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Effects of Additional Premedication on Romifidine and Ketamine Anaesthesia in Horses

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Effects of additional premedication on romifidine and ketamine anaesthesia in horses. Acta vet. scand. 1996, 37, 315-325. – The clinical and cardiorespiratory effects of premedication with acepromazine, butorphanol or diazepam in addition to romifidine before induction of anaesthesia with ketamine were studied in 6 horses on 4 random occasions. Administration of romifidine alone or in combination with butorphanol resulted in an increase in arterial blood pressure, accompanied by a significant decrease in heart rate with second-degree atrio-ventricular heart block. Induction of anaesthesia with ketamine returned the heart rate to baseline value, but the arterial blood pressure was significantly increased compared to baseline. Including acepromazine in the premedication prevented the hypertension and bradycardia induced by romifidine. The respiratory rate was slightly decreased after premedication in all groups, but returned to the baseline value after induction of anaesthesia. Mild hypercapnia and significant hypoxaemia were observed during sedation and anaesthesia, reflecting an impairment of pulmonary function. Premedication with acepromazine before sedation with romifidine resulted in a fast induction and good anaesthesia. Inclusion of butorphanol in the premedication resulted in individual variation in the quality of induction and anaesthesia. Addition of diazepam to the sedation with romifidine resulted in good muscle relaxation with a smooth induction and maintenance of anaesthesia and an increased time before the horses responded to noxious stimuli, compared with romifidine and ketamine anaesthesia. All horses reached a standing position at the first attempt, but horses premedicated with diazepam in combination with romifidine showed mild ataxia after recovery.

acepromazine; butorphanol; diazepam.

Introduction

Dissociative anaesthesia with romifidine (0.1 mg/kg) and ketamine (2.2 mg/kg) for short-duration anaesthesia in horses has proved to be of good quality with excellent recoveries *(Marntell et al.* 1994). A recent study has shown that the cardiopulmonary effects of a combination of romifidine and ketamine are similar to those of a combination of xylazine and ketamine *(Kerr et al.* 1994). However, an increased recumbency time and improved muscle relaxation would be an advantage for routine equine surgical procedures in field practice. The principal aim of this study was to determine whether premedication with acepromazine, butorphanol or diazepam in addition to romifidine before induction of anaesthesia with ketamine would improve the anaesthesia without adverse effects on the cardiorespiratory function. A further aim was to assess the clinical effects of this additional premedication upon the anaesthetic

period, including the duration and quality of induction, anaesthesia and recovery.

Materials and methods

Six Standard bred trotters were studied. The group comprised 3 mares and 3 geldings, weighing 405-540 kg (mean 450) and with an age of 5-14 (mean 9) years. The horses were considered healthy on clinical examination.

Each horse served as its own control and was anaesthetised 4 times, each time with a different drug combination. The order of drug combinations was randomised and at least 7 days elapsed between the anaesthetic protocols. Food was withheld before anaesthesia, but access was given to straw bedding and water. On the basis of published literature and preliminary tests, the following drug combinations and dosages were chosen:

RK = romifidine 0.1 mg/kg iv (Sedivet®vet., 10 mg/ml Boehringer Ingelheim Vetmedica, Ingelheim am Rhein, Germany) + ketamine 2.2 mg/kg iv (Ketaminol® 100 mg/ml Veterinaria AG, Zurich, Schweiz).

RBK = romifidine 0.1 mg/kg i. + butorphanol 0.025 mg/kg iv (Torbugesic, 10 mg/ml, Fort Dodge Laboratories, Fort Dodge, Iowa, USA, 10 mg/ml) + ketamine 2.2 mg/kg iv.

RDK = romifidine 0.1 mg/kg iv + diazepam 0.05 mg/kg iv (Apozepam® , 5 mg/ml, Apothekernes Laboratorium, Oslo, Norway) + ketamine 2.2 mg/kg iv.

ARK = romifidine 0.1 mg/kg iv + acepromazine 0.025 mg/kg im (Plegicil®vet, 10 mg/ml, Pherovet AB, Malmö, Sweden) + ketamine 2.2 mg/kg iv.

Romifidine was injected 7 min before ketamine on every occasion. Butorphanol was injected intravenously 5 min before ketamine and thus 2 min after romifidine. Diazepam was administered intravenously immediately before ketamine. Acepromazine was given intramuscularly 23 min before romifidine and thus 30 min before ketamine. All drugs except acepromazine were administered through a catheter placed in the jugular vein.

All catheterizations were performed with the horse standing, unsedated and after local analgesia. An arterial catheter (18G/1.4×200 mm, Ohmeda, Swindon, UK) was introduced percutaneously into the transverse facial artery. A teflon catheter (G14, 105 mm, Intranyl, Vygon, Ecouen, France) was introduced percutaneously into a jugular vein.

Arterial blood pressure was measured invasively through the arterial catheter connected via a fluid-filled line to a pressure transducer (Baxter Medical AB, Eskilstuna, Sweden) positioned at the level of the scapulo-humeral joint in the conscious horse and at the level of the sternal manubrium during lateral recumbency. The pressure was recorded on an ink-jet recorder (Sirecust 730, Siemens-Elema, Solna, Sweden). A base-apex electrocardiogram was recorded from surface electrodes to obtain the heart rate and rhythm.

The respiratory rate was measured by observing the costo-abdominal movements. Arterial blood was obtained anaerobically and kept airtight in iced water for 30 min until arterial oxygen and carbon dioxide tensions (PaO_2, $PaCO_2$) were measured with a standard electrode technique (ABL 3, Radiometer, Copenhagen, Denmark).

The only response to noxious stimulus tested was that to pin-prick in the coronary band and in the skin on the legs and shoulder. The response was evaluated 5 min after ketamine injection and thereafter every 10th min during lateral recumbency. Any distinct body movement was recorded as response.

Measurements were initially made in the standing, unsedated horse, and were repeated 5 min after the sedation with romifidine. All horses were anaesthetised in a padded recovery room

with 1 person holding the halter during induction. All horses breathed room air spontaneously. The first samples during anaesthesia in lateral recumbency were taken 5 min after ketamine injection. Measurements were repeated every 10th minute until the horse assumed sternal recumbency. The final samples were taken in the standing horse approximately 5 min after recovery. The time from ketamine injection to lateral recumbency and to muscle relaxation, and the time spent in lateral recumbency and in sternal recumbency were recorded. The induction, anaesthesia, and recovery were assessed subjectively by 2 observers using a 0 to 3 scale, where 0 = poor, 1 = fair, 2 = good and 3 = very good.

All data are presented as mean values and standard error of the mean (SE). Two-way analysis of variance for repeated measures (ANOVA) was used to examine differences over time and between the different methods of anaesthesia. As some of the horses were in the sternal or standing position 25 or 35 min after induction of anaesthesia, these measurement times were excluded from ANOVA. If ANOVA indicated a significant ($p<0.05$) difference, further analysis was carried out using the Tukey HSD post hoc test to describe the patterns of differences as significant ($p<0.05$). The length of time from ketamine administration to lateral recumbency (induction), to muscle relaxation, to response to noxious stimuli and to standing, and the time spent in lateral and sternal recumbency were compared between the different anaesthetic protocols by the Wilcoxon rank-sum test.

Results

The haemodynamic results are presented in Figs. 1 and 2. The heart rate did not differ significantly between the RK, RBK, and RDK protocols at any time point. The heart rates in the horses given these 3 protocols followed the same pattern and decreased significantly from baseline 5 min after injection of romifidine (Fig. 1). The decreased heart rate was accompanied by periods of second-degree atrio-ventricular (A.V.) heart block in all horses. After the ketamine injection the heart rate increased and, except during RK after 15 min of anaesthesia, was no longer significantly different from baseline. During ARK the heart rate did not differ significantly during sedation and anaesthesia from baseline. Less frequent periods of A.V. heart blocks were noted after additional premedication with acepromazine.

The arterial blood pressure was significantly increased in RK, RBK, and RDK 5 min after ketamine injection and was also increased in RBK after 15 min of anaesthesia. During ARK arterial blood pressure did not change significantly during the anaesthetic period compared with baseline. There was a significant difference in the arterial blood pressure between ARK and RBK at 15 min of anaesthesia and between ARK and RDK at standing.

The respiratory rate and blood gas tensions are presented in Figs. 3 and 4. ANOVA indicated that the respiratory rate changed significantly with time, but not between the 4 anaesthetic protocols. The arterial oxygen tension decreased significantly during sedation and anaesthesia in lateral recumbency, but there were no significant differences between the drug combinations. A slight but significant increase in carbon dioxide tension during the anaesthetic periods was indicated by ANOVA, but no differences were found between anaesthetic protocols.

Anaesthetic periods

The observations during the anaesthetic period, including the duration and quality of induction, anaesthesia, and recovery are presented in Tables 1 and 2.

The induction time in the ARK protocol was

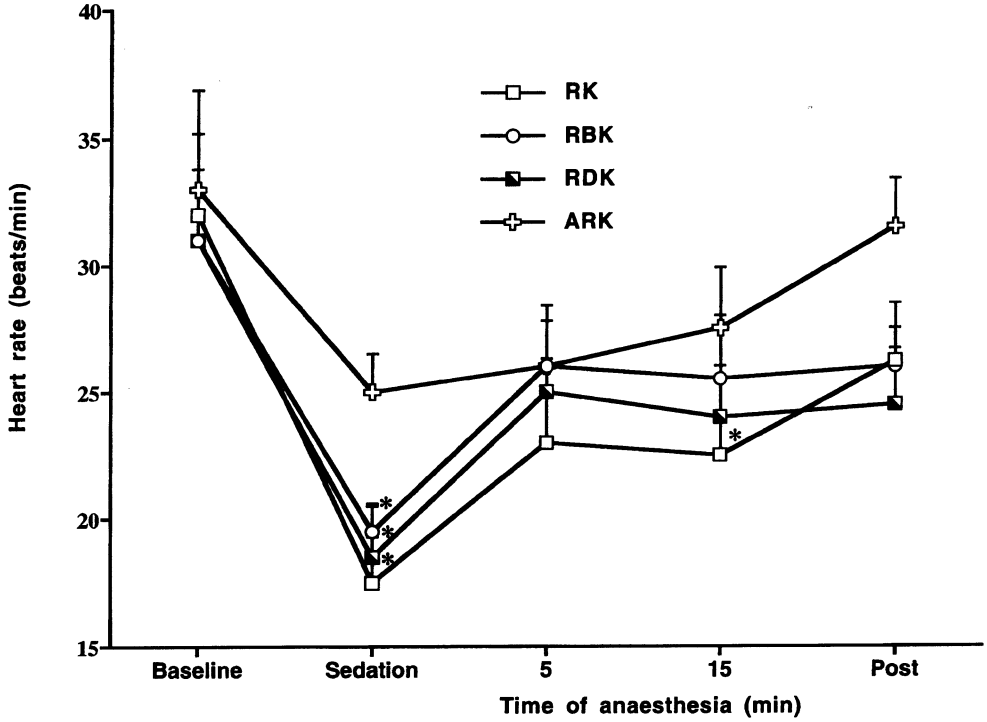

Figure 1. Heart rate in the standing, unsedated horses before anaesthesia (Baseline), 4 min after sedation with romifidine (Sedation), during anaesthesia in lateral recumbency (5 to 15 min) and 5 min after the horses had returned to a standing position (Post). Anaesthesia with romifidine and ketamine (RK), romifidine, butorphanol and ketamine (RBK), romifidine, diazepam and ketamine (RDK) and acepromazine, romifidine and ketamine (ARK). Data are presented as mean ± SE of 6 horses. * significantly different from baseline ($p<.05$).

significantly shorter than that with the other 3 combinations. Addition of diazepam immediately before the ketamine injection resulted in rapid and effective muscle relaxation, which in some cases was noted before recumbency was attained. Addition of butorphanol to the sedation with romifidine resulted in a more unpredictable induction, which was judged as poor in 2 horses and good in 4 horses.

The quality and length of anaesthesia after premedication with acepromazine were similar to those in RK, but the muscle relaxation was more pronounced in ARK and muscle twitching after induction of anaesthesia was less marked in these horses. Addition of diazepam to the romifidine/ketamine anaesthesia resulted in improved muscle relaxation. The length of time before the horses responded to the pinprick test was significantly longer during RDK than during RK and ARK. Individual differences in the quality of induction and maintenance of anaesthesia were seen with RBK.

The recoveries were calm, and all horses reached a standing position at the first attempt. The horses anaesthetised with RBK spent a significantly longer time in sternal recumbency. Horses premedicated with a combination including diazepam were slightly more ataxic af-

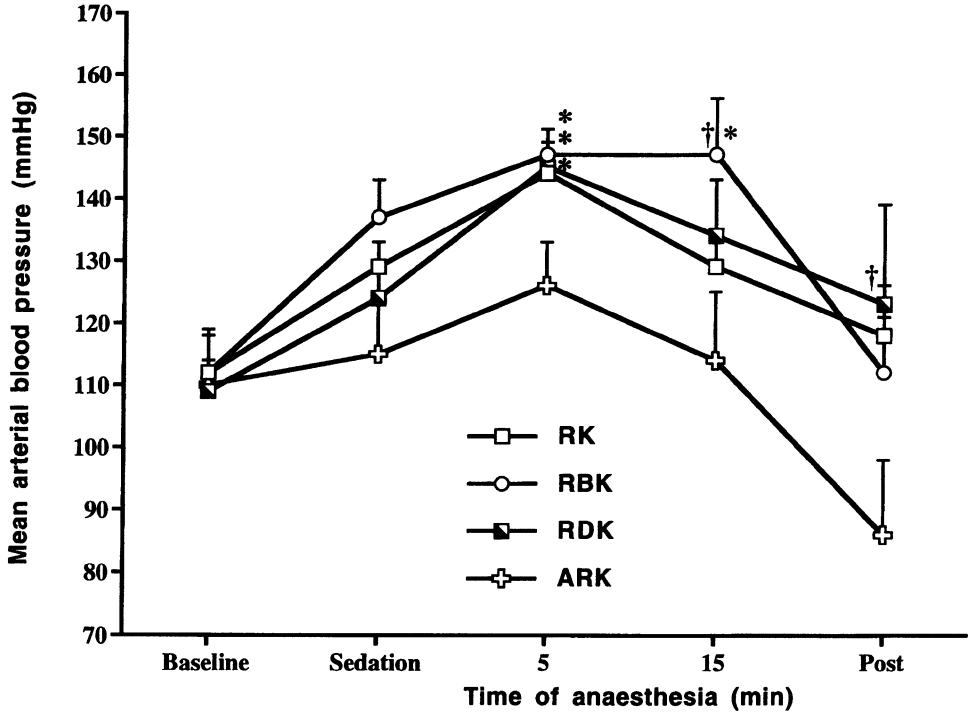

Figure 2. Mean arterial blood pressure. For abbreviations, see Fig. 1.* Significant difference from baseline ($p<.05$), † significantly different from and ARK ($p<.05$).

ter recovery. In RK, ARK and RBK all horses gained their feet without signs of ataxia.

Discussion

The present study demonstrates that additional preanaesthetic medication with acepromazine, butorphanol, or diazepam can modify the effects on the cardiorespiratory function and/or the clinical effects of short-term anaesthesia with romifidine and ketamine.

Profound sedation and cardiorespiratory depression have previously been reported after intravenous use of romifidine *(Gasthuys et al.* 1990, *Clarke et al.* 1991, *England et al.* 1992). Administration of romifidine in adult horses in the present study resulted in a significant decrease in heart rate accompanied by second-degree A.V. heart block. It has been suggested that initial vasoconstriction and increased systemic vascular resistance after sedation with $\alpha 2$-agonists may result in a reflex increase in parasympathetic tone with a concomitant decrease in heart rate and cardiac output *(Muir et al.* 1977, *Short* 1991, *Wagner et al.* 1991). Additional premedication with butorphanol or diazepam did not alter the influence of romifidine on the cardiorespiratory function. Administration of ketamine after premedication with romifidine alone or in combination with butorphanol or diazepam, returned the heart rate to the baseline values, but the arterial blood pressure was sig-

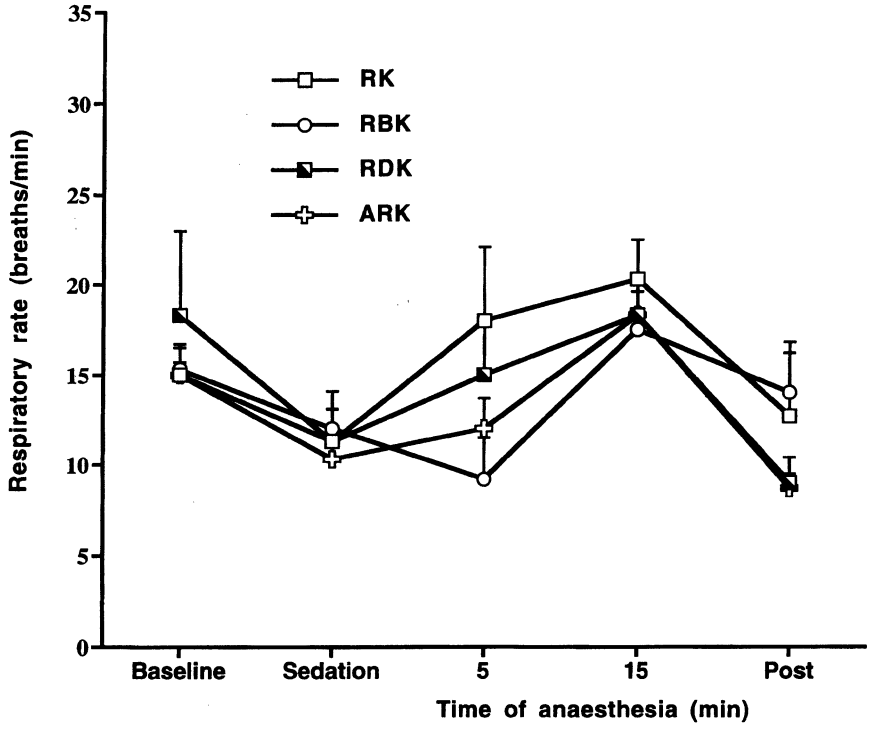

Figure 3. Respiratory rate. For abbreviations, see Fig. 1. * Significant difference from baseline ($p<.05$).

nificantly increased. Including acepromazine in the premedication in our study prevented the hypertension and bradycardia induced by romifidine. The benefits and dangers of using a combination of acepromazine and $\alpha 2$-agonists have been discussed *(Short* 1987, *Hall & Clarke* 1991). Veterinarians in field practice have observed more profound sedation when $\alpha 2$-agonists have been used in combination with acepromazine and *Nilsfors et al.* (1987) supported this finding. *Short* (1987) reported that high doses of both drugs in combination may increase the risk for severe hypotension. However, a recent report suggests that acepromazine could have a beneficial effect on cardiovascular functions when included in premedications with $\alpha 2$-agonists *(Grøndahl-Nielsen* 1992). In agreement with a previous observation *(Fisher* 1984), the induction of anaesthesia with ketamine in the present study was more rapid when acepromazine was used in combination with $\alpha 2$-agonists. A decrease in blood flow to the central nervous system after administration of $\alpha 2$-agonists have been observed *(Short* 1991). Since ketamine has a rapid distribution phase and because of its high lipid solubility it is absorbed quickly after administration *(Cohen & Trevor* 1974, *Waterman et al.* 1987). Consequently, a possible explanation for the faster induction of anaesthesia after a premedication including acepromazine could be a well maintained blood flow to the central nervous system. Our data do not include cardiac output, but there have been previous reports on

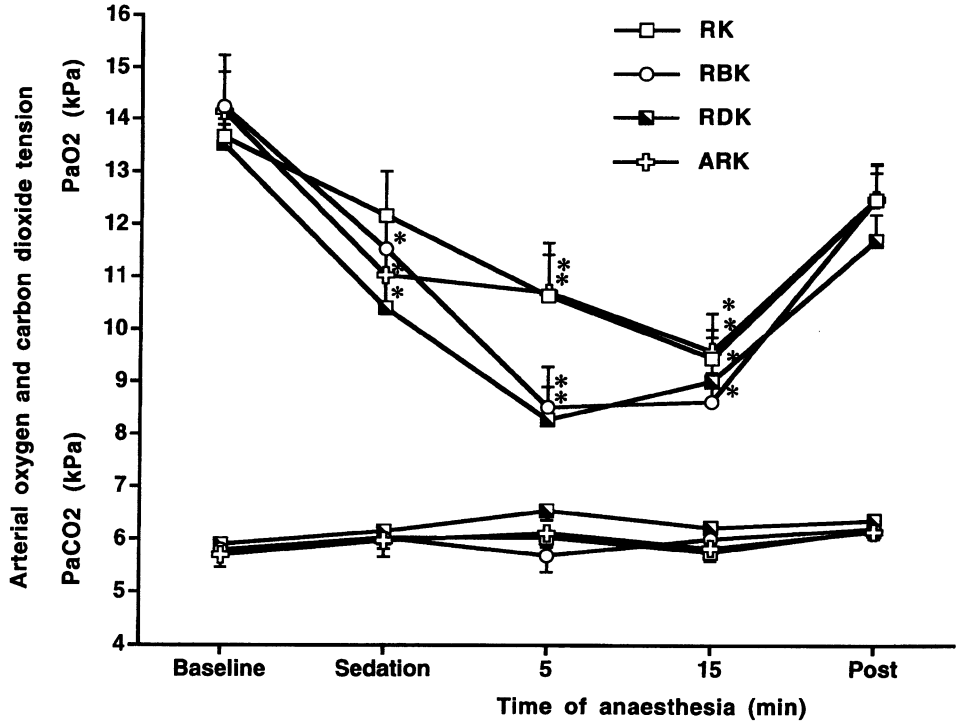

Figure 4. Arterial oxygen tension (PaO_2) and arterial carbon dioxide tension ($PaCO_2$). All horses breathed room air spontaneously. For abbreviations, see Fig. 1. * significantly different from baseline ($p<.05$).

a decrease in cardiac output and blood flow after premedication with other α2-agonists *(Muir et al.* 1977). From our data we suggest that premedication with a low dose of acepromazine seems to prevent some cardiovascular side effects commonly induced by romifidine/ketamine. However, it must be stressed that the horses included in this study were considered healthy and normovolemic, and the dose of acepromazine administered intramuscularly was considered low.

The capability of ketamine of maintaining the respiratory rate close to conscious values did not prevent gas exchange disturbances during dissociative anaesthesia in lateral recumbency. Although the respiratory rate returned to baseline values after induction of anaesthesia, a mild hypercapnia and significant hypoxaemia reflected impaired pulmonary function. The respiratory rate per min was not affected, but several animals showed periodic breathing, with breaths in quick successions followed by an apnoeic period of 15-30 sec, as observed earlier during ketamine anaesthesia *(Hall & Taylor* 1981, *Muir* 1991). Such a gas exchange disturbance has been reported to occur during ketamine anaesthesia in combination with other α2-agonists *(Muir et al.* 1977, *Hall & Taylor* 1981). No further attempts were made to evaluate the mechanism of the impaired pulmonary function in this study.

A second aim of the present study was to assess the clinical effects of additional premedication upon the anaesthetic period, including the dura-

Table 1. Clinical effects of short-term anaesthesia with romifidine and ketamine (RK), romifidine, butorphanol and ketamine (RBK) romifidine, diazepam and ketamine (RDK) and acepromazine, romifidine and ketamine (ARK). The length of time from ketamine administration to lateral recumbency (induction), to muscle relaxation, to reaction to pin-prick and to standing and the time spent in lateral and sternal recumbency are given. Data are presented as mean ± SE. * significantly different from all other combinations. † significantly different from RK and ARK.

Drug combination	Time to Induction (sec)	Time to Muscle-relaxation (min)	Time to Pinprick response (min)	Time spent in lateral recumbency (min)	Time spent in sternal recumbency (min)	Time to Standing (min)
RK	118±10	3.5±0.5	18±6	27±2	8±2	37±4
RBK	96±4	3.9±2.5	27±3	32±3	19±5 *	51±7
RDK	103±8	1.9±0.3 *	31±2 †	38±4	6±2	45±5
ARK	74±6 *	2.8±0.4	22±2	32±2	7±3	39±4

tion and quality of induction, anaesthesia and recovery. Controlled and smooth induction of anaesthesia is important, particularly under field conditions *(Matthews & Hartsfield* 1993). Addition of diazepam to the sedation with romifidine resulted in good muscle relaxation with smooth induction and maintenance of anaesthesia. However, a disadvantage observed after premedication with diazepam was that in some horses muscle relaxation occurred before lateral recumbency, sometimes resulting in an abrupt fall. The largest variation of induction was seen after premedication including butorphanol. One horse staggered around for 15 sec, had excessive muscle stiffness and expressed excitation during the first min of lateral recumbency. Another horse receiving butorphanol could hardly be controlled by 1 person as it moved strongly forwards, but the induction of anaesthesia was acceptable once the ketamine was administered. *Tranquilli et al.* (1983) described hyperresponsiveness in one out of 4 horses after additional butorphanol premedication, but the only disadvantage reported by *Matthews et al.* (1991) was slightly prolonged induction in some horses receiving this drug. Even though the dose used in our study was relatively low compared with other reports, the variation in behaviour during induction may be attributed to the opiate effect.

The anaesthesia, as judged subjectively, was regarded as very good when diazepam was included, and good with both RK and ARK (Table 2). Inclusion of butorphanol in the premedication resulted in the largest variation in the anaesthetic quality in our study, where 3 of these horses showed muscle twitching during anaesthesia and one horse had leg movements. The only attempt to evaluate response to noxious stimulus in this study was to a pin-prick test. Additional premedication with diazepam resulted in an increased length of time before the horses responded to noxious stimulus compared with RK and ARK.

The period of recovery from short-term anaesthesia in horses is a critical stage *(Brouwer* 1985). It is well known that preanaesthetic medication can prolong the recovery period *(Brock & Hildebrand* 1990, *Matthews et al.* 1991). Also, it has been reported that other $\alpha 2$-agonists may influence the quality of recovery differently *(Clark et al.* 1986, *Matthews et al.* 1991). We observed an increased length of time before standing in horses receiving additional premedication (Table 1). Interestingly and somewhat unexpectedly, all horses achieved a

Table 2. Mean score ± SE for induction, anaesthesia and recovery judged subjectively on a 0 to 3 scale, where 0 = poor, 1 = fair, 2 = good and 3 = very good. For abbreviations, see Table 1.

Drug combination	Induction	Anaesthesia	Recovery	Total score
RK	1.5±0.2	2.0±0.3	3.0±0.0	6.5
RBK	1.3±0.2	1.8±0.5	3.0±0.0	6.1
RDK	2.2±0.3	3.0±0.0	2.5±0.3	7.7
ARK	2.2±0.3	2.2±0.2	3.0±0.0	7.4

standing position on the initial attempt, irrespective of the type of premedication and of the recumbency time in the sternal position. It has been suggested that occasional poor recoveries after anaesthesia with detomidine/ketamine, compared to xylazine/ketamine may be caused by the longer length of action of detomidine *(-Clarke et al.* 1986). Although the sedative effect of romifidine has been reported to be of longer duration of sedation than that of xylazine and detomidine *(England et al.* 1992), very stable recoveries were seen after premedication with romifidine. There seems reason to speculate whether other properties of romifidine, such as a different specificity and selectivity for $\alpha 2$-receptors, could modify the cardiorespiratory and behavioural responses. Also, the equipotency of different $\alpha 2$-agonists has been evaluated more from a clinical than a physiologic standpoint. Additional premedication with butorphanol resulted in a significantly longer time spent in sternal recumbency. One mare, which always rose immediately from the lateral to the standing position without resting in sternal recumbency, rested for 12 min in the sternal position after receiving butorphanol. Horses premedicated with diazepam in combination with romifidine showed mild ataxia after recovery, probably due to the muscle-relaxant effects of diazepam. The mare that rose immediately from the lateral to the standing position was ataxic and weak in the hindquarters, but remained standing on the first attempt.

We conclude that additional premedication with acepromazine, butorphanol, or diazepam may be used in combination with romifidine and ketamine for short term anaesthesia in the horse. Both acepromazine and diazepam improved the quality of induction and anaesthesia, while addition of butorphanol to the premedication resulted in individual variations.

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Sammanfattning

Effekter av kompletterande premedicinering vid anestesi av häst med romifidin och ketamin.

Kliniska och kardiorespiratoriska effekter av sedering med romifidin enbart eller i kombination med kompletterande premedicinering med acepromazin, butorphanol eller diazepam inför induktion av dissociativ anestesi med ketamin studerades hos 6 hästar vid fyra olika tillfällen. Vid samtliga tillfällen sederades hästarna med romifidin 0.1 mg/kg intravenöst (iv) och när kompletterande premedicinering gavs utgjordes denna av acepromazin 0.025 mg/kg (im) 23 min före romifidin, butorphanol 0.025 mg/kg (iv) 2 min efter romifidin eller diazepam 0.05 mg/kg (iv) 7 min efter romifidin omedelbart före ketamin. Sederingskombinationerna gavs i slumpmässig ordning och hästarna placerades i sidoläge under anestesin. Administrering av romifidin enbart eller i kombination med butorphanol resulterade i en stegring av det arteriella blodtrycket, åtföljt av bradykardi med förekomst av andra gradens atrioventrikulära hjärtblock. Efter induktion av dissociativ anestesi med ketamin 2.2 mg/kg (iv) steg hjärtfrekvensen till referensvärdets nivå, men det arteriella blodtrycket ökade signifikant jämfört med referensvärdet som registrerats på stående, osederad häst. När acepromazin inkluderades i premedicineringen registrerades varken hypertension eller bradykardi efter sedering. Minskad andningsfrekvens registrerades efter samtliga preme-

dicineringar men andningsfrekvensen ökade till referensvärdets nivå efter induktion av anestesin. Mild hypercapni och signifikant hypoxemi observerades under både sedering och anestesi vilket speglar den försämrade lungfunktionen. Anestesin bedömdes som bra med kombinationen av romifidin och ketamin. Kompletterande premedicinering med acepromazin resulterade i en snabb induktion och bra anestesi. Kompletterande premedicineringen med butorphanol resulterade i den största individuella variationen beträffande induktionens och anestesins duration och kvalitet. Kompletterande premedicinering med diazepam resulterade i god muskelrelaxering och en smidig induktion och underhåll av anestesin. Reaktionen på kanylstick i form viljemässiga avvärjningsrörelser återkom senare när diazepam var inkluderad i premedicineringen jämfört med sedering enbart med romifidin. Alla hästar inkluderade i studien reste sig på första försöket oavsett premedicinering, men hästarna visade lindrig ataxi efter resningen när diazepam varit inkluderad i premedicineringen.

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