

The cardiovascular and respiratory effects of medetomidine and thiopentone anaesthesia in dogs breathing at an altitude of 1486 m

K E Joubert^{a†} and R Lobetti^{b§}

ABSTRACT

The purpose of this study was to evaluate the cardio-respiratory effects of the combination of medetomidine and thiopentone followed by reversal with atipamezole as a combination for anaesthesia in 10 healthy German Shepherd dogs breathing spontaneously in a room at an altitude of 1486 m above sea level with an ambient air pressure of 651 mmHg. After the placement of intravenous and intra-arterial catheters, baseline samples were collected. Medetomidine (0.010 mg/kg) was administered intravenously and blood pressure and heart rate were recorded every minute for 5 minutes. Thiopentone was then slowly administered until intubation conditions were ideal. An endotracheal tube was placed and the dogs breathed room air spontaneously. Blood pressure, pulse oximetry, respiratory and heart rate, capnography, blood gas analysis and arterial lactate were performed or recorded every 10 minutes for the duration of the trial. Thiopentone was administered to maintain anaesthesia. After 60 minutes, atipamezole (0.025 mg/kg) was given intramuscularly. Data were recorded for the next 30 minutes. A dose of 8.7 mg/kg of thiopentone was required to anaesthetise the dogs after the administration of 0.010 mg/kg of medetomidine. Heart rate decreased from 96.7 at baseline to 38.5 5 minutes after the administration of medetomidine ($P < 0.05$). Heart rate then increased with the administration of thiopentone to 103.2 ($P < 0.05$). Blood pressure increased from 169.4/86.2 mmHg to 253.2/143.0 mmHg 5 minutes after the administration of medetomidine ($P < 0.05$). Blood pressure then slowly returned towards normal. Heart rate and blood pressure returned to baseline values after the administration of atipamezole. Arterial oxygen tension decreased from baseline levels (84.1 mmHg) to 57.8 mmHg after the administration of medetomidine and thiopentone ($P < 0.05$). This was accompanied by arterial desaturation from 94.7 to 79.7 % ($P < 0.05$). A decrease in respiratory rate from 71.8 bpm to 12.2 bpm was seen during the same period. Respiratory rates slowly increased over the next hour to 27.0 bpm and a further increase to 51.4 bpm after the administration of atipamezole was seen ($P < 0.05$). This was maintained until the end of the observation period. Arterial oxygen tension slowly returned towards normal over the observation period. No significant changes in blood lactate were seen. No correlation was found between arterial saturation as determined by blood gas analysis and pulse oximetry. Recovery after the administration of atipamezole was rapid (5.9 minutes). In healthy dogs, anaesthesia can be maintained with a combination of medetomidine and thiopentone, significant anaesthetic sparing effects have been noted and recovery from anaesthesia is not unduly delayed. Hypoxaemia may be problematic. Appropriate monitoring should be done and oxygen supplementation and ventilatory support should be available. A poor correlation between SpO_2 and SaO_2 and $ETCO_2$ and $PaCO_2$ was found.

Key words: cardiovascular, dog, medetomidine, respiratory, spontaneous ventilation, thiopentone.

Joubert K E, Lobetti R L **The cardiovascular and respiratory effects of medetomidine and thiopentone anaesthesia in dogs breathing at an altitude of 1486 m.** *Journal of the South African Veterinary Association* (2002) 73(3): 104–110 (En.). Section of Anaesthesiology, Department of Companion Animal Clinical Studies, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

INTRODUCTION

Medetomidine has been used as a premedication for thiopentone anaesthesia^{1,29}. It has been recommended that medetomidine should not be used in critical patients, as the lowered dose of thiopentone does not necessarily balance

the negative effects of medetomidine on the cardiovascular and respiratory systems¹. In dogs sedated with medetomidine and breathing 50 % oxygen, it was demonstrated that an increase in alveolar-arterial oxygen difference occurred¹. Dogs breathing room air may have an

increased risk of hypoxaemia.

Medetomidine and thiopentone are used in private practice, either on their own or in combination, for routine procedures such as radiographs, gastroscopy, and minor surgical procedures. Supplemental oxygen is seldom given under these conditions. There are reports of hypoxia, cyanosis and acute cardiovascular collapse as well as speculation that this combination may be contra-indicated in the dog^{1,8,29}. There are few reported studies of the use of medetomidine and thiopentone in combination.

The purpose of this study was to evaluate the cardiovascular and respiratory effects of the combination of medetomidine and thiopentone as well the reversal with atipamezole as an anaesthetic protocol in the healthy dog breathing spontaneously in a room.

MATERIALS AND METHODS

The Ethics and Research Committees of the Faculty of Veterinary Science, University of Pretoria, and the South African National Defence Force (SANDF) approved this study. It was conducted at an altitude of 1486 m above sea level at an ambient air pressure of 651 mmHg (range 648–655) with a FiO_2 of 20.98 %. Ten healthy German Shepherd dogs (7 males, 3 females) from the SANDF were used in this study. The mean age was 14.5 months and the mean weight was 27.5 kg.

The right cephalic vein and pedal artery were aseptically prepared and a 20G intravenous catheter (Jelco, Johnson and Johnson, Halfway House) was inserted into the cephalic vein and a 22G intra-arterial catheter (Jelco) was inserted into the pedal artery. After the catheters were positioned, the dog was placed in left lateral recumbency and allowed to calm down. The blood pressure transducer was calibrated to a standard mercury column and the pressure transducer was adjusted to the height of the sternum before baseline samples were collected for heart and respiratory rates, blood pressure, blood gas analysis and arterial lactate. Baseline samples were collected prior to the administration of 0.010 mg/kg of medetomidine (Domitor, Novartis Animal Health, Isando) intravenously

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Received: February: 2002. Accepted: July 2002.

through the cephalic catheter and the catheter was flushed with 5 ml of heparin-saline (Heparin, Novo-Nordisk) (10 IU/ml). Blood pressure, temperature and heart rate (Propaq Protocol Systems, Beaverton, Oregon) were monitored continuously and recorded every minute for 5 minutes. Heart rate and rhythm were assessed from the blood pressure tracing. After 5 minutes, a 5% thiopentone solution (Intraval, Rhône-Poulenc Animal Health, Halfway House) was given intravenously in 1-ml increments every 30 seconds until the eyeball rotated downwards, jaw tone allowed for endotracheal intubation and the pedal reflex was just present. This was considered to represent the point of induction of anaesthesia and the total dose of thiopentone used up to this time was considered the induction dose. Once anaesthetised, a pulse oximetry probe (Satelite Plus, Datex Instrumentarium Corp., Helsinki) was placed on the tongue and an endotracheal tube tonged to ensure a patent airway and to allow for attachment of the capnograph sampling line. The dogs were allowed to breathe room air spontaneously. Blood pressure, pulse oximetry, respiratory and heart rate, end tidal CO₂ (ETCO₂) (Capnomac II, Datex Instrumentarium Corp., Helsinki) were monitored continuously. Blood gas (ABL 300, Radiometer, Copenhagen), and lactate (AcuSport, Boehrering Mannheim, Mannheim) analysis was performed every 10 minutes. The PaO₂, PaCO₂, pH and haemoglobin concentration were measured while arterial oxygen content and arterial saturation were calculated. These values were recorded every 10 minutes for the duration of the trial. Anaesthesia was maintained with thiopentone for 60 minutes. Additional increments (1-ml bolus doses) were given to maintain the desired level of anaesthesia. Anaesthesia was considered inadequate if the palpebral and pedal reflexes and a moderate jaw tone were present. The depth of anaesthesia was assessed 60 seconds after the administration of an additional bolus dose and if the depth of anaesthesia was not similar to that at intubation, an additional dose of thiopentone was given. This data was recorded separately to the induction dose and if an additional dose was required within 60 seconds, the combined dose was recorded. After 60 minutes, atipamezole (25 µg/kg) (Antisedan, Novartis Animal Health, Isando) was given intramuscularly. Data was recorded every 10 minutes for an additional 30 minutes. SpO₂ and ETCO₂ values were not recorded or analysed during the last 30-minute period, as values were lost due to the activity of the dogs as they recovered.

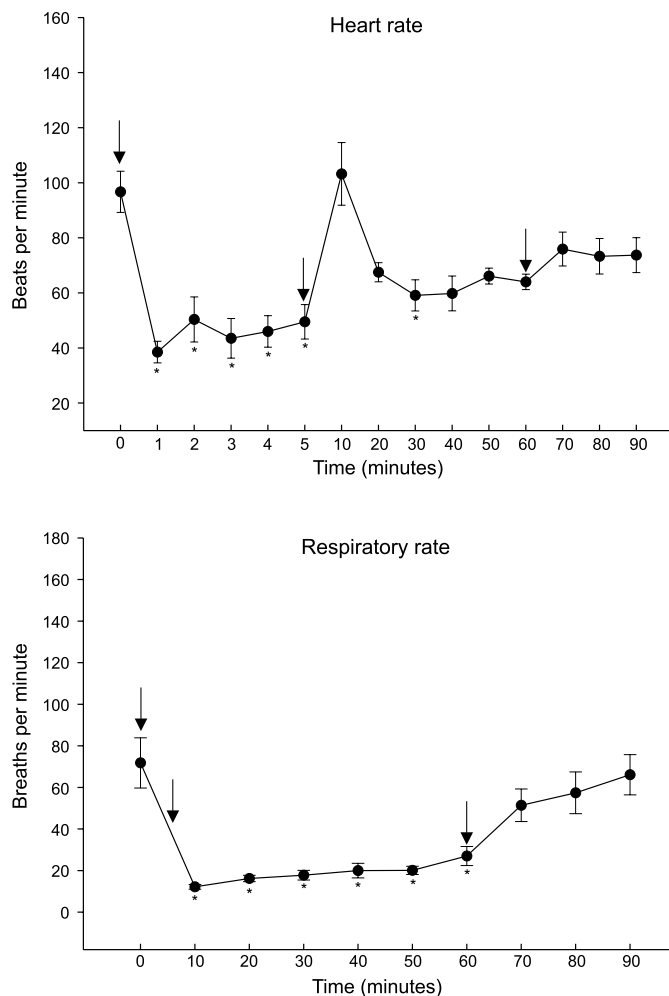


Fig. 1: Heart and respiratory rates. Data presented as mean with error bars representing standard error of mean. The 1st arrow indicates the administration of medetomidine, the 2nd arrow indicates the administration of thiopentone and the 3rd arrow the administration of atipamezole. Statistical significance from baseline is indicated by asterisks.

Time from administration of atipamezole to the 1st attempt at sternal recumbency was recorded as the recovery time.

Arterial blood samples were collected in heparinised syringes (Heparin, Centaur Laboratories, Isando) from the intra-arterial catheter. Blood gas and lactate samples were analysed immediately. Temperature (Propaq Protocol Systems, Beaverton, Oregon) was monitored continuously with a thermocouple placed in the oesophagus over the heart after the induction of anaesthesia. The temperature was used to correct blood gas values.

Data analysis

Data were tabulated in a spreadsheet (Excel, Microsoft Corp.). Statistical analysis was performed with SigmaStat (Jandel Corp.) and SigmaPlot (SPSS Incorp.). Descriptive statistics were used to summarise the data. Repeated measures analysis of variance with Dunn's method for pairwise comparison was used to test for statistical differences between time intervals when data were not normally distributed (mean blood pressure, heart

rates, arterial O₂ saturation, blood pH, arterial O₂ content, and pulse oximetry O₂ saturation). For normally distributed data, the repeated measures of analysis of variance with the Tukey test were used (respiratory rate, systolic and diastolic blood pressure, PaO₂, PaCO₂). Statistical significance was set at $P < 0.05$. The data at baseline and the time intervals at the administration of the various agents were compared for statistical significance.

RESULTS

The mean dose of thiopentone to induce the desired level of anaesthesia was 8.7 ± 0.35 mg/kg. The time to the 1st additional dose of thiopentone was 30 ± 2.5 minutes. Additional doses of 2.1 ± 0.16 mg/kg of thiopentone were given every 11.1 ± 1.14 minutes to maintain the desired level of anaesthesia. Two dogs (25 kg, 28 kg) required the administration of 2 bolus doses of thiopentone within 60 seconds of each other during the maintenance phase of anaesthesia. An additional 6.1 ± 0.58 mg/kg of thiopentone was required during the 60-minute period to maintain

anaesthesia. In total, 15.4 ± 0.94 mg/kg was used. The mean time to recovery was 5.9 ± 0.55 minutes after atipamezole administration.

Before the administration of medetomidine, the mean heart rate was 96.7 ± 7.5 beats per minute. Post-medetomidine, there was a marked decrease in heart rate (38.5 ± 3.9), which increased after thiopentone induction (103.2 ± 11.4) and then decreased towards values obtained after medetomidine administration and remained stable until the end of the study (Fig. 1). There was no statistical difference between baseline readings and those obtained after the administration of thiopentone except for the reading at 30 minutes. Seven dogs demonstrated atrio-ventricular or sino-atrial blocks after the administration of medetomidine. Two dogs showed tachycardia (heart rate >180 bpm) after the administration of thiopentone.

Before the administration of medetomidine, the mean systolic blood pressure was 169.4 ± 11.6 mmHg; the mean diastolic blood pressure was 86.2 ± 2.9 mmHg; and the mean blood pressure was 109.5 ± 11.9 mmHg. Post-medetomidine, the systolic pressure increased to a mean of 253.2 ± 14.6 mmHg; the diastolic pressure increased to 143.0 ± 7.9 mmHg; and the mean pressure increased to 180.1 ± 11.1 mmHg (Fig. 2).

After thiopentone induction the systolic pressure decreased to a mean of 248.2 ± 10.9 mmHg; diastolic pressure increased to 152.1 ± 4.6 mmHg; and the mean blood pressure decreased to 181.7 ± 6.4 mmHg (Fig. 2). After the administration of thiopentone, blood pressure tended to return towards baseline levels. With the systolic, diastolic and mean blood pressures there was a statistically significant difference before and after medetomidine administration.

The baseline mean respiratory rate was 71.8 breaths per minute. The lowest respiratory rate was 36 while other dogs were panting at this time. Post-medetomidine, there was a marked decrease in the respiratory rate (12.2 ± 1.1). Respiratory rates slowly increased over the next hour to 27.0 ± 4.9 bpm and a further increase to 51.4 ± 7.9 bpm after the administration of atipamezole was seen (Fig. 1). Respiratory rates were statistically significantly depressed after the administration of medetomidine until the administration of atipamezole. Two animals showed induction apnoea after the administration of thiopentone with spontaneous respiration resuming within 2 minutes.

There was no statistically significant change in arterial lactate for the duration of the study (Fig. 3). However, some of the

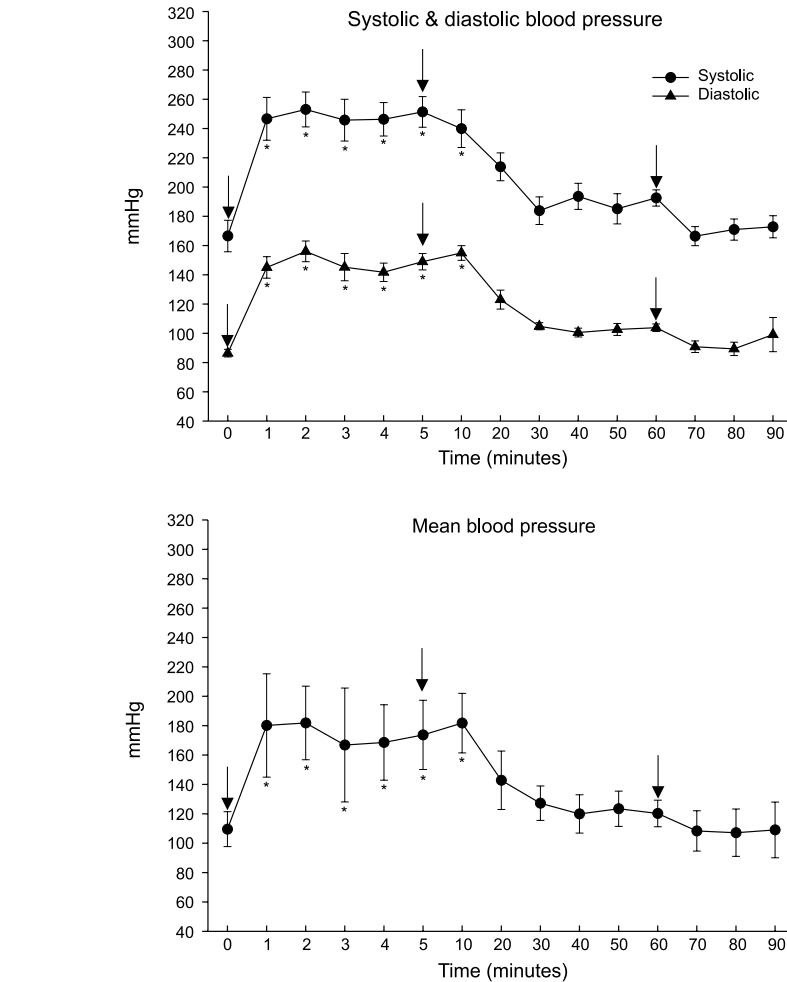


Fig. 2: Systolic, diastolic and mean blood pressures. Data presented as mean with error bars representing standard error of mean. The 1st arrow indicates the administration of medetomidine, the 2nd arrow indicates the administration of thiopentone and the 3rd arrow the administration of atipamezole. Statistical significance from baseline is indicated by asterisks.

dogs did show an increase in lactate after 20, 30 and 50 minutes. The mean lactate level was 2.8 ± 0.15 mmol/l.

Before the administration of medetomidine, the mean pH was 7.404 ± 0.007 .

Post-medetomidine, there was a marked decrease in the pH (7.354 ± 0.01), which was statistically significant. After thiopentone induction the pH gradually increased to a mean of 7.380 at 60 minutes.

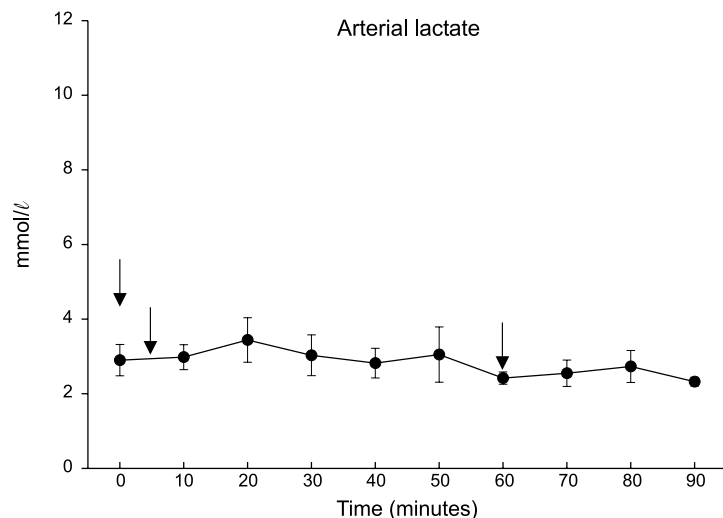


Fig. 3: Arterial blood lactate levels. Data presented as mean with error bars representing standard error of mean. The 1st arrow indicates the administration of medetomidine, the 2nd arrow indicates the administration of thiopentone and the 3rd arrow the administration of atipamezole. Statistical significance from baseline is indicated by asterisks.

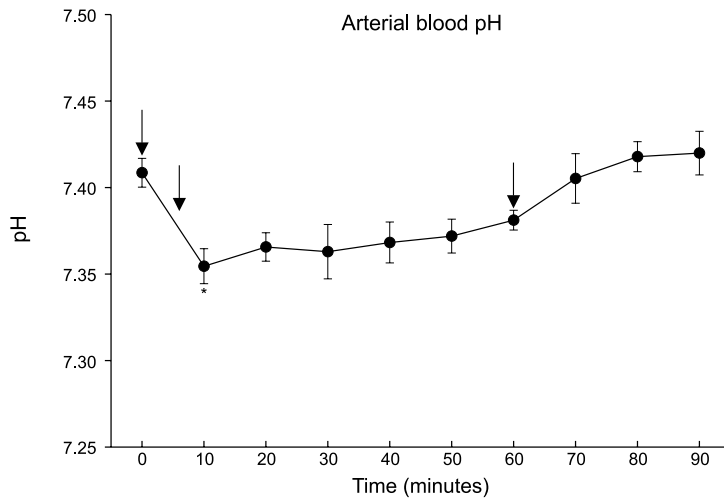


Fig. 4: Arterial blood pH. Data presented as mean with error bars representing standard error of mean. The 1st arrow indicates the administration of medetomidine, the 2nd arrow indicates the administration of thiopentone and the 3rd arrow the administration of atipamezole. Statistical significance from baseline is indicated by an asterisk.

After atipamezole administration, the pH increased and remained stable until the end of the study (Fig. 4). There was a statistically significant difference before and after medetomidine but not after the atipamezole administration.

Before the administration of medetomidine, the mean PaO₂ was 84.1 ± 1.4 mmHg. Post-medetomidine, there was a marked decrease in the PaO₂ (57.8 ± 4.1 mmHg). After thiopentone induction the PaO₂ increased and remained stable. After atipamezole administration, PaO₂ increased and remained high until the end of the study (Fig. 5). There was a statistically significant difference before and after medetomidine and atipamezole administration.

Before the administration of medetomidine, the mean PaCO₂ was 30.2 ± 1.1 mmHg. Post-medetomidine, there

was an increase in the PaCO₂ (37.9 ± 1.3 mmHg). After thiopentone induction the PaCO₂ decreased and remained stable. After atipamezole administration, PaCO₂ decreased and remained stable until the end of the study (Fig. 5). There was a statistically significant difference before and after medetomidine administration but not after the atipamezole administration.

After induction and intubation, the mean ETCO₂ was 41.9 ± 1.8 mmHg. The ETCO₂ values fluctuated during the anaesthesia time. At extubation, the mean ETCO₂ was 34.3 ± 2.3 mmHg. There was no statistically significant difference between the readings during the anaesthesia. There was no correlation between ETCO₂ and PaCO₂ in this study.

Before the administration of medetomidine, the mean SaO₂ was 94.7 ± 0.3 %.

Post-medetomidine there was a marked decrease in the SaO₂ (79.7 ± 4.4 %). After thiopentone induction the SaO₂ increased to a mean of 89.5 % and remained stable. After atipamezole administration, the SaO₂ transiently dropped before increasing until the end of the study. There was a statistically significant difference before and after medetomidine administration. The arterial O₂ content mimicked the SaO₂. SaO₂ increased statistically after the administration of atipamezole from values obtained at 10–40 minutes after the administration of medetomidine and thiopentone. Arterial oxygen content was not statistically significantly different for the duration of the study. The arterial oxygen content immediately after the administration of medetomidine and thiopentone was lower than all other readings obtained (17.4 ± 0.99 vol/%) (Fig. 6).

After induction and intubation, the mean pulse oximetry saturation was 87.6 ± 2.7 % and was associated in 3 dogs with cyanosis. This gradually increased during anaesthesia to a mean of 93.4 ± 1.2 %. There was no statistically significant difference between the readings during the anaesthesia. There was also no correlation between the pulse oximetry readings and arterial O₂ saturation.

DISCUSSION

This study demonstrated that the combination of medetomidine and thiopentone could be used in healthy dogs, with the most severe adverse effects being on arterial oxygen status. The standard recommended doses of thiopentone for the induction of anaesthesia is 10–12 mg/kg. Anaesthetic time can be extended for 10–20 minutes with the additional administration of 18–20 mg/kg of thiopentone in dogs when thiopentone is used as the sole anaesthetic agent²⁶. However, larger doses of thiopentone will saturate peripheral compartments and delay recovery. With premedication, the induction dose of thiopentone may be reduced to 8mg/kg²⁶. Medetomidine has been shown to have a dose-dependent barbiturate sparing effect^{8,29}. This study showed that a mean dose of 8.7 mg/kg thiopentone was required to induce anaesthesia and that the pedal and palpebral reflexes returned and the jaw tone increased 31 minutes after induction of anaesthesia. The total dose of thiopentone used during the 60 minutes of anaesthesia did not exceed the recommended maximum dose of 30 mg/kg. The concentration of thiopentone in the blood is dependent on cardiac output and the rate of injection¹². A high cardiac output results in rapid dilution (mixing) of the

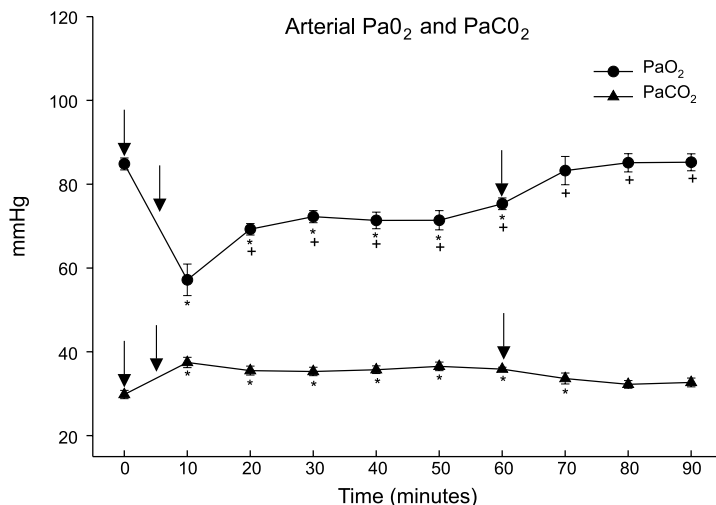


Fig. 5: Arterial PaO₂ and PaCO₂. Data presented as mean with error bars representing standard error of mean. The 1st arrow indicates the administration of medetomidine, the 2nd arrow indicates the administration of thiopentone and the 3rd arrow the administration of atipamezole. Statistical significance from baseline is indicated by asterisks, and from the 10 minute time interval by plus signs.

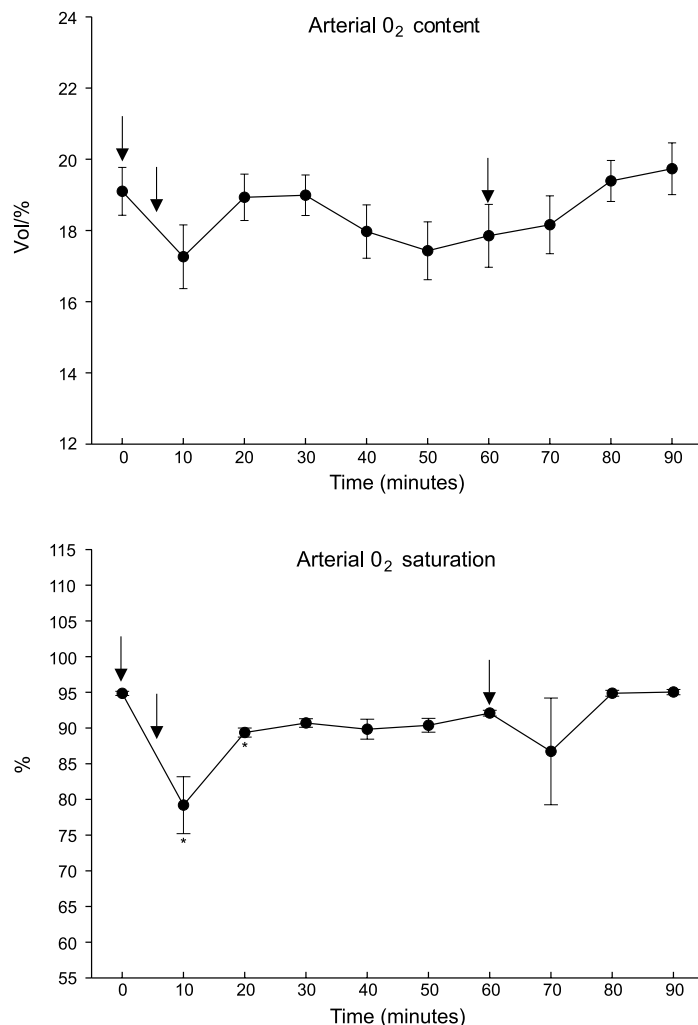


Fig. 6: Arterial oxygen content and oxygen saturation. Data presented as mean with error bars representing standard error of mean. The 1st arrow indicates the administration of medetomidine, the 2nd arrow indicates the administration of thiopentone and the 3rd arrow the administration of atipamezole. Statistical significance from baseline is indicated by asterisks.

bolus dose administered while poor perfusion tends to reduce mixing, resulting in higher concentrations. As medetomidine is responsible for vasoconstriction^{15,22,27} and a drop in cardiac output¹⁸, anaesthesia should be slowly titrated to effect after the administration of medetomidine. The pharmacokinetic effects of medetomidine may, in part, explain the reduction in anaesthetic requirements. A delay in induction of barbiturate anaesthesia has been reported after premedication with medetomidine, which has been attributed to the reduction in cardiac output caused by medetomidine²⁹. A pharmacodynamics interaction is also present but less well characterised^{18,29}. Recovery from thiopentone anaesthesia is usually the result of redistribution, with metabolism and elimination playing a smaller role.²⁵ The recovery from medetomidine-thiopentone anaesthesia may be delayed due to vasoconstriction, a reduction in cardiac output, or altered organ blood flow¹⁴ decreasing redistribution.

This results in longer sleeping times after the induction of anaesthesia⁸. Atipamezole reverses the cardiovascular effects of medetomidine and may improve redistribution of thiopentone and hence enhance recovery^{11,19,28,29}. In our study, a lower dose of atipamezole was used to reverse the anaesthesia in view of the metabolism/redistribution of medetomidine that occurred during the 60 minutes before its administration.

The 2 dogs (25 kg, 28 kg) requiring 2 bolus doses of thiopentone within 1 minute to maintain anaesthesia had initially received the lowest dose of thiopentone for induction of anaesthesia (7.41 mg/kg, 6.8 mg/kg). These dogs also had the shortest time to receive their 1st additional dose of thiopentone (22 minutes, 13 minutes). In all likelihood, these dogs were in a lighter plane of anaesthesia and required more thiopentone to return to the desired plane of anaesthesia.

Atrioventricular or sino-atrial heart blocks and bradycardia associated with

medetomidine administration and ventricular tachycardia associated with the administration of thiopentone were observed. As the blood pressure curve was used for the determination of arrhythmias, a definitive diagnosis of either atrioventricular or sino-atrial block was not possible. Arrhythmias have been reported with the use of both thiopentone (sinus and ventricular tachycardia, ventricular bigeminy, extrasystole and ventricular fibrillations^{2,17} and medetomidine (sino-atrial and atrio-ventricular blocks, bradycardia)²⁷. The mechanism of action of the medetomidine-induced heart block includes a baroreceptor response to initial hypertension following its administration and a central decrease in sympathetic tone^{22,27}.

The cardiovascular effects observed include a marked rise in blood pressure after the administration of medetomidine. Medetomidine binds to peripheral α_2 -receptors, increasing peripheral vascular resistance^{15,22,27}, which is followed by a compensatory, mild hypotension due to a notable decrease in heart rate and reduction in sympathetic tone^{15,22}. An increase in heart rate and hypertension was seen after the administration of thiopentone. Sympathetic tone and catecholamines are increased during induction of anaesthesia with thiopentone, which results in an increased heart rate and blood pressure¹⁷. Vascular resistance may still be high because of the effect of medetomidine. Mean blood pressures as high as 175–210 mmHg have been recorded after premedication with medetomidine and atropine followed by a thiopentone induction¹. Similar blood pressure changes have been reported for medetomidine-propofol anaesthesia without the administration of atropine¹⁹. Atropine is contra-indicated with the use of α_2 agonists due to the tachycardia, hypertension and arrhythmias that result⁶. Blood pressure has been shown to decrease to below baseline levels within 10 minutes of administration of medetomidine alone¹⁸. The tachycardia associated with the administration of thiopentone may be responsible for the maintenance of hypertension for a longer period than expected with the administration of medetomidine alone.

Hypoxaemia, as noted by a decrease in arterial oxygen tension, saturation and oxygen content, was observed during this study. Hypoxaemia may result from ventilatory failure, an increase in ventilation/perfusion mismatching, shunting and a decrease in inspired oxygen content. Medetomidine has been shown to have little effect on respiratory minute volume^{23,27}. Barbiturate-induced respira-

tory depression that is characterised by a decrease in tidal volume and respiratory rate²¹. Respiratory rates have been shown to decrease with medetomidine and thiopentone¹. However, after the administration of medetomidine, the partial pressure of oxygen has been shown to decrease along with a rise in blood pH^{22,23}. The pH rise is not always accompanied by a rise in partial pressure of carbon dioxide, indicating that a respiratory acidosis is unlikely²². The lower than expected baseline readings of PaCO₂ in this study could be ascribed to the fact that the dogs were initially excited with higher than normal respiratory rates resulting in an increase in alveolar ventilation with a reduction in PaCO₂. There was not a clinically significant increase in carbon dioxide tension in this study despite a reduction in respiratory rate indicating that hypoventilation is an unlikely cause of the hypoxaemia. However, dexmedetomidine has been shown to reduce metabolic rate^{14,15}. A reduction in metabolic rate would reduce carbon dioxide production and reduce ventilation due to normal homeostatic mechanisms. No correlation was found between ETCO₂ and PaCO₂ in this study. An initial lack of correlation was ascribed to panting at the beginning of the study. The maintenance of normal PaCO₂ levels in the body is dependent on CO₂ production, alveolar ventilation and perfusion of the lungs. Medetomidine and thiopentone have notable effects on these variables and hence it is difficult to evaluate which component was responsible for the lack of correlation. It probably is due to a combination of parameters.

Cyanotic and pale mucous membranes have been reported in 25 % of dogs premedicated with medetomidine as part of an induction protocol with either propofol or ketamine¹³. The arterial partial pressure of oxygen has been shown to decrease from 90 mmHg to 78.8 mmHg in dogs under medetomidine-propofol anaesthesia¹⁹. The cyanosis observed under medetomidine-propofol anaesthesia has been ascribed to venous desaturation and not arterial desaturation¹⁹. A decrease in arterial oxygen tension has been reported under medetomidine-thiopentone anaesthesia in dogs from 97.5 mmHg to 60–82.5 mmHg, which then slowly returned to normal¹. A similar reduction in arterial oxygen tension has been reported in dogs following the administration of xylazine and xylazine-ketamine (97.8 to 69.9 mmHg)¹⁰. Altitude reduces the partial pressure of oxygen available in the inspired air, as the proportion of oxygen available (20.98 %) remains constant, while the barometric pressure decreases. At our altitude, the

inspired partial pressure of oxygen would be 126.5 mmHg compared with 149.7 mmHg at sea level. The reduced inspiration percentage may have played a role in the hypoxaemia observed. Hypoxaemia, as reported in this study and with other alpha₂ agonists, appears to be a common event following the administration of alpha₂ agonists.

Causes of hypoxaemia that have not been excluded include changes in the alveolar-capillary unit, a reduction in cardiac output and an increase in shunt fractions. Lung oedema has been demonstrated in sheep following the administration of alpha₂ agonists³⁻⁵. Lung oedema has not been demonstrated in dogs.

Both the cardiac output and stroke volume decrease in dogs with increasing doses of medetomidine¹⁵ indicating that hypoperfusion is present. A reduction in blood flow to the skin, kidneys, spleen and muscle has been demonstrated in dogs¹⁴. A net decrease in oxygen transport has been noted in dogs after the administration of xylazine or xylazine-ketamine and this has been ascribed to a reduction in cardiac output¹⁰. Venous desaturation results from increased oxygen extraction from blood. A reduction in mixed venous saturation in response to increasing doses of medetomidine has also been reported in dogs²². The above studies indicate that hypoperfusion may be present following the administration of medetomidine. Lactate has been used as an indicator of hypoperfusion⁷. In the present study no changes in arterial lactate concentration were evident. Dexmedetomidine administered to dogs is known to reduce myocardial and total body oxygen consumption¹⁴. The reduction in oxygen demand is paralleled by a reduction in blood lactate^{14,15}. In these studies, mixed venous extraction increased in spite of a reduction in oxygen consumption¹⁴, which is most likely the result of a reduction in blood flow.

A lack of correlation between SpO₂ and SaO₂ was shown in this study. A pulse oximeter is most accurate when haemoglobin saturation is between 80–95 %⁹. Several of the readings were below the level of accuracy of pulse oximeters and may have resulted in the poor correlation. A good correlation between pulse oximetry and arterial saturation has been demonstrated in pentobarbital-anaesthetised dogs²⁰. Vasoconstriction, reduced blood flow and hypotension are associated with poor performance of pulse oximeters^{9,16,24}. A pulse oximeter may not be useful when medetomidine and thiopentone are used as the peripheral pulses may be weak in spite of the central hypertension, and the combination of

medetomidine and thiopentone is known to induce vasoconstriction^{15,17,23,27,28} and reduce cutaneous blood flow¹⁴.

CONCLUSION

This study has shown that in healthy dogs, anaesthesia can be maintained with a combination of medetomidine and thiopentone but there is a significant risk of hypoxaemia. Appropriate monitoring is required and oxygen supplementation and ventilatory support should be available. Anaesthetic requirements are reduced and recovery from anaesthesia is not delayed. The cardiovascular effects observed during this study are the result of the effects of thiopentone and medetomidine and have been previously separately described for the drugs. A reduction in respiratory rate, PaO₂ and pH were seen. Blood lactate values did not change during the study. No correlation was found between SpO₂ and SaO₂ and ETCO₂ and PaCO₂.

ACKNOWLEDGEMENTS

We thank the South African National Defence Force for funding the study, the Roodeplaat Military Clinic for the use of their facilities and the staff of the Roodeplaat Military Clinic for invaluable assistance.

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