirms the minor β₂-adrenergic receptor activation by NOR. Additionally the infusion of NOR not only reverted ACP's α-adrenergic blocking effect, but also induced a significantly higher SAP compared with the baseline values. The present study appears to be the first to report such results, where there are no significant changes from the baseline of systemic arterial blood pressure, following the administration of a different α-adrenergic agonist drug, such as romifidine, subsequent to ACP's administration, although romifidine is known to induce a transitory initial period of arterial hypertension (34).

The capacity of NOR to reverse ACP's induced hypotension could eventually allow a better and more frequent use of ACP in the pre-anaesthetic medication protocol in some patients, permitting equine practitioners to take advantage of the protective effect of ACP during general anesthesia (35). Indeed, when horses from the same group at risk received an α₂-adrenergic receptor agonist for preanesthetic medication, there was a reduction in the cardiac output (36). It would be of interest to evaluate the effect of using a reduced dose of preanesthetic α₂-adrenergic receptor agonist, concurrent with ACP administration, as an alternative protocol option in horses. This could be possible if NOR administration could be used to modulate the hypotensive and vasodilatory effects of ACP, although this administration would need to be closely monitored.

During the preliminary phase of this experiment, the same 5 mares that received an intravenous infusion of NOR at 1 µg/kg BW/min, without being premedicated with ACP, showed second degree atrioventricular blocks (2AVB). Although other types of arrhythmia can be linked to sympathetic activation (37,38), to the authors' knowledge, the induction of 2AVB by NOR administration has not been reported. Additionally, when these mares were premedicated with ACP before the infusion of NOR, they developed a statistically lower

frequency of 2AVB. It has been reported that ACP has the capacity to increase the arrhythmogenic dose of epinephrine (39), thus the ability of ACP to enhance the baroreceptor reflex (40), which mediates rapid changes in sympathetic and parasympathetic activity in response to changes in blood pressure (41), is most probably related to the ACP's protective effect observed in this study. It is possible that the benefits of ACP are related to its α-adrenergic blocking effect and vasodilation; therefore, the NOR infusion, even when allowing a safer ACP administration to horses at risk, could reduce the beneficial properties induced by ACP. Studies are needed to investigate maintenance of the beneficial effects of ACP, when administered with NOR, to counteract its hemodynamic effects.

In the present study, the use of Doppler ultrasonography proved to be sensitive enough to detect vascular and hemodynamic alterations in the standing horse, while evaluating the effect of drugs with opposite properties. These results help to confirm that Doppler ultrasonography can be successfully applied as a noninvasive technique to measure hemodynamic changes in horses with blood flow alteration disorders, and promote its use in studies to control the hemodynamic effects of treatments on the standing horse.

We conclude that a 15-minute continuous infusion of NOR at 1 μg/kg BW/min has the capacity to reverse the hypotension and vasodilation induced by ACP at 0.1 mg/kg BW, restoring hemodynamic parameters in healthy standing horses. In particular, the significant rise in SURF, DIAM, CIRC, PSV, EDV, MV, and VF and the reduction in RI and SAP which were significantly counteracted from T_{50} to T_{60} for EDV, VF, MV, and RI, and to T_{65} for SAP, from T_{50} to T_{90} for CIRC and SURF, and from T_{50} to T_{60} for DIAM.

Acknowledgment

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Book Review
Compte rendu de livre

Exotic Animal Medicine for the Veterinary Technician — 2nd edition

Ballard B, Cheek R. 2010. Blackwell Publishing, Ames, Iowa, USA. 484 pp. ISBN: 9780-8138-2206-8. $65.59.

As a veterinarian with a passion for exotic animal medicine, I am all too aware of the range of challenges facing veterinarians wanting to work in this field. Exotic animal medicine is a relatively new area of specialization, and is evolving rapidly. It takes a concerted effort to keep on top of the latest trends. One of the things that has allowed me to continue pursuing my passion in my practice is having a skilled team of animal health technologists who share my love of all things weird and wonderful. It is for this reason that I was so excited to read this book, as I hoped it would prove to be a valuable resource for our staff.

The book is divided into sections on birds, reptiles, amphibians, exotic companion mammals, wildlife, and hematology. Each section is further subdivided into chapters, each covering a range of topics, including anatomy, physiology, husbandry, nutrition, common diseases, behavior, reproduction, restraint, parasitiology, radiology, euthanasia, etc. The pictures within the chapters are in black and white, but many of them are also printed in color in the center of the book.

I was impressed with over-all quality of the information covered in the book; it is up-to-date and well-explained. I particularly enjoyed the avian chapters, which included excellent information on nutrition and behavior. The amphibian section has an excellent description of anesthetizing frogs with topical isoflurane, which I have seen discussed in other references, but never in such detail.

The book presents a lot of very practical information. Most of the sections included diagrams of the locations of vessels for venipuncture, which is very much appreciated. In the wildlife chapter, there is a very useful section that simply covered what you should recommend on the phone when people find injured or orphaned wildlife, including tricks and tips on how to determine if an animal is actually orphaned. There are also extensive tables in the appendices of how to house and what to feed different wildlife species, and even how to tell nestling songbirds apart.

The one complaint I would have is that I found the lizard chapter a bit frustrating to read, as though the author was talking over my head. Given that the intended audience for this book are AHTs, I believe that this is a significant weakness. For example, the author would often give the scientific name of a species without listing the common name as well, which made it difficult to keep track of what species was being discussed. On page 95, the author mentions *Heloderma* spp. as being dangerous to humans, but never mentions anywhere in the chapter what the common name is, or why they are dangerous. This frustrated me, and I ended up having to search for this information on the Internet (for those who are interested, *Heloderma* spp. include gila monsters and bearded lizards, both of which are venomous). Given that the author was trying to inform the reader about the risks these animals could pose, it would have been prudent to provide more detail, rather than assuming people already knew what he was referring to, or forcing them to search for this information elsewhere. Another thing I found a little odd is that in the parasitology portion of the chapter, there are a number of photo plates of different species of parasites. One of the photos is of a parasite named *Nycototherus,* but this parasite is not mentioned anywhere in the text. It is actually an interesting looking parasite, which I assume was part of the reason it was included, but it would have been nice to have a bit more information on the species it affects, and what the clinical symptoms associated with it are.

Over-all, I feel that this is a very useful reference, both for animal health technologists, and veterinarians. There are plenty of tables, diagrams, and pictures that would be helpful as a quick reference.

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