Veterinary
Anaesthesia and Analgesia
Formerly the Journal of Veterinary Anaesthesia

Veterinary Anaesthesia and Analgesia, 2014, **41**, 14–24 doi:10.1111/vaa.12055

RESEARCH PAPER

Evaluation of sedation for standing clinical procedures in horses using detomidine combined with buprenorphine

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Abstract

Objectives To examine the effect of including buprenorphine with detomidine for sedation of horses undergoing clinical procedures.

Study design Partially blinded, randomised, prospective clinical field trial.

Animals Eighty four client-owned horses scheduled for minor surgery or diagnostic investigation under standing sedation.

Methods The effects of buprenorphine (5 μg kg^{-1}) (Group B, $n = 46$) or placebo (5% glucose solution) (Group C, $n = 38$) in combination with detomidine (10 μg kg^{-1}) were compared in standing horses undergoing minor clinical procedures. The primary outcome measure was successful completion of the procedure. The degree of sedation and ataxia were scored using simple descriptive scales. Heart and respiratory rates were recorded at 15–30 minute intervals. Parametric data from each group were compared using ANOVA or *t*-test and non parametric data using the Mann–Whitney *U* test.

Results The procedure was carried out successfully in 91% of Group B and 63% of Group C ($p < 0.01$). Repeat dosing was required in 24% of Group B and 32% of Group C ($p < 0.05$). Sedation was more profound and lasted longer (60 *versus* 45 minutes) in Group B ($p < 0.01$). Ataxia occurred after detomidine, increased after buprenorphine but not glucose administration, was more profound in group B and lasted longer (60 *versus* 30 minutes) $p < 0.001$). Heart and respiratory rates remained within normal limits in both groups and there were no serious adverse events.

Conclusions and clinical relevance Buprenorphine 5 and 10 μg kg^{-1} enhanced the sedation produced by detomidine 10 and 20 μg kg^{-1} with minor side effects similar to other alpha$_2$ agonist/opioid combinations. Detomidine–buprenorphine sedation is suitable for standing procedures in horses.

Keywords alpha$_2$ adrenoceptor agonist, buprenorphine, detomidine, equine, opioid, sedation.

Introduction

Numerous diagnostic and minor surgical procedures are performed on sedated, standing horses because general anaesthesia carries a substantial risk of mortality and morbidity in this species (Johnston et al. 2004). General anaesthesia also requires appropriate facilities and expertise as well as incurring considerable cost.

Acepromazine and the alpha$_2$ adrenoceptor agonists are the most common individual agents used for chemical restraint, but it is well recognized that addition of an opioid enhances their sedative effects without seriously compromising vital function (Clarke & Paton 1988; Taylor & Clarke 2007). Alpha$_2$ adrenoceptor agonist combinations using xylazine, detomidine or romifidine have been described with a number of different opioids given as a bolus injection (Muir et al. 1979; Robertson & Muir 1983; Taylor & Clarke 2007) or by infusion for longer procedures (Hainisch 2001; Ringer et al. 2012a,b; 2013). In the UK at least, detomidine or romifidine with butorphanol are probably the most commonly used for sedation from a simple bolus injection (Taylor et al. 1988; Browning & Collins 1994). Both combinations have UK Market Authorization.

Buprenorphine hydrochloride (Vetergesic Multidose; Alstoe Ltd, UK) has recently received UK Market Authorization for analgesia and as an adjunct to sedation in horses. Buprenorphine is generally regarded as a partial mu (OP3)-agonist opioid and is used widely for analgesia and sedation in cats and dogs as well as laboratory animals and exotics (Roughan & Flecknell 2002). A number of investigations document buprenorphine's effect in horses: in accordance with this class of drug, in normal, pain-free, research animals it produces antinociception and some sympathetic and locomotor stimulation (Carregaro et al. 2006, 2007; Love et al. 2012). Combinations of buprenorphine (4–10 $\mu g\ kg^{-1}$) with sedatives have also been evaluated under laboratory conditions, showing opioid-enhancement of xylazine, detomidine and romifidine sedation similar to that of other opioids (Nolan & Hall 1984; Love 2009; Cruz et al. 2011; Love et al. 2011a,b). One investigation has examined the use of buprenorphine for sedation under clinical conditions (van Dijk et al. 2003). Detomidine (10 $\mu g\ kg^{-1}$) and buprenorphine (6 $\mu g\ kg^{-1}$) were given prior to laparoscopy and sedation was maintained by continuous infusion of detomidine. The combination was effective for the purposes, and there were no adverse effects.

Studies investigating buprenorphine–detomidine sedation after injection only for a range of common clinical procedures have not yet been published. This study describes a field trial which examined the effects of inclusion of buprenorphine with detomidine for sedation of horses undergoing clinical procedures.

Materials and methods

Study design

A prospective, multi-centre, placebo-controlled investigation of buprenorphine's effects on detomidine sedation was carried out in 86 client-owned horses undergoing clinical diagnostic or surgical procedures in general practices in the UK. The study was carried out in accordance with Good Clinical Practice (CVMP/VICH/595/98) under Animal Test Certificate (ATC) as part of the registration process for Market Authorisation. Informed owner consent was obtained in all cases.

Horses

Horses admitted to one of seven participating UK equine clinics were randomly allocated (block randomisation, GraphPad Prism; GraphPad Software Inc., CA, USA) to receive either buprenorphine (group B) or 5% glucose placebo (group C) in addition to detomidine for standing sedation to enable a diagnostic or minor surgical procedure to be carried out. All horses admitted for such treatment were eligible, but were excluded if they had been heavily sedated in the previous week, had cardiac dysrrhythmias, colic, impaired respiratory or liver function, were pregnant or lactating, or were under treatment with sympathomimetic amines, potentiated sulphonamides or drugs causing respiratory depression. Other antibiotics, non steroidal anti-inflammatory agents and intravenous fluids were permitted. Food was withheld at least four hours prior to treatment and was made available again after recovery. Water was available *ad libitum* prior to treatment and after recovery.

Drug treatment

All horses were sedated with 10 $\mu g\ kg^{-1}$ detomidine hydrochloride (Domosedan; Pfizer Ltd, UK) given by slow intravenous (IV) injection 3–5 minutes before administration of the test/control substance (BorG): either 5 μg^{-1} buprenorphine (Vetergesic Multidose; Alstoe Ltd, UK) (group B) or an equivalent volume (0.8 mL 50 kg^{-1}) of 5% glucose solution (Baxter Healthcare Ltd, UK) (group C). If, 10 minutes after administration of the BorG, sedation was judged inadequate to carry out the procedure, then the doses of detomidine and BorG were repeated at the same time interval. If, 10 minutes after administra-

tion of the second doses, sedation was judged still to be inadequate to carry out the procedure, rescue sedation was implemented.

If rescue sedation was required, this was recorded, and the horse withdrawn from further participation in the study. The identity of the BorG was revealed, and further sedation at the discretion of the veterinarian responsible for the case was given.

At each clinic, the identity of the BorG injection was withheld from the assessor by using a dispenser, who drew up the appropriate BorG solution into an unidentified syringe for IV administration. The assessor undertook all the assessments without knowing the identity of the BorG solution administered. Buprenorphine and glucose solutions were clear and colourless, administered on the basis of 0.8 mL 50 kg^{-1}, making it impossible to distinguish visually between them.

Assessments

The depth of sedation and degree of ataxia were assessed using simple descriptive scales (SDS, Table 1) prior to and at 15, 30, 45, 60, 90 and 120 minutes after the injection of BorG. Heart and respiratory rates were also recorded at the same time points. The primary outcome measure was overall success of the procedure assessed on a scale of 1–4 (score 1–2 = success, score 3–4 = unsuccessful) (Table 1).

Table 1 Simple descriptive scale (SDS) scoring system for sedation, ataxia and overall outcome. All assessors gained experience in the SDS systems prior to trial data collection. Assessments falling between descriptors were awarded the higher score

Score	Signs
Sedation	
0	No sedation. Animal is alert, with normal posture and response to contact with assessor. Normal objection to intervention
1	Mild sedation. Low head carriage, relaxed facial muscles and pendulous lower lip. Some response to intervention
2	Moderate sedation. Head lowered towards ground and swaying of hind legs. Slight response to intervention
3	Marked sedation. Attempts to or becomes recumbent. No response to intervention
Ataxia	
0	No ataxia. Animal stands and walks normally; is able to turn tightly
1	Mild ataxia. Animal able to walk, but some lack of limb control
2	Moderate ataxia. Animal can walk only with support, staggers but saves itself from falling
3	Marked ataxia. Animal is unable to walk without danger of falling, staggers, falls if turned
Overall outcome	
1	Satisfactory (procedure carried out easily with no interference from the horse)
2	Acceptable (procedure completed fairly easily, but with some interference from the horse's response)
3	Not satisfactory (procedure completed but with some difficulty due to interference from the horse)
4	Impossible (procedure had to be abandoned due to lack of sedation) Outcome score 1–2 = success, score 3–4 = failure)

Statistical analysis

The null hypothesis was that there would be no statistical difference in primary outcome measure between the groups indicating that buprenorphine did not confer additional sedative effect over that of detomidine alone. Power calculations (GraphPad StatMate; GraphPad Software Inc) were based on data describing sedation of horses using butorphanol and detomidine (Taylor et al. 1988) and in donkeys comparing detomidine with detomidine/butorphanol (Joubert et al. 1999) and expecting buprenorphine and detomidine to provide a similar effect (Love et al. 2011a,b). A sample size of 50 in each group gave 80% power to detect a 20% difference in the proportion of cases in groups B and C scored as successful or unsuccessful, with $p < 0.05$, whilst still allowing for withdrawal of 10 cases in each group. Interim analysis of the data after 86 horses had completed the trial indicated that the null hypothesis should be rejected and the trial was stopped.

All data were analysed using GraphPad Prism software. Normally distributed data are presented as mean ± SD. Categorical data are presented as proportions within each category in each group. The primary outcome (success or failure) was analysed using Fisher's exact test. Other non parametric categorical data were analysed using Fisher's exact test for 2 × 2 contingency tables (requirement for second dose) or the Chi-squared test for larger tables (gender, breed, procedure). The Mann–Whitney *U* test was used to compare overall sedation and ataxia scores in each group; if a significant difference was indicated, the proportion of horses in each group awarded each score was compared using the

Chi-squared test for each time point. Within each group, changes in sedation and ataxia scores over time were examined using the Kruskall Wallis test (Freidman's test was not used as the group sizes were uneven); if a significant difference was indicated, Dunn's test was used to compare each time point with pre-sedation values, and to compare post-detomidine with post-BorG injection values. Single parametric data from each group (body weight, age) were compared using an unpaired *t*-test. Heart rate and respiratory rates in each group were compared using two way repeated measures ANOVA; no significant differences were detected, so no *post hoc* analyses were performed (less than five horses in each group were excluded due to missing data points). Within each group, changes in heart and respiratory rates over time were compared using one way repeated measures ANOVA; where a significant difference was indicated Dunnett's test was used to assess changes from pre-sedation baseline values, and Tukey's MCT was used to compare post-detomidine and post-BorG injection values. $p < 0.05$ was considered significant.

Results

Animals

Data from 84 horses were analysed ($n = 46$ group B, $n = 38$ group C). Two horses were rejected at recruitment, one due to respiratory disease and the other as it had been heavily sedated within the last seven days. Data on gender, breed, procedure, body weight and age are shown in Table 2. There were no significant differences between the groups in any of these variables.

Table 2 Mean ± SD body weight and age; and gender, breed and procedure performed in 84 horses after sedation with either detomidine and buprenorphine (group B) or detomidine and 5% glucose solution (group C)

Group B ($n = 46$)	Group C ($n = 38$)
Gender	
14 Mares	19 Mares
26 Geldings	15 Geldings
6 Entire male	4 Entire male
Breed	
7 Thoroughbred & Thoroughbred cross	9 Thoroughbred & Thoroughbred cross
6 Sport horse & Warmblood	8 Sport horse & Warmblood
15 Pony	11 Pony
8 Cob	5 Cob
Body weight	
10 Cross breed & others	5 Cross breed & others
498 ± 114 kg	496 ± 106 kg
Age	
10.5 ± 5.7 years (median 10)	5.6 ± 5.6 years (median 10)
Procedure performed	
10 Clipping	7 Clipping
3 Farriery	2 Farriery
9 Dentistry	3 Dentistry
17 Imaging*	15 Imaging*
7 Minor surgery with local anaesthesia	11 Minor surgery with local anaesthesia

*Imaging = radiography, ultrasound, MRI, endoscopy. No significant differences between groups: gender $p = 0.1847$, breed $p = 0.5964$, body weight $p = 0.9402$, age $p = 0.9382$, procedure $p = 0.4036$.

Primary outcome measure

The procedure was carried out successfully (primary outcome measure) in 42 horses in group B (91%) and in 24 horses (63%) in group C ($p = 0.0027$), therefore the null hypothesis was rejected and buprenorphine was shown to enhance the sedation produced by detomidine alone (Fig. 1).

A second dose of detomidine and BorG was required in 11 horses in group B (24%) and 12 in group C (32%) because of inadequacy of sedation at 10 minutes ($p = 0.6248$). The procedure was subsequently completed satisfactorily after the second dose in all horses in group B (outcome score 1 in six horses and 2 in five horses). However, five group C

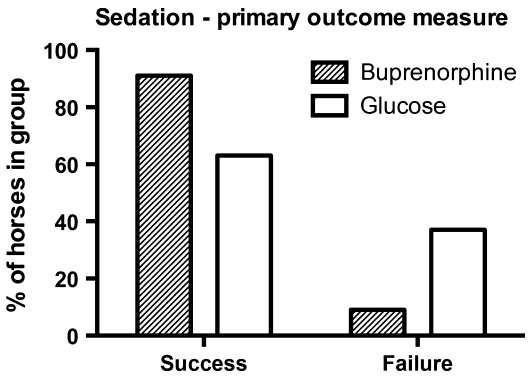

Figure 1 Primary outcome. Proportion of 84 horses after sedation with detomidine followed by buprenorphine ($n = 46$) or glucose ($n = 38$) with successful or failed completion of the procedure. Significant difference between the groups ($p = 0.0027$). Success = score 1 & 2, Failure = score 3 & 4.

horses requiring the second dose received an outcome score of 4, and the procedure was either abandoned or rescue sedation given. Two further horses had outcome score 3, three score 2 and two score 1. Rescue sedation was given to five horses in group C with either a further 5 μg kg^{-1} buprenorphine (two horses) or 20 μg kg^{-1} detomidine and 40 μg kg^{-1} butorphanol (two horses) or 50 μg kg^{-1} romifidine and 13 μg kg^{-1} butorphanol. Procedures were subsequently carried out successfully in all these five horses and there were no adverse effects.

Some additional doses of sedative were given outwith the protocol. Three horses, (two group B, one group C), were given a second dose of sedation more than 10 minutes after the first BorG injection because sedation was judged adequate at 10 minutes, but proved inadequate during the following 30–60 minutes. This allowed the procedure to be carried out satisfactorily (outcomes 1&2). A further horse in group B was given only detomidine for its second dose, which proved inadequate. This was the only horse in group B awarded an outcome score 4 and the procedure abandoned.

Sedation

Sedation developed immediately after injection of detomidine and increased further after buprenorphine but not after glucose injection. Sedation (scores significantly higher than pretreatment) lasted 60 minutes in group B and 45 minutes in group C. Overall sedation scores were higher in group B than group C ($p = 0.0086$). A greater proportion of the horses in group B were awarded higher sedation scores than in group C at 15, 30 and 45 minutes. At all other time points the differences were not significant (Fig. 2). Overall sedation in the horses requiring a second dose of detomidine and test drug at 10 minutes was not statistically different from the overall sedation in those not requiring a second dose in either group B ($p = 0.6043$) or group C ($p = 0.1459$).

Ataxia

Ataxia developed immediately after injection of detomidine and increased further after buprenorphine but not after glucose. Ataxia (scores significantly higher than pretreatment) lasted 60 minutes in group B and 30 minutes in group C. Overall ataxia scores were higher in group B than in group C ($p = 0.0005$). A greater proportion of the horses in group B were awarded higher ataxia scores than in group C at 15, 30 and 45 minutes after injection of the test substance. At all other time points the differences were not significant (Fig. 3). Overall ataxia in the horses requiring a second dose of detomidine and test drug at 10 minutes was not statistically different from the overall ataxia in those not requiring a second dose in either group B ($p = 0.4299$) or group C ($p = 0.7886$).

Heart and respiratory rate

Heart rate decreased after detomidine injection in both groups, and remained below pre-treatment values until 45 minutes post BorG injection in group B and until 30 minutes in group C (Fig. 4). Second degree AV block developed after detomidine injection in six horses in group B and two in group C. This persisted for no more than 15 minutes after BorG injection. There were no significant differences between the two groups in heart rate and no values were abnormally low or high.

Respiratory rate decreased after detomidine injection in group B and was below pre-treatment values from 30 to 120 minutes post buprenorphine injection (Fig. 5). Respiratory rate did not change significantly from pre treatment values in group C. There were no significant differences between the two groups and few values were abnormally low or high. Rates of 4 per minute were recorded in two horses in group B, one of these after detomidine, but before buprenorphine was given. All increased to normal values within a few minutes. A brief period (<3 minutes) of tachypnoea (65 minute) was recorded in one horse after buprenorphine.

Adverse events

There were no serious adverse events and few minor unwanted effects in either group. Stertorious breathing was noted in one group B horse which resolved as sedation abated. Sedation was shorter than desired in seven group C animals, and a twitch or physical restraint was used to complete the procedure in a further four cases (one group B, three group C). One horse in group B was so heavily sedated, or the analgesia so profound, that skin lacerations during clipping went unnoticed. Four cases in group B were initially too heavily sedated for farriery or precise foot placement for imaging. Two horses in group B were inadequately sedated after

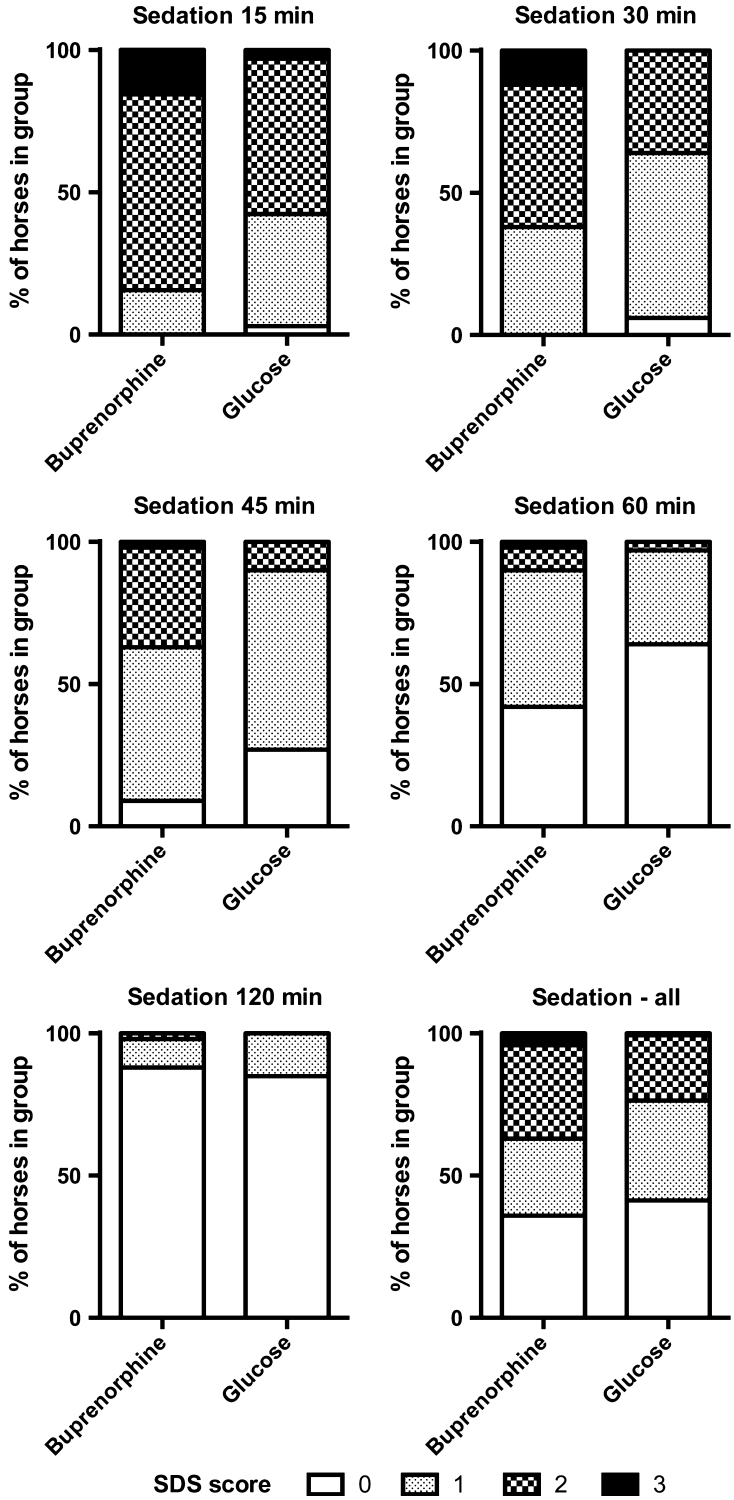

Figure 2 Distribution of simple descriptive scale (SDS) sedation scores in 84 horses after sedation with detomidine and buprenorphine (n = 46) or glucose (n = 38). Overall sedation scores were higher in the buprenorphine group (p = 0.0086) (Mann–Whitney U test). Groups were significantly different at 15 (p = 0.051), 30 (p = 0.029) and 45 (p = 0.034) minutes (min) after test substance injection (Chi squared test).

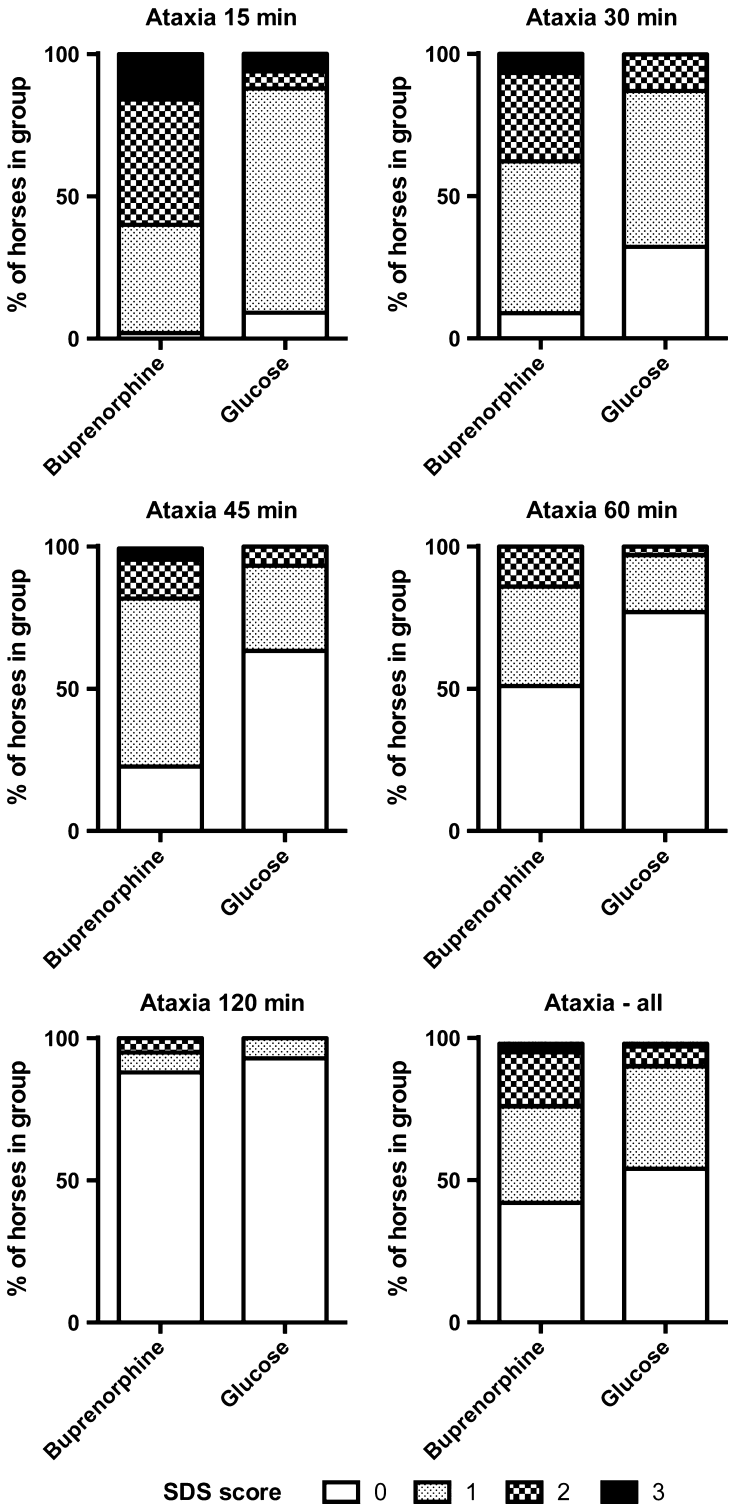

Figure 3 Distribution of simple descriptive scale (SDS) ataxia scores in 84 horses before and after sedation with detomidine and buprenorphine ($n = 46$) or glucose ($n = 38$). Overall ataxia scores were higher in the buprenorphine group ($p = 0.0005$) (Mann–Whitney U test). Groups were significantly different at 15 ($p = 0.0003$), 30 ($p = 0.0179$) and 45 ($p = 0.0049$) minutes (min) after test substance injection (Chi squared test).

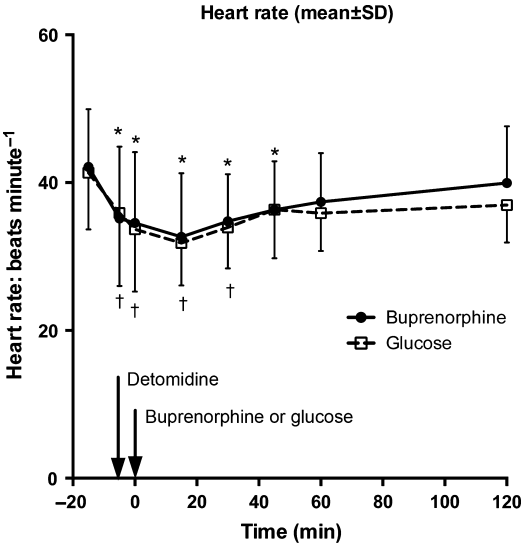

Figure 4 Mean ± SD heart rate in 84 horses sedated with detomidine and buprenorphine ($n = 46$) or glucose ($n = 38$). No significant difference between groups. * (buprenorphine group) and † (glucose group) = significant ($p < 0.05$) decrease from pre treatment value.

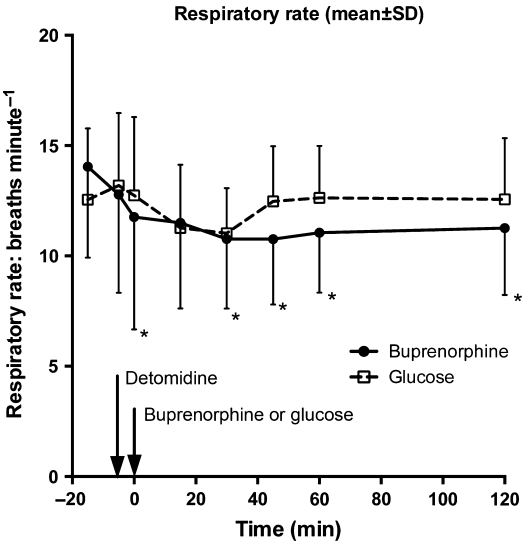

Figure 5 Mean ± SD respiratory rate in 84 horses sedated with detomidine and buprenorphine ($n = 46$) or glucose ($n = 38$). No significant difference between groups. * (buprenorphine) = significant ($p < 0.05$) decrease from pre treatment value. No significant change from pre-treatment value in the glucose group.

the first dose, became very ataxic after the second dose of detomidine and no further buprenorphine was given. The procedure was completed successfully in both cases.

Discussion

This field study demonstrated that buprenorphine enhanced the sedation provided by detomidine alone in common with the effects of other opioids used in this way (Clarke & Paton 1988; Taylor et al. 1988; Browning & Collins 1994). Longer procedures are often carried out with infusions of alpha$_2$ and opioid combinations (Hainisch 2001; Ringer et al. 2012a, b), and buprenorphine has been used in this way with detomidine infusion (van Dijk et al. 2003). However, the aim of our study was to evaluate the effect of adding buprenorphine to detomidine when given as a single injection rather than by infusion.

Some of the additional sedative effect seen with the inclusion of buprenorphine in group B may been related to better analgesia with the opioid. However, since analgesia was not specifically evaluated, the extent of such a contribution is unknown, and probably minimal, as most of the procedures were non painful.

The horses and ponies used in this investigation were drawn from first opinion general practice and were a fair representation of the UK equine population likely to be treated under standing sedation in routine equine practice. The two groups were well matched and individual variation should not have confounded the data. No attempt was made to score individual temperament, the environmental conditions or the invasiveness of the procedure. Whilst these may have affected the degree of sedation, the groups were equally represented in each clinic and differences between treatments would not be obscured.

The doses of detomidine and buprenorphine used in this investigation were based on both previous experience with other opioids used with alpha$_2$ adrenoceptor agonists (Taylor et al. 1988; Browning & Collins 1994) as well as on laboratory data from horses given buprenorphine with sedatives (Love et al. 2011a,b). These investigations, and studies of buprenorphine alone in horses (Carregaro et al. 2006, 2007; Love et al. 2012), suggested that doses of up to 10 μg kg^{-1} buprenorphine would lead to effective sedation when given with detomidine, without serious adverse effects. Love's studies (Love et al. 2011a,b) also indicated that sedation was improved if a larger dose of detomidine was given when higher doses of buprenorphine were used. In the present study, the initial 5 μg kg^{-1} buprenorphine with 10 μg kg^{-1} detomidine proved adequate in most cases. However, when more profound

sedation was required, taking the total buprenorphine dose to 10 μg kg^{-1}, it was preceded by a further dose of detomidine, taking its total to 20 μg kg^{-1}, as suggested by Love's data (Love et al. 2011b).

In the present study, the initial dose of 10 μg kg^{-1} detomidine and 5 μg kg^{-1} buprenorphine was sufficient for successful completion of the procedure in approximately three quarters of the cases. Addition of the second dose of detomidine and buprenorphine allowed the procedure to be carried out satisfactorily in all the remainder. The only failure in the buprenorphine group was a horse given only detomidine in the second dosing. The procedure was completed successfully in only around two-thirds of the glucose group after the first dose of detomidine, and after the second dosing there were still a few horses where the procedure was abandoned or rescue sedation was required. The data also indicate that the quality of sedation was better after buprenorphine.

Effective sedation lasted for 60 minutes in the buprenorphine group; additional sedation made little impact on the effective duration as detected by the sampling points. One hour of sedation is probably sufficient for most common clinical procedures carried out on standing horses. After sedation with detomidine only, the effective sedation was only 45 minutes, and this proved inadequate in several horses. The need for more sedation at 10 minutes appeared to be related to the individual horse and the proposed procedure, as overall sedation was not increased in horses given a second dose compared with those where one was sufficient.

Ataxia is always a potential hazard when standing sedation is used in horses. This occurred in both groups, but was more serious and long lasting after buprenorphine. No horses fell, but two became sufficiently ataxic after a second dose of detomidine that the second dose of buprenorphine was withheld. Second dosing of detomidine and BorG made little impact on the duration of ataxia as detected by the sampling points. After sedation with detomidine only, ataxia lasted only 30 minutes whereas several of the buprenorphine horses were still ataxic at 60 minutes. In previous laboratory studies (Love et al. 2011b) sedation was better when higher doses of both detomidine and buprenorphine were given than when lower doses of detomidine were used with higher doses of buprenorphine. However, the horses in those studies were standing in stocks which may have masked the ataxia. It is clear that, as with other opioids (Clarke & Paton 1988), buprenorphine will exacerbate the $alpha_2$ adrenoceptor agonist-induced ataxia, and care must be taken not to move a heavily sedated horse or it may fall. Overall ataxia within either group was not increased when a second dose was given, suggesting that ataxia was related more to the individual than to the actual dose of detomidine and buprenorphine.

There was little evidence of any serious cardiovascular or respiratory effects. Decreased heart rate is a well known effect of $alpha_2$ adrenoceptor agonist administration and this was not enhanced. Respiratory rate was lower in the buprenorphine group, but the values were within a normal range and this may simply reflect the degree of sedation and lack of any anxiety. Second degree atrioventricular block was seen in a few horses after the detomidine injection. This is a well recognised effect of $alpha_2$ adrenoceptor agonists (Clarke & Taylor 1986) and is not uncommon in normal undisturbed horses standing quietly. Transient tachypnoea was reported in one horse in the buprenorphine group. It is difficult to ascertain whether this was related to the buprenorphine as it occurred only once. Opioids cause locomotor activity and some stimulation in healthy, non sedated horses (Carregaro et al. 2007) and the transient tachypnoea may have been related to this. Alternatively, it may simply have been a response to the horse being startled as it recovered from sedation. Stertorious breathing was seen in one horse. This is not uncommonly seen with $alpha_2$ adrenoceptor agonist sedation as a result of the low head position causing fluid accumulation in the nasal mucous membranes. This can be alleviated by raising the horse's head to a more normal position (Taylor & Clarke 2007).

Buprenorphine appeared to perform in a similar manner to other opioids in enhancing sedation (Clarke & Paton 1988). Use of opioids in horses remains controversial because this class of drug may cause locomotor stimulation and decreased gastrointestinal motility (Mama et al. 1992; Taylor et al. 2002; Senior et al. 2006). As a mu-agonist opioid, buprenorphine presumably has similar potential. Locomotor and sympathetic stimulation and decreased abdominal auscultation scores were reported after 5–10 μg kg^{-1} buprenorphine (Carregaro et al. 2007; Love 2009; Love et al. 2011a,b). However, no signs of actual colic were reported, and locomotor stimulation is prevented when a sedative is given with the opioid. Opioids used for equine sedation do not appear to cause a significant problem

with characteristics such as box walking and post-treatment colic (Taylor et al. 1988; Browning & Collins 1994; Love et al. 2011a).

Buprenorphine, as a mu agonist opioid, has considerable analgesic potential. This has been demonstrated in horses under laboratory conditions where mechanical and thermal nociceptive thresholds were raised for several hours after 5–10 μg kg^{-1} buprenorphine (Love et al. 2012). Effective analgesia was also achieved with 10 μg kg^{-1} buprenorphine given prior to surgical castration under general anaesthesia (Taylor et al. 2008). Analgesic effect was not specifically investigated in the present study. However, the notable lack of response to clearly painful stimuli such as skin laceration observed in this study suggests that buprenorphine may be a particularly appropriate opioid for standing sedation in horses when a procedure known to be painful is undertaken.

In conclusion, buprenorphine 5 and 10 μg kg^{-1} enhanced the sedation produced by detomidine 10 and 20 μg kg^{-1}, enabling all procedures to be carried out successfully. There were no serious adverse effects but marked ataxia necessitates careful handling of the horse during peak sedation. Buprenorphine appears to be a suitable opioid for use to enhance $alpha_2$ adrenoceptor agonist sedation for clinical procedures in standing horses.

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Received 19 January 2013; accepted 14 February 2013.

