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The following studies all received ethical approval by institutional and/or national review committees

Equine

Continuous thermodilution (CCO), lithium dilution (LiDCO) and pulse contour analysis (PulseCO) cardiac output measurement methods compared to iced bolus thermodilution (BTD) technique in anaesthetized ponies

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Cardiac output (Q) measurement is a key element for cardiovascular evaluation. The BTD is considered as a reference method. New semi-continuous (CCO), continuous (PulseCO) or less invasive methods (LiDCO, PulseCO) have become available. The aim of this study was to evaluate their possible interchangeability with BTD.

Eight healthy ponies were anaesthetized and instrumented for the different Q measurement methods. Q changes were induced by increasing followed by decreasing isoflurane concentration and dobutamine infusion. The data were studied using regression analysis and Bland-Altman methodology. An *a priori* limit of agreement of up to ±30 % was defined for acceptance of the interchangeability with BTD.

A total of 135 paired measurements for BTD-CCO, 133 for BTD-PulseCO and 46 for BTD-LiDCO were available. Mean Q (range) measured by BTD was 6.9 L minute^{-1} (1.3 to 18.5) By CCO, PulseCO and LiDCO, mean Q (range) was respectively 7.6 (2.7 to 20.0), 10.1 (3.1 to 26.7) and 9.8 L minute^{-1} (3.8 to 23.1). Compared to BTD, correlation factors (r), biases and limits of agreement (95% CI) were respectively: 0.82, 0.7 ± 4.1 L minute^{-1} for CCO, 0.91, 3.2 ± 3.5 L minute^{-1} for PulseCO and 0.95, 2.9 ± 2.7 L minute^{-1} for LiDCO.

Good correlation with BTD was found for the three methods. However, all three methods (CCO, PulseCO, LiDCO) tended to overestimate the cardiac output. This is especially true for LiDCO and PulseCO. Only the LiDCO method showed acceptable limits of agreement within 30%. CCO and PulseCO are not interchangeable with BTD.

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Sedative Effects of Increasing the Doses of Detomidine and Buprenorphine in Horses

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Sedative effects of detomidine and buprenorphine have previously been reported (Love et al 2007). Buprenorphine (10 μg kg^{-1}) may produce analgesia for up to 11 hours (Carregaro et al 2007), but when combined with 10 μg kg^{-1} detomidine, sedation was reduced compared to sedation from detomidine and 5 or 7.5 μg kg^{-1} buprenorphine. The aim of this study was to investigate the effects of increasing the doses of detomidine and buprenorphine on sedation.

Six healthy adult horses (aged 3–11 years, mass 400–550 kg) were used in a randomized, cross over, blinded study. Horses received four treatments intravenously at two week intervals; saline S/S, detomidine 10 μg kg^{-1} and buprenorphine 7.5 μg kg^{-1} (D10/B7.5), detomidine 20 μg kg^{-1} and buprenorphine 7.5 μg kg^{-1} (D20/B7.5) and detomidine 20 μg kg^{-1} and buprenorphine 10 μg kg^{-1} (D20/B10). Sedation was assessed and recorded using an interactive VAS (0 mm = no sedation, 100 mm = unconscious) before and 1, 5, 10, 15, 20, 25, 30 minutes after treatment and at 10 minute intervals thereafter until three consecutive scores were 0.

Peak sedation, duration of sedation and area under curve (AUC) for sedation scores were investigated using a univariate general linear model for multiple factors with post-hoc Tukey tests ($p < 0.05$).

Peak sedation and duration of sedation (mean ± SD) following S/S (0 ± 0 mm, 0 ± 0 minutes) were significantly different from D10/B7.5 (75 ± 13 mm, 35 ± 13 minutes), D20/B7.5 (77 ± 10 mm, 57 ± 26 minutes) and D20/B10 (80 ± 5 mm, 52 ± 21 minutes) $p < 0.000$. There were no significant differences between treatments containing detomidine. AUC for S/S (0 ± 0 mm^2) were significantly different from D10/B7.5 (1815 ± 938 mm^2), D20/B7.5 (3280 ± 1157 mm^2) and D20/B10 (3196 ± 1450) ($p < 0.000$). D10/B7.5 was significantly different from D20/B7.5 ($p = 0.033$) and D20/B10 ($p = 0.046$) but D20/B7.5 was not significantly different from B20/B10 ($p = 0.998$).

Increasing the dose of detomidine from 10 μg kg^{-1} to 20 μg kg^{-1} increased the degree of sedation produced (AUC) when administered with the same dose of buprenorphine (7.5 μg kg^{-1}). Increasing the dose of buprenorphine from 7.5 μg kg^{-1} to 10 μg kg^{-1} when administered with 20 μg kg^{-1} detomidine did not adversely affect the degree of sedation produced.

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Carregaro AB, Luna SPL, Mataqueiro MI et al. (2007) Effects of buprenorphine on nociception and spontaneous locomotor activity in horses. American Journal of Veterinary Research 68, 346–250.

Love EJ, Taylor PM, Murrell J et al. (2007) Buprenorphine Administered With Detomidine: Assessment of Sedative Effects in Horses. Proceedings of the Association of Veterinary Anaesthetists Autumn Meeting. September 19–21, Leipzig, Germany. pp35

Cardiovascular function in anaesthetized horses during transvenous electrical cardioversion as therapy for atrial fibrillation

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The aims of this study were to evaluate cardiovascular function in anaesthetized horses with lone atrial fibrillation (AF) and to examine the influence of direct current shock application and of conversion to sinus rhythm (SR).

Seven anaesthetic records were reviewed after successful treatment for AF (transvenous electrical cardioversion after amiodarone pre-treatment (6.52 mg kg^{-1} $hour^{-1}$ for 1 hour, 1.1 mg kg^{-1} $hour^{-1}$ during 23 hrs)). Cardioversion catheters and a pacing catheter were placed under sedation (detomidine 10 μg kg^{-1}, methadone 0.1 mg kg^{-1} IV). After induction (ketamine 2.2 mg kg^{-1}, midazolam 0.06 mg kg^{-1} IV) in a sling, anaesthesia was maintained with isoflurane in oxygen. Flunixin meglumine (1.1 mg kg^{-1}) was administered IV. Shocks were delivered as biphasic truncated exponential waves, synchronized with the R wave of the electrocardiogram. Monitoring included pulse oximetry, electrocardiography, anaesthetic gases, arterial blood pressure, LiDCO and PulseCO cardiac index (CI) and arterial blood gases. Descriptive statistics were obtained for the first 30 minutes of anaesthesia (before first shock). Values before and after the first unsuccessful shock and before and after conversion to SR were compared using paired t-tests.

Values before the first shock were comparable to reports in healthy, isoflurane-anaesthetized horses (Grosenbaugh & Muir 1998). Reliable CI measurements could not be obtained using the PulseCO. Intermittent positive pressure ventilation was needed in most horses (bradypnea and/or $PaCO_2$ > 60 mmHg), dobutamine in 2 horses (0.3–0.5 μg kg^{-1} $minute^{-1}$). Recorded values were not influenced by unsuccessful cardioversion attempts (CI measurements not available for this analysis). During SR, stroke index was higher ($p = 0.006$) and systolic arterial pressure (SAP) lower ($p = 0.03$) than during AF.

Despite the presence of lone AF, cardiovascular function was well maintained during anaesthesia and was not affected by shock application. Stroke index increased and SAP decreased after cardioversion.

Grosenbaugh DA, Muir WW (1998) Cardiorespiratory effects of sevoflurane, isoflurane and halothane anesthesia in horses. Am J Vet Res 59, 101–106