Controlled mechanical ventilation in horses under vecuronium blockage

Ventilação controlada mecânica em cavalos com o emprego de vecurônio

CORRESPONDENCE TO:
Denise Tabacchi Fantoni
Departamento de Cirurgia
Faculdade de Medicina Veterinária e Zootecnia da USP
Cidade Universitária Armando de Salles Oliveira
Av. Orlando Marques de Paiva, 87
05508-000 – São Paulo – SP
e-mail: dfantoni@usp.br

1 - Departamento de Cirurgia da Faculdade de Medicina Veterinária e Zootecnia da USP – SP

Denise Tabacchi FANTONI[1]; José de ALVARENGA[1]; Luis Claudio Lopes Correia da SILVA[1]; Silvia Renata Gaido CORTOPASSI[1]; Regina Mieko Sakata MIRANDOLA[1]

SUMMARY

The purpose of this study was to investigate the metabolic and cardiorespiratory effects of the administration of vecuronium under controlled mechanical ventilation versus spontaneous ventilation in horses. Twenty healthy horses scheduled to elective surgery were randomized and assigned in two groups. All animals were pre-medicated with romifidine (100 mg/kg iv), anesthesia was induced with an association of tiletamine-zolazepam (2 mg/kg iv) and was maintained with halothane. Animals of group I remained in spontaneous ventilation without muscle relaxation while group II received vecuronium (0.1 mg/kg IV) and was submitted to controlled mechanical ventilation. Vecuronium administration did not cause any significant change of heart rate or rhythm, central venous pressure and arterial pressure. With the animals that remained under spontaneous ventilation, no differences were observed in these parameters. The animals that received vecuronium showed lower values of $PaCO_2$ and normal values of pH in relation to the spontaneous ventilation group. Vecuronium duration was 12.83 ± 1.72 minutes. After halothane discontinuation the weaning time in group I I was 6.09 minutes with mean final $PaCO_2$ values of 50.78 mmHg. There was no need of pharmacological reversion of vecuronium effect and recovery from anesthesia was similar in the two groups. In conclusion, the use of mechanical controlled ventilation and vecuronium in horses is easily performed, does not induce further cardiovascular depression and should be employed in equines undergoing major operations.

UNITERMS: Mechanical ventilation; Vecuronium; General anesthesia; Horses.

INTRODUCTION

Mechanical ventilation nowadays is routinely employed during general anesthesia. The use of this procedure is largely justified due to many undesirable physiological alterations caused by anesthesia in spontaneous breathing subjects. The main alterations described during general anesthesia are: depression of the respiratory center due to the use of anesthetic drugs, oxygenation disturbances due to atelectasis and ventilation/perfusion mismatching, hypercapnia, and metabolic acidosis. In the anesthetized animal mechanical ventilation permits the respiratory frequency, gas exchange and pulmonary compliance to return to normal values and reduces respiratory work. In addition to this, the recumbence during general anesthesia increases ventilation/perfusion mismatch and decreases lung volume[24,33]. In the majority of the anesthetized animals breathing spontaneously, the partial arterial pressure of carbon dioxide ($PaCO_2$) rises to values higher than 45 mmHg[3,6]. The gas exchange process is impaired, arterial hypoxaemia occurs and a significant difference in the partial pressure of oxygen from the alveolus to the arterial blood is always observed[32,33,34]. The sound patient can tolerate these alterations, however, those suffering important metabolic disturbances, due to the lack of physiological reserves, are unable to compensate the respiratory alteration, which is aggravated by the use of drugs for induction and maintenance of general anesthesia[6,30].

Moreover, other injuries such as thoracic trauma, pneumothorax, pleural effusion, pulmonary edema and diaphragmatic hernia, may also contribute to worsen the alterations previously described. This fact, reinforces the use of controlled ventilation during general anesthesia[1,2].

Controlled ventilation can be obtained in three different ways: depressing ventilatory centers by hyperventilation thus reducing $PaCO_2$ below the level at which the horse is stimulated to breathe, anesthesia deepening or use of neuromuscular blocking agents[13,28]. Muscle relaxants are routinely used in human anesthesia, to ease tracheal intubation and to allow smooth controlled mechanical ventilation, avoiding unnecessary hyperventilation and deep levels of anesthesia. Another important favourable point for the use of neuromuscular blocking agents during mechanical ventilation is related to the decrease of lung barotrauma incidence. Several studies have demonstrated that lung parenchyma injury is due to excessive volume ventilation[20,23]. A tidal volume higher than 10 ml/kg associated to elevated respiratory frequency may cause air trapping, alveoli injury and hemodynamic disturbances. The temptation to overdrive the neural respiratory stimulus by hyperventilation, a common way to induce apnea in animals, is a condemned practice

in modern human mechanical ventilation. In equine Veterinary Medicine, the neuromuscular blocking agents are mainly indicated with the primary intention of promoting a better muscle relaxation during specific procedures where muscle relaxation is mandatory as seen in orthopedic and ophtalmologic surgery[9,17].

Among the new adespolarizing neuromuscular blocking agents, vecuronium is characterized to be more potent and to have a rapid onset and shorter duration of action than pancuronium. In addition to its cardiovascular stability and absence of deleterious effects on kidneys and liver, no cardiac muscarinic receptors blockage and no histamine release are observed with the use of this agent. Another interesting characteristic described during vecuronium utilization is the infrequent possibility of residual blockage[7,15,16]. Vecuronium excretion is done by kidneys and liver. However renal failure has little influence on its depuration[15]. This means that the end of its action is less dependent of renal function. This vecuronium property could be an advantage on patients with nephropathies. Hepatic diseases however, jeopardize its elimination, because 50% of the administered drug dosage is excreted through the billiary route[21]. The knowledge about vecuronium in the horse is limited, but a dose of 0.1 mg/kg produces neuromuscular blockage of 20 - 30 minutes[8]. Similar to other agents frequently used in horses, the effect of vecuronium can be reverted by the use of neostigmine[3,10,19]. In dogs vecuronium is the muscle relaxant with fewer undesirable effects on the cardiovascular system, not affecting cardiac output, arterial pressure and central venous pressure[7,16]. Another advantage of this agent in this species could be its short duration of action and limited cumulative effects that could minimize or even eliminate the incidence of residual weakness during the recovery phase and the need of antagonists such as neostigmine[15]. The purpose of this study is to evaluate the metabolic and cardiorespiratory effects of mechanical controlled ventilation in horses paralyzed by vecuronium.

MATERIAL AND METHOD

This study is based on the observations done on 20 sound horses scheduled to routine surgery, 18 of those where male and 2 females. The horses were of different breeds, with ages varying from 2 to 8 years and bodyweight varying from 300 to 476 kg.

The animals were pre-medicated with romifidine (Sedivet, Boehringer Ingelheim, Brazil) (100 ug/kg iv) through a 16-gauge catheter placed percutaneously into the jugular vein. After 15 minutes, an association of tiletamine-zolazepam (Zoletil, Virbac do Brasil) (2 mg/kg iv) was given. Once the animal was recumbent, orotracheal intubation was performed and the animals connected to a large animal carbon dioxide absorption circuit in a semi closed system (Matrix VML, Large Animal Absorber Circuit, USA). Anesthesia was maintained with halothane (Halotano, Hoechst do Brasil S.A.) in 100% oxygen using an out-of circle halothane precision vaporizer, at a concentration high enough to keep an adequate level of surgical anesthesia (regular assessment of the palpebral and corneal reflexes). Afterwards the animals were randomly assigned to two groups: Group I - animals were kept in spontaneous ventilation and Group II - after 15 minutes of the beginning of inhalation anesthesia the horses received vecuronium (Norcuron - Labs. Organon do Brasil Ltda.) (0.1 mg/kg iv). While the total paralyzing effect was not achieved, ventilatory support was started by manually triggering the demand valve of the anesthetic machine at least tree times. Once the respiratory movements had ceased, the animals were set on controlled ventilation using a volume-limited/time-cycled ventilator (Rachel Model 2800 - Large Animal Anesthesia Ventilator - Mallard Medical, USA) designed for large animals. The respiratory rate was established at 9 to 10 respiratory movements per minute, inspiratory time at 2 seconds, inspiratory: expiratory rate 1:2.5. Tidal volume was initially estimated on 10 ml/kg and adjusted to maintain the $PaCO_2$ between 40 and 50 mmHg. Peak inspiratory pressure remained between 20 and 30 cm H_2O. At the end of operation the anesthetic inhalation was turned off, the weaning of controlled ventilation was done by gradually reducing the respiratory frequency with the intention of increasing the $PaCO_2$ thus stimulating the respiratory center and observing the presence of spontaneous respiratory movements. Before extubation the adequacy of the respiratory function was checked evaluating if the inspiratory force was good enough to provide adequate tidal volume during the exhalation, verified through the bellows movements to a normal range. This findings allowed to indicate or not the necessity of pharmacological antagonism of the neuromuscular blocking agent.

The parameters evaluated during the experiment were: heart rate and rhythm, arterial systolic and diastolic pressure, respiratory rate, oxyhemoglobin saturation, central venous pressure, blood gases and also blood pH and HCO_3^-. These parameters were evaluated as follows: cardiac rate and rhythm continuously on an oscilloscope (Dixtal 910 - Dixtal, Brazil); the arterial pressure directly through catheterization of the facial artery and connection to the pressure transducer (Biomonitor, Bese, Brazil); respiratory frequency through observation of thoracic wall movements during 1 (one) minute; oxyhemoglobin saturation by pulse oximetry (Modell 3100, BCI - Biochen International, USA) with the transducer positioned at the tip of the tongue, central venous pressure (CVP) through catheterization of the jugular vein and animals were in lateral recumbence. Blood samples for blood gas, pH and HCO_3^- analysis were obtained anaerobically through the catheter placed at the facial artery. The examination of the blood samples was done in a pH and gas analyzer (Gasometer ABL 330, Radiometer, Denmark), immediately after blood sampling.

The parameters were evaluated at the following moments: immediately before and 15 minutes after romifidine administration; 15, 30, 45 minutes after beginning of anesthetic maintenance with halothane and after recovery of spontaneous breathing. The evaluation of the onset of action of vecuronium was done by observing the cessation of respiratory movements. The criteria used to evaluate the cessation of vecuronium's action was observation of spontaneous palpebral movements and rotation of the ocular globe back to a forthmedial position.

The quality of the anesthetic recovery was evaluated by observing the presence or absence of: excitation, significant muscular tonus, motor incoordenation, sudden movements of head and limbs, number of times the animals tried to stand up and if they were able to remain standing. The recovery time was measured from the time the animal was extubated until it managed to stand straight on its own.

The results were statistically analyzed by parametric examination. Analysis of variance (ANOVA), followed by the

Tukey-Kramer test for comparison of the different observation moments of the same group were used. The Student-Newman-Keuls test was used to evaluate possible differences between the two groups. Significance was considered as $p<0.05$. Both statistical analysis were done by computer program (INSTAT, Graphpad Software, USA).

RESULTS

No differences related to age and weight between the animals of the two groups were verified. The surgical procedures had a mean duration of sixty minutes. The administration of the pre-anesthetic drug caused the expected effects on heart rate and rhythm. A significant decrease of heart rate was observed after romifidine administration in both groups. Mobitz type-I second-degree atrioventricular (AV) block was observed in 7 horses of group I and in 8 animals of group II; Mobitz type-II second-degree AV block occurred in 2 animals of group I and 1 animal of group II; First-degree AV block was observed in 1 animal of group I and sinoatrial block was diagnosed in 1 horse of group II. After the administration of the tiletamine-zolazepan association the normal sinus rhythm was reestablished. All data of heart rate, expressed in mean values and SD of mean are described in Tab. 1. A significant decrease of heart rate was verified in both experimental groups after romifidine administration. During halothane anesthesia the heart rate was similar to base line values. The injection of vecuronium did not cause any alteration of the cardiac rhythm or rate. A significant reduction of arterial systolic and diastolic pressure after administration of the inhalatory agent, was noticed in both groups, with no return to normal values until the end of the recording period. During the experiment central venous pressure remained unchanged.

In comparison to the respiratory rate before anesthesia, both groups had inferior values during inhalatory anesthesia. Values obtained after romifidine administration in horses of group II were also significantly greater than those values verified during halothane anesthesia but inferior to animals of group I. Animals that remained in spontaneous ventilation, showed a significant decrease of respiratory rate at 30 and 45 minutes, in relation to those animals that had controlled ventilation.

The pH of the animals of group I decreased during the course of inhalatory anesthesia, with no restoration of base values. In group II the values decrease only during the first 15 minutes of the inhalatory anesthetic procedure. When controlled ventilation was performed, theses values returned to the pre-anesthetic values.

Table 1

Metabolic and cardiorespiratory data of animals submitted to spontaneous ventilation (Group I) versus controlled ventilation (group II). São Paulo, 1995.

Parameters	Groups	Control values	15 min after romifidine	15 min after halothane	30 min after halothane	45 min after halothane	weaning time
HR	I	39,6±6,6	32,8±9,1	37,5±6,9	35,4±4,7	36,2±4,6	-
(beasts/min)	II	38,2±6,2	31,7±2,8	34,7±3,3	33,1±5,0	33,3±4,2	31±3,9
RR	I	22,2±5,1	17,6±4,6*	6,1±3	6,9±3,0*	7,3±2,8	-
(mov/min)	II	17±6,3	13,8±2,9*	8,3±3,9	9,3±0,8*	9,6±0,7	9,9±2,0
SAP	I	140,7±27,0	141,4±27,0	101,3±27,1	92,7±22,4	95,6±13,6	-
(mmHg)	II	129,5±25,9	135,7±36,7	84,6±15,7	85,5±27,1	90±32	103,5±28,6
DAP	I	99,5±26,2	110,3±29,1	64,8±17,7	72,3±26,8	80±16,7	-
(mmHg)	II	79,3±17,5	101,7±28,6	57,9±14,5	64±31,1	63±37,5	76,5±34,2
CVP	I	7,7±7,3	7±7,5	8,2±6,5	13,5±10,3	-	-
(cmH2O)	II	1,6±6,8	2,8±7,2	3,5±5,1	5,3±7,9	-	-
pH	I	7,35±0,02	-	7,29±0,05	7,25±0,03*	7,26±0,03*	-
	II	7,37±0,03	-	7,28±0,04	7,35±0,06*	7,36±0,07*	7,32±0,06
$PaCO_2$	I	41,15±3,3	-	57,82±3,81	62,27±5,26*	57,31±7,4*	-
(mmHg)	II	40,49±3,6	-	56,31±7,15	46,41±7,21*	44,06±8,4*	50,78±9,6
PaO_2	I	90,7±8,1	-	191,6±55,9	220,9±44,1*	237,9±55,7*	-
(mmHg)	II	95,4±10,8	-	248,2±30,7	287,2±60,6*	308,7±67,8*	252,6±79,3
HCO3	I	22,5±1,9	-	25,4±1,8	26,4±2,1	25,4±1,4*	-
(mmol/L)	II	22,8v1,4	-	26,0±2,4	24,8±1,4	23,7±1,8*	24,6±2,1
SaO_2	I	96,9±0,6	-	97,9±2,1	98,7±1,3	99,1±0,7*	-
(%)	II	97,1±1,5	-	99,2±0,8	99,4±0,9	99,7±0,2*	99,3±0,8
SpO_2	I	-	-	95,1±2,8	94,8±2,5	93,4±2,6	-
(%)	II	-	-	96,7±1,8	95,4±2,1	95±3,0	94,3±2,3

HR - heart rate; RR - respiratory rate; SAP - systolic arterial pressure; DAP - diastolic arterial pressure; CVP - central venous pressure; $PaCO_2$ - partial pressure of carbon dioxide; PaO_2 - partial pressure of oxygen; HCO_3^- - plasma bicarbonate level; SaO_2 - oxyhemoglobin saturation on arterial blood; SpO_2 - oxyhemoglobin saturation by pulse oxymeter. * significantly different ($p<0.05$) - group I in relation to group II.

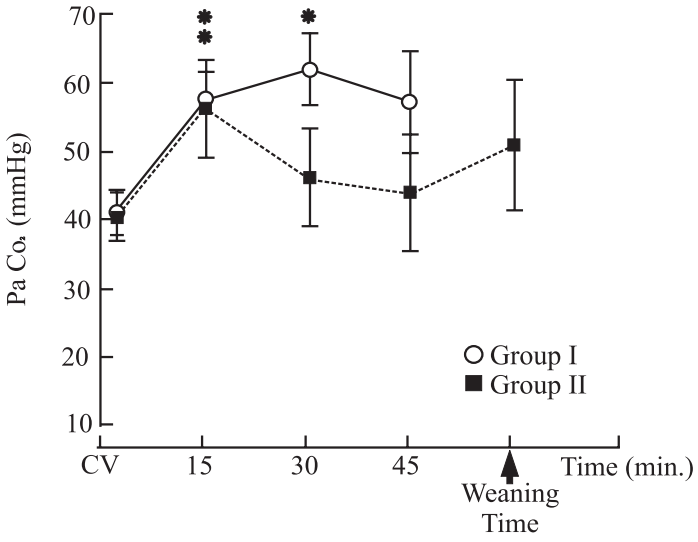

Figure 1

The solid line represents the $PaCO_2$ values of animals of group I. The dash line represents the $PaCO_2$ values of animals submitted to controlled ventilation (group II); mean ± s.d.

CV - control values;

* group I presented significant higher levels of $PaCO_2$ when compared to control values and to the group II at the same time ($p<0.05$);

** both groups presented significant higher values of $PaCO_2$ when compared to control values ($p<0.05$).

In both groups the PaO_2 increased after administration of 100% oxygen. Animals of the 2nd group reached higher PaO_2 values than animals of the first group. The $PaCO_2$ increased considerable during the inhalatory anesthesia procedure in animals of group I, not returning to pre-anesthetic values. In the animals of group II however a rise in $PaCO_2$ values occurred only at 15 minutes after the establishment of inhalatory anesthesia, recovering baseline values during controlled ventilation and increasing again after re-establishment of spontaneous ventilation (Fig. 1). Only at 45 minutes after the begin of inhalatory anesthesia both groups showed significant difference of the HCO_3^- values. The oxyhemoglobin saturation in arterial blood, increased in both groups since 100% oxygen was used. There was a significant difference in values obtained by pulse oximetry and those of the arterial blood.

The time period necessary for apnea establishment was 2.5 ± 0.5 minutes. The duration of action of vecuronium was 12.83 ± 1.72 minutes.

Concerning the anesthetic recovery, only 3 animals (one of group I and two of group II) made more than one attempt to stand up, before assuming the standing position. These three animals assumed a position in which they stood on their rear leg toes, showing strong contraction of the hindlimb muscles. The time taken by the animals to stand up was 34.12 ± 12.07 minutes for animals of group I and 29.25 ± 9.37 minutes for animals of group II and the difference was not statistically significant. Three animals of group I and four animals of group II made violent attempts to get out from the recovery room when they stood up but until that moment no signs of excitement were observed.

DISCUSSION

Uncountable clinical situations require the use of ventilatory assistance, in order to improve tissue oxygenation and/or to remove carbon dioxide. The use of mechanical ventilation is frequently necessary, when drugs, specially during general anesthesia, have a negative effect on the respiratory system impairing gas exchanges in the lungs. Yet, even if improving oxygenation, the use of ventilatory assistance may cause hemodynamic embarrassment[25]. The cardiovascular depression observed during controlled ventilation is caused by multifactorial effects most of them due to mechanical restraint of venous return, caused by the positive pressure on lungs and thorax. In addition to this mechanical factor, excessive reduction of carbon dioxide achieved with this kind of ventilation, decreases circulating cathecolamines[25].

Concerning the observed cardiovascular variations, hypotension was verified during inhalatory anesthesia, in both groups of this study. Romifidine produces cardiovascular depression, characterized by bradicardia and hypertension which is followed by persisting hypotension[4,5]. The association of tiletamine-zolazepam, used to induce anesthesia, does not determine cardiovascular depression. The ability of cyclohexamines (ketamine, tiletamine) to maintain cardiac function and arterial blood pressure by a variety of different mechanisms is well known[36]. Hubbel *et al.*[12] evaluated the association of tiletamine-zolazepan (2.2 mg/kg IV) in horses pre-medicated with xylazine (1.1 mg/kg IV) and verified a significant decrease in arterial blood pressure after xylazine administration that was not intensified or modified by the injection of the tiletamine-zolazepan association. In the same way, Vecunorium does not cause any undesirable effects on the cardiovascular system[15]. In dogs this agent does not promote any significant change in arterial blood pressure[16]. Hackett *et al.* noticed an increase in cardiac output and systolic volume as well as a decrease in pulmonary vascular resistance after vecuronium. However halothane, the inhalatory anesthetic, causes an important reduction of myocardial contractility, decreasing cardiac output, left ventricular work and systemic arterial pressure[30]. Considering the previous reports mentioned above on the effects of the drugs used in this study and the results obtained in both experimental groups, we may suppose that the bradicardia and hypotension noticed here are related to the use of romifidine and halothane. In the present study the decrease in arterial pressure in the animals of group II was similar to the variation noticed in the group I. Coincidentally to the observations of Steffey; Howland[31] and Hodgson *et al.*[11], the decrease in arterial pressure in our study may be attributed to the halogenated agent, instead of the ventilation mode employed in the animals.

Hypoventilation characterized by hypercapnia, hypoxia and respiratory acidosis is a common complication during horse anesthesia, specially when kept on spontaneous ventilation[6,13,18,30,32]. Various assumptions were theorized to explain the hypoventilation observed during general anesthesia in this species. The fact that the animal assumed lateral or dorsal recumbence was for long thought to be the principal cause of respiratory malfunctions noticed during general anesthesia. Many studies done on anesthetized horses reinforce the deleterious

effects of recumbence on respiratory function[24,32,33]. Unfavorable blood flow distribution in the lung that remains in a ventral position is observed and is due mostly to mechanical forces. As a result of the increase in pleural pressure a reduction of pulmonary volume occurs[24]. An augmentation in the pulmonary shunt, that can reach values up to 51% and an increase of alveolar-arterial oxygen difference is observed[34]. Rugh *et al.*[26] pointed out that the effects of lateral recumbence on PaO_2 and $PaCO_2$ are to be accounted to the depression caused by anesthetic agents used during the procedure with no relation to the recumbence alone. In fact Schatzmann *et al.*[27] evaluating the effects of lateral and dorsal recumbence in ponies given glycerol guaiacolate, did not notice any difference concerning $PaCO_2$, when comparing animals in recumbence to those standing. Halothane and other inhalatory anesthetics cause dose-dependent respiratory depression, characterized by increase of $PaCO_2$, decrease in the capacity to oxygenate arterial blood and decrease in respiratory rate[29,31]. Theses drugs also induce intense relaxation of respiratory muscles and decrease the chemoreceptor sensitivity[6,13,30].

Animals of group I showed hypercapnia and a marked decrease of pH during inhalatory anesthesia as expected. The values of $PaCO_2$ of 58, 62 and 57 mmHg at 15, 30 and 45 minutes respectively, are similar to those observed by Hubbel; Muir[13], who had mean $PaCO_2$ values of 57.6 mmHg. Grandy *et al.*[6] noticed higher $PaCO_2$ levels reaching 78.5 mmHg. The presence of hypercapnia and low values of pH can be explained by the use of halothane and the fact that animals were in lateral recumbence. On the other hand, animals on mechanical ventilation showed $PaCO_2$ values of 46 and 44 mmHg at 30 and 45 minutes, values close to the ones observed by Hubbel; Muir[13], who had average values of 45 mmHg during 60 minutes of controlled ventilation. There was a significant difference between values of oxyhemoglobin saturation obtained by pulse oximetry and those obtained at the arterial blood, the same results were obtained by Whitehair[35]. The absence of accuracy of pulse oximeter was due to the kind of the probe used in the tongue in this specie.

In this study the weaning time was 6.09 minutes with mean $PaCO_2$ values of 50.78 mmHg. Hubbel; Muir[13] had a weaning time of 3 minutes and $PaCO_2$ of 65.8 mmHg. This difference is probably due to the fact that in this study, the weaning time was considered until animals had reassumed spontaneous ventilation with adequate respiratory rate and amplitude.

Vecuronium had a neuromuscular blockage effect of 12.83 minutes against 20-30 mentioned by Hall; Clarke[8]. Despite the short duration of action subsequent doses of vecuronium were not necessary. The maintenance of adequate levels of PaO_2, $PaCO_2$ along with the respiratory center depression caused by halothane may explain this facts. The use of neostigmine to revert vecuronium effect was not necessary as the animals were able to maintain a normal tidal volume after weaning. This last observation is very important, because the use of other blockage drugs such as pancuronium require pharmacological antagonism[3,14]. Neostigmine may promote undesirable effects like bradicardia, hypotension and increased gastrointestinal motility even if atropine is administered[19]. Besides that, unpredictable effects such as increase of blood pressure and tachycardia may also occur[10].

Vecuronium has shown to be an excellent agent to allow smooth controlled ventilation in horses, preventing the animals from fighting the ventilator and without promoting undesirable cardiovascular effects such as tachycardia and hypertension commonly verified with pancuronium in horses[22]. The use of controlled ventilation in horses was easily performed and was not associated with cardiovascular embarrassment during inhalatory anesthesia.

ACKNOWLEDGMENTS

The authors would like to thank doctor Melanie Klenn for the translation of this manuscript.

RESUMO

O objetivo do presente estudo foi avaliar os efeitos metabólicos e respiratórios da administração de vecurônio em cavalos submetidos a ventilação controlada e compará-los àqueles que permaneceram em respiração espontânea. Foram empregados vinte animais hígidos alotados em dois grupos experimentais. Todos os animais foram pré-medicados com romifidina (100 mg/kg IV) sendo a anestesia induzida com a associação de tiletamina-zolazepam (2 mg/kg IV) e a manutenção realizada com halotano. Os animais do grupo I permaneceram em respiração espontânea enquanto os animais do grupo II receberam vecurônio na dose de 0,1 mg/kg IV sendo submetidos a ventilação controlada mecânica. A administração do vecurônio não promoveu qualquer alteração significativa da freqüência ou ritmo cardíaco, pressão venosa central ou pressão arterial. No atinente aos animais que permaneceram em respiração espontânea, não houve qualquer diferença em relação a estes parâmetros quando comparados aos dos animais que permaneceram em respiração espontânea. Os animais que receberam o vecurônio apresentaram valores inferiores de $PaCO_2$ e valores normais de pH em relação aos animais do grupo I. A duração de ação do vecurônio foi de 12,83 ± 1,72 minutos. Após o término da administração do halotano, os animais do grupo II retornaram à ventilação espontânea em 6,09 minutos demonstrando valores de $PaCO_2$ da ordem de 50,78 mmHg. Não houve necessidade de reversão farmacológica do bloqueador e a qualidade da recuperação foi semelhante nos dois grupos. Frente aos resultados obtidos, pode-se concluir que o emprego de ventilação controlada mecânica e vecurônio em eqüinos é factível e isenta de efeitos adversos, sendo portanto indicada nesta espécie.

UNITERMOS: Respiração Artificial; Brometo de vecurônio; Anestesia geral; Eqüinos.

REFERENCES

1- BEECH, J. Miscellaneous lung and pleural injuries. *In*: BEECH, J. **Equine respiratory disorders**. Philadelphia : Lea & Febiger, 1991. p.215-21.

2- BOYSEN, P.G.; MODELL, J.H. Pulmonary edema. *In*: SHOEMAKER, P.R.; GRENVIK, A.; AYRES, S. **Textbook of critical care**. 2.ed. Philadelphia : W.B. Saunders, 1989. p.515-9.

3- CALDWELLL, J.E.; ROBERTSON, N.E.; BAIRD, W.L.M. Antagonism of profound neuromuscular blockade induced by vecuronium or atracurium **British Journal of Anaesthesia**, v.58, n.11, p.1285-9, 1986.

4- CLARKE, K.W.; ENGLAND, G.C.W; GOOSSENS, E.L. Sedative and cardiovascular effects of romifidine alone and in combination with butorphanol, in the horse. **Journal of Veterinary Anaesthesia**, v.18, n.2, p.25-30, 1991.

5- ENGLAND, G.C.W.; CLARKE, K.W.; GOOSSENS, E.L. A comparison of tree alpha 2-adrenoceptor agonists (romifidine, detomidine, xylazine) in horses. **Journal of Veterinary Pharmacology Therapeutics**, v.15, n.2, p.194-201, 1992.

6- GRANDY, J.L.; STEFFEY, E.P.; MILLER, M. Arterial blood pO_2 and pCO_2 in horses during early halothane-oxygen anaesthesia. **Equine Veterinary Journal**, v.19, n.3, p.314-8, 1987.

7- HACKET, G.H.; JANTZEN, J.P.A.H.; EARNSSHAW, G. Cardiovascular effects of vecuronium, atracurium, pancuronium, metocurine and RGH-4201 in dogs. **Acta Anaesthesiologica Scandinavica**, v.33, n.4, p.298-303, 1989.

8- HALL, L.W.; CLARKE, K.W. **Veterinary anaesthesia**. London : Baillière Tindall, 1991. p.120.

9- HILDEBRAND, S.V.; HOLLAND, M.; COPLAND, V.S.; DAUNT, D.; BROCK, N. Clinical use of the neuromuscular blocking agents atracurium and pancuronium for equine anesthesia. **Journal of the Veterinary Medical Association**, v.195, n.2, p.212-9, 1989.

10- HILDEBRAND, S.V.; HOWITT, G.A Antagonism of pancuronium neuromuscular blockade in halothane-anesthetized ponies using neostigmine and edrophonium. **American Journal of Veterinary Research**, v.45, n.11, p.2276-80, 1984.

11- HODGSON, D.S; STEFFEY, E.P.; GRANDY, J.L.; WOLINER, M.J. Effects of spontaneous, assisted and controlled ventilatory modes in halothane-anesthetized geldings. **American Journal of Veterinary Research**, v.47, n.5, p.992-6, 1986.

12- HUBBELL, J.A.E.; BEDNARSKI, R.M.; MUIR, W.W. Xylazine and tiletamine-zolazepam anesthesia in horses. **American Journal of Veterinary Research**, v.50, n.3, p.731-41, 1989.

13- HUBBELL, J.A.E.; MUIR, W.W. Rate of rise of arterial carbon dioxide tension in the halothane anesthetized horse. **Journal of the American Veterinary Medical Association**, v.186, n.5, p.374-6, 1985.

14- HUNTER, J.M. Resistence to non-depolarizing neuromuscular blocking agents. **British Journal of Anaesthesia**, v.67, n.5, p.511-4, 1991.

15- HUNTER, J.M.; JONES, R.S.; UTTING, J.E. Comparison of vecuronium, atracurium and tubocurarine in normal patients and patients with no renal function. **British Journal of Anaesthesia**, v.56, n.9, p.941-56, 1984.

16- JONES, R.S. Neuromuscular blocking action of vecuronium in the dog and its reversal by neostigmine. **Research in Veterinary Science**, v.38, n.2, p.193-6, 1985.

17- KALHORO, A.B.; REX, M.A.E. The dose rate of pancuronium bromide for horses. **Australian Veterinary Journal**, v.60, n.11, p.348-9, 1983.

18- KARIMI, A. Comparison of the effects of two sets of anesthetic agents and posture on respiratory rate, heart rate, pH, blood gas and acid-base status in the horse. **British Veterinary Journal**, v.67, n.6, p.506-10, 1987.

19- KLEIN, L.; HOPKINS, J.; BECK, E.; BURTON, B. Cumulative dose response to gallamine, pancuronium, and neostigmine in halothane-anesthetized horses: neuromuscular and cardiovascular effects. **American Journal of Veterinary Research**, v.44, n.5, p.786-92, 1983.

20- KOLOBOW, T.; MORETTI, M.P.; FUMAGALLI, R. Severe impairment in lung function induced by peak airway pressure during mechanical ventilation. **American Review Respiratory Disease**, v.135, n.2, p.312-5, 1987.

21- LEBRAULT, C.; BERGER, J.L.; D'HOLLANDER, A.A.; GOMENI, R.; HENZEL, D.; DUVALDESTIN, P. Pharmacokinetics and pharmacodinamics of vecuronium (ORG NC 45) in patients with cirrhosis. **Anesthesiology**, v.62, n.5, p.601-5, 1985.

22- MANLEY, S.V.; STEFFEY, E.P.; HOWITT, G.A; WOLINER, M. Cardiovascular and neuromuscular effects of pancuronium bromide in the pony. **American of Journal Veterinary Research**, v.44, n.7, p.1349-53, 1983.

23- MASCHERONI, M.; KOLOBOW, T.; FUMAGALLI, R. Acute respiratory failure following pharmacologically induced hyperventilation: an experimental animal study. **Intensive Care Medicine**, v.15, n.1, p.8-14, 1988.

24- MCDONELL, W.N.; HALL, L.W.; JEFFCOTT, L.B. Radiographic evidence of pulmonary function in laterally recumbent anaesthetized horses. **Equine Veterinary Journal**, v.11, n.1, p.24-32, 1979.

25- PEREL, A.; PIZOV, R. Cardiovascular effects of mechanical ventilation. *In*: PEREL, A.; STOCK, M.C. **Mechanical ventilatory support**. Baltimore : Williams & Wilkins, 1992. p.51-65.

26- RUGH, K.S.; GARNER, H.E.; HATFIELD, D.J.; HERROLD, D. Arterial oxygen and carbon dioxide tensions in conscious laterally recumbent ponies. **Equine Veterinary Journal**, v.16, n.3, p.185-8, 1984.

27- SCHATZMANN, U.; KOEHI, M.; DUDAN, F.; ROHR, W.; JONES, R.S. Effect of postural changes of certain circulatory and respiratory values in the horse. **American Journal of Veterinary Research**, v.43, n.6, p.1003-5, 1982.

28- SHAWLEY, R.V.; MANDSAGER, R.E. Clinical use of positive-pressure ventilation in the horse. **Veterinary Clinics North America- Equine Practice**, v.6, n.3, p.575-85, 1990.

29- STEFFEY, E.P. Enflurane and isoflurane anesthesia: a summary of laboratory and clinical investigation. **Journal of the American Veterinary Medical Association**, v.172, n.3, p.367-5, 1978.

30- STEFFEY, E.P. Inhalation anesthetics and gases. *In*: MUIR, W.W.; HUBBEL, J.A.E. **Equine anesthesia**. St Louis : Mosby Year Book, 1991. p.366-79.

31- STEFFEY, E.P.; HOWLAND, J.R. Cardiovascular effects of halothane in the horse. **American Journal of Veterinary Research**, v.39, n.4, p.611-5, 1978.

32- STEGMANN, G.P. Pulmonary function in the horse during anesthesia: a review. **Journal of the South African Veterinary Association**, v.57, n.1, p.49-53, 1986.

33- STEGMANN, G.F.; LITTLEJOHN, A. The effect of lateral and dorsal recumbency on cardiopulmonary function in the anaesthetized horse. **Journal of the South Africa Veterinary Association**, v.58, n.1, p.21-7, 1987.

34- WEAVER, B.M.Q.; WALLEY, R.V. Ventilation and cardiovascular studies during mechanical controlled ventilation in horses. **Equine Veterinary Journal**, v.5, n.1, p.9-15, 1975.

35- WHITEHAIR, K.J. Pulse oximetry in horses. **Veterinary Surgery**, v.19, n.3, p.243-8, 1990.

36- WRIGHT, M. Pharmacologic effects of ketamine and its use in veterinary medicine. **Journal of the American Veterinary Medical Association**, v.180, n.12, p.1462-71, 1982.

Received: 11/04/1997
Accepted: 07/11/1997