Cardiopulmonary effects of Detomidine-Propofol and Ketamine administration in the Donkeys

A.A. Amin and M. S. Mohammed
College of Veterinary Medicine\ University of Baghdad

Abstract

The effects of detomidine-propofol and ketamine on the heart rate, respiratory rate, blood gases and temperature were investigated in six healthy donkeys weighting from(100-150kg). The animals received premedication with detomidine HCL (10 µg/kg bwtiv) followed after 5 min with a single intravenous (i.v.) bolus dose of propofol (1mg/kg/bwt iv) and approximately 2 to 3 min by a single i.v. bolus dose of ketamine HCL (2.2 mg/kg bwt iv). All parameters were evaluated before the administration of detomidine and at 5,10, 15,20,25and 30 min after administration of detomidine and anesthetics.The heart rate decreased significantly after administration of the anesthetic drugs(propofol and ketamine). The mean respiratory rate decreased significantly after administration of the detomidine and anesthetic drugs (propofol and ketamine). PaO2 was littleeffect. The body temperature slightly decreased at all points in time. The data showed that the combination of detomidine-ketamine,propofol caused an inhibition of the cardiovascular system. This combination is responsible for little distributed ventilation, very small effect PaO_2 and a declined in body temperature in the anaesthesia period in donkeys.

تأثير أعطاء الديتوميدنوالبروبوفول والكيتامين على الجهاز القلبي الوعائي والتنفسي في الحمير

أياد عبد الجبار أمين ومعاذ سالم محمد
كلية الطب البيطري/ جامعة بغداد

الخلاصة

تهدف هذه الدراسة تقييم تأثير أعطاء عقار الديتومدين والبروبوفولول الكيتامين على الجهاز القلبي الوعائي والجهاز التنفسي في الحمير حيث أجريت الدراسة على ستة حيوانات بالغة يتراوح وزنها بين (100–150) كغم. أعطيت الحيوانات عن طريق الحقن الوريدي الديتومدين بجرعة 10 مايكروغرام/ كغم وزن الجسم وبعد خمسة دقائق أعطيت البروبوفول بجرعة 1 ملي غرام وزن الجسم ثم بعد 2–3 دقيقة كحد أقصى أعطيه الكيتامين بجرعة 2.2 ملي غرام/ كغم وزن الجسم. أخذت المعايير السريرية (ضربات القلب ومعدل التنفس ودرجات الحرارة ومعدل الأوكسجين بالدم) قبل الحقن أظهرت النتائج الإحصائية وجود انخفاض معنوي في معدل ضربات القلب في الأوقات 10، 15 و20 دقيقة وجود انخفاض معنوي في معدل التنفس في الأوقات 5، 10، 15، 20 و25 دقيقة بعد التخدير. بينما كان هناك نقصان بسيط في درجات الحرارة ومستوى الأوكسجين في الدم بعد التخدير. ومن النتائج يمكن استنتاج أن هذه الأدوية تسبب تثبيط الجهاز القلبي الوعائي والجهاز التنفسي وتأثير بسيط على درجات الحرارة ومستوى الأوكسجين في الدم خلال فترة التخدير.

Introduction

Equine practitioners are often required to perform surgical procedures under field conditions and although these surgical procedures are often similar to those performed in a hospital setting, management of general anesthesia may be quite different (1). There is no available anaesthetic drug which can provide proper anesthesia alone now a day. Therefore, combinations of sedatives and other anesthetics have been widely used in

animal practice. The anesthetic combination should congregate different characteristics, having adequate sedation and a deep unconsciousness state, without greatly changing the patient's physiologic parameters (2). General anesthesia is needed for certain surgical procedures, which are otherwise cannot be performed under regional and local anesthesia (3). Detomidine HCl (Domosedan) is a potent sedative and analgesic for use in veterinary practice (3). Detomidine has similar effects to xylazine but detomidine produces sedation and analgesia of a greater magnitude and a longer duration than xylazine Sedation effects become apparent within two to five minutes after intravenous injection (4). Propofol, an alkyl phenol derivative (2, 6¬ di-isopropyl phenol). The present formulation consists of 1 per cent propofol in intralipid, a parental nutritional agent consisting of 10 per cent soybean oil, 2.25 per cent glycerol and 1.2 per cent egg Lecithin. It has a pH of 7 and appears as a slightly viscous milky white substance (5). Propofol has several mechanisms of action, both through potentiating of gamma-aminobutyric acid (GABA) receptor activity, thereby slowing the channel closing time, and also acting as a sodium channel blocker (6, 7, 8). Ketamine is the most commonly used agent to anesthetize not only cat and dogs but also birds, horses, and exotic species. It has a rapid onset of action after IV or IM administration. This is a result of its high lipid solubility which allows quick entry into brain tissue. it can be administered repeatedly to maintain anesthesia (9).

Materials and Methods

The present study was carried out on 6 adult donkeys. The animals were healthy and their body weights were ranged from 120-150 kg respectively. All animals were fasted for about 12 hours and freely given water. The dose of each anesthetic drug was calculated. The effect of the regimen on the heart and respiratory rates and the body temperature as well as SPO_2. Were also measured and tabulated. They were recorded before injection (0.0 time) for control data and at 5, 10, 15, 20, 25 and 30 minutes after injection. The animals received premedication with detomidine HCL (10 µg/kg bwtiv) followed after 5 min with a single intravenous (i.v.) bolus dose of propofol (1mg/kg/bwt iv) and approximately 2 to 3 min by a single i.v. bolus dose of ketamine HCL (2.2 mg/kg bwt iv) (10). The heart rate (beat/min) and the body temperature (c^o) as well as SPO_2 Were measured by patient monitor (Omni II Touch Screen), the Respiratory rate (breath/ min) was counted by Movement of the chest and abdominal muscle.

Result and Discussion

The statistical analysis revealed significant decreased in heart rate started five minutes after injection of detomidine and gradually decreased after injection of propofol and ketamine, lasted up to 25 min. Heat rate returned to normal ($P<0.05$) (Table1). These results agreed with the result of other researches which attributed to the Detomidine cause heart rate rapidly declines initially within the first minute and an atrioventricular or sinoatrial block often accompanies the bradycardia (11). The bradycardia may be mediated through an increase in parasympathetic and a decrease in sympathetic tone over the heart due to effect of Detomidine (12). The heart decreased may because Propofol reduce blood pressure and cardiac output by lowering systemic vascular resistance in addition to this direct reduction in the systemic vascular resistance, propofol causes a blunting of the barorecepter reflex thus preventing compensatory tachycardia (13, 14). In addition to this direct reduction in the systemic vascular resistance, propofol causes a blunting of the barorecepter reflex thus preventing compensatory tachycardia (15). The effect of ketamine due to up take of the heart to catecholamine done direct vasodilatation of vascular smooth muscle which lead to increase heart rate and mean arterial blood pressure (16). This effect of ketamine not appeared may be due to effect of detomidine and propofol on the heart rate This result were in agreement with (17) and with (18)who used detomidine and ketamine in ponies.

Table (1) Effect of anesthesia (detomidine + propofol + ketamine) on heart rate (beat/ minute)

Time	Zero (control)	5 min	10 min	15 min	20 min	25 min	30 min
Effect	A 42.00 ±0.51	AB 41.00 ±0.81	B 39.83 ±0.31	B 37.00 ±0.51	B 38.16 ±0.54	AB 41.00 ±0.57	A 41.83 ±0.30

L.S.D=1.4

Different capital letters referred to the significant differences (P<0.05).

The respiratory rate gradually decreased through the first five minutes after injection of detomidine, and graduallydecreased after injection of the propofol and the ketamine and return to normal. The statistical analysis revealed significant differences among the zero time and 5, 10, 15, 20 and 25 times (P<0.05) (Table 2). Using detomidine may causerelaxation of the laryngeal and nasal alar muscles predisposes horses to upper airway obstruction and strider and respiratory rate is reduced Airways relaxation is a result of the presynaptic inhibition of acetylcholine release from cholinergic nerves in airways, which is mediated by stimulation of α2- adrenoceptors (19). Ketamine differs from most other anesthesia in that it does not depress ventilator responses to hypoxia. Skeletal muscle tone is maintained or even increased; thus arterial oxygenation and functional residual capacity are usually well maintained during ketamine anesthesia (5, 20). This result showed agreement with (17) who used ketamine in horses and with (21) using propofol in horseand with (18) who used detomidine and ketamine in horses.

Table (2) Effect of anesthesia (detomidine + propofol + ketamine) on respiratory rate (breath/minute)

Time	Zero (control)	5min	10min	15min	20min	25min	30min
Effect	A 20.83 ±0.47	B 17.83 ±0.54	B 15.83 ±0.40	B 14.00 ±0.61	B 14.66 ±0.66	B 17.16 ±0.65	A 20.66 ±0.33

L.S.D=1.5

Different capital letters referred to the significant differences (P<0.05).

Body temperature was mildly decreased during the first five minutes (37.13) c^{o} after injection of detomidine, continuously decreased after injection of propofol and ketamine. The statistical analysis revealed no significant different among the zero time and other times (P<0.05) (Table 3). The body temperature of the animals, showed a decrease in body temperature recorded following systemic administration of detomidine, this result was in agreed with results of other research which may be attributed to the effect of detomidine which causes depression of hypothalamic thermoregulatory center, and probably the result of reduced basal metabolic rate and muscle activity (22). The results agree with the results of Ketamine and other dissociative anesthetics which cause hypothermia by releasing monoamines responsible for centrally mediated hypothermia by inhibiting endogenous release of norepinephrine (23). Body temperature and Heart rate significantly decrease after propofol injection. (24). The body temperature showed significant decrease and this decrease Was evidenced by shivering of all animals of this group, this is similar to the finding of (25) in donkeys. Data of body temperature of the animals agree with other observations used detomidine, with anesthetic doses cause reduce in basal metabolic rate resulting in lowered body temperature (26). Body temperature and heart rate significantly decrease after propofol injection (24).The body temperature showed significant decrease and this decrease was similar to the finding of (25) in donkeys.

Table (3) Effect of anesthesia (detomidine + propofol + ketamine) on body temperature (c^o)

Time	Zero (control)	5 min	10 min	15 min	20 min	25 min	30 min
Effect	A 37.25±0.05	A 37.13±0.09	A 36.75±0.15	A 36.55±0.13	A 36.48±0.13	A 36.26±0.14	A 36.180.13

L.S.D=0.34

Different capital letters referred to the significant differences (P<0.05).

There was a mildly decreased in SPO_2 during the first five minutes followed the injection of detomidine, continuously decreased after injection of propofol and ketamine. The statistical analysis revealed no significant different among the zero time and other times (P<0.05) (Table 4).Use of detomidine cause reduced in the arterial partial pressure of oxygen. This reduction has not been associated with any clinical symptoms but arterial hypoxemia is possible (4). Propofol alone (27) and in combination with detomidine-butorphanol (28). Increases the mean $paCO_2$ and decreases the mean paO_2 in different species of animals. Hypoxemia with decrease in paO_2 is produced in ponies (29) The ketamine did not cause significant differences on blood gases this is agreement with (30). The initial mild decreased followed by an increase in PaO2 was attributed to the short-lasting depressant effect of ketamine (31). This result agreed with (17).

Table (4) Effect of anesthesia (detomidine+ propofol+ ketamine) on SPO_2%

Time	Zero (control)	5min	10min	15min	20min	25min	30min
Effect	A 92.83 ±2.44	A 92.50 ±1.85	A 91.50 ±2.11	A 90.16 ±1.88	A 92.16 ±1.13	A 95.16 ±1.81	A 94.66 ±2.04

L.S.D = 5.5

Different capital letters referred to the significant differences (P<0.05).

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