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The Relationship between Arterial and End-tidal Partial Pressures of CO_2 in Halothane-anesthetized Heavy Breed Horses with respect to Operative Positions and the Modes of Ventilation

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Halothane으로 마취된 거대말에서 수술자세와 호흡방법에 따른 동맥혈 및 호기말 이산화탄소 분압 사이의 관계

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요 약 : 체중이 700~750 kg 인 4마리의 중종마에 70분동안 마취를 실시하여 동맥혈의 이산화탄소 분압과 호기말 가스내의 이산화탄소 분압 사이의 관계를 관찰하였다. 마취도중 자발 호흡, 인공호흡 1(흡기시간 2.0초), 인공호흡 2(흡기시간 2.5초)를 각각 30분, 20분, 20분씩 실시하였으며 매 10분마다 동맥혈가스분석과 호기말 가스내 이산화탄소분압 측정을 실시하였다. 동시에 혈압, 심전도, 체온측정을 실시하여 마취된 환축의 상태를 관찰하였다. 2주후에 자세를 달리하여(앙와에서 측와로) 같은 방법으로 재 실험하였다. 호기말 이산화탄소분압은 동맥혈에서보다 평균 10 mmHg 정도 낮은 양상을 보였으나 높은 상관관계를 보였으며 자세에 따른 유의적 차이는 없었다(앙와자세; r=0.949, 측와자세; r=0.920, P<0.01). 이러한 결과를 토대로 조직에 창상을 줄 수 있는 동맥혈 가스분석 대신 비침습적방법인 호기말 가스내 이산화탄소 분압을 측정하는 것이 환축의 모니터링에 효과적으로 사용 가능하다는 것을 알수있었다.

Key word : Arterial CO_2 tension, End-tidal CO_2 tension, Halothane-anesthetized horses

Introduction

General anesthesia tends to cause respiratory depression. This often cause hypoventilation, characterized by decreased tidal volume and slightly increased respiratory rate, which may progress hypoxia, hypercabia and respiratory failure.

To help prevent these complications, some measurements of the functional aspect of ventilation are required. Carbone dioxide measurement in arterial blood is the most sensitive indicator of adequacy of pulmonary ventilation. End tidal CO_2 tension ($P_{ET}CO_2$) is an estimate of alveolar CO_2 tension, which is closely related to arterial CO_2 tension ($PaCO_2$). The use of end-tidal partial pressure of CO_2 as a measure of $PaCO_2$ has been demonstrated in clinical use in halothane-anesthetized dogs[2]. More recently, use of the capnometer has been reported in isoflurane-anesthetized ponies breathing spontaneously and in horses with controlled ventilation[13].

The present study was undertaken to evaluate the ability of $P_{ET}CO_2$ to predict $PaCO_2$ in the heavy-breed horses (700~850 kg) during halothane anesthesia with respect to operative positions (dorsal and lateral)

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and the modes of ventilation (spontaneous and ventilator supported breathing).

Materials and Method

Preparation of animals and anesthesia

Four nonmedicated horses (3 females and 1 male) weighing 713~825 kg were used. All horses were free of cardiopulmonary diseases, as determined by physical examination, electrocardiography and blood analysis. Feed was withheld about 12 hours before induction of anesthesia but water was always available. All horses were anesthetized twice in either lateral or dorsal recumbency at 2 weeks intervals.

Medetomidine (4.0 μg/kg) was given 10 minutes before induction of anesthesia. Anesthesia was induced with the combination of diazepam (0.03 mg/kg) and ketamin (2.0 mg/kg). Horses were positioned in right lateral or dorsal recumbency and then orotracheal intubation was performed using an cuffed endotracheal tube. The halothane concentration was adjusted to maintain the anesthesia to be adequate or deep(concentration: 4~10%). The palpebral reflex was slow or absent and eye ball rotated slowly or centered without any movement. A standard large animal anesthetic semiclosed circular system and pressure- and time-cycled ventilator were used to deliver the halothane in oxygen and control the animal's breathing(Compos EV, METRAN, Japan). The oxygen flow rate was 8.8~13.2 ml/kg/min. The soda lime canister was always filled with fresh soda lime before anesthesia induction. Ventilation was maintained spontaneously during first 30 minutes and then controlled by ventilator for a minimum of 60 minutes. During controlled ventilation, airway pressure and inspiration time were maintained 30 mmHg and 2.0 seconds for the first 20 minutes (mechanical ventilation I) and 30 mmHg, 2.5 seconds for the second 20 minutes (mechanical ventilation II). During the ventilator-controlled anesthesia, all horses were given lactated Ringer's solution including dobutamine (1.5~2.0 μg/kg/min), IV, at a rate of 3 ml/kg/hr. Dobutamine was excluded in case of maintaining adequate blood pressure (mean blood pressure; 70 mmHg). A branch of facial or metatarsal artery was catheterized with a 22-gauge teflon catheterization set.

After completion of measurements, catheter was removed and horses were transported across the hall to a padded equine recovery stall. During lateral recumbency in the recovery stall, horses spontaneously breathed an oxygen-enriched air mixture delivered via a tube positioned at the tracheal end of the endotracheal tube for about 10 minutes. Horses were returned to their paddock 30 to 60 minutes after standing.

Circulatory and respiratory measurement

Circulatory and respiratory parameters were measured 30 minutes after induction of anesthesia in spontaneous ventilation and 50, 70 minutes in mechanical ventilation I and II, respectively.

Arterial blood pressure was measured continuously by the direct method. Arterial pH, $PaCO_2$, and PaO_2 were measured every 10 minutes within 10 minutes after blood sampling using a blood gas analyzer (238 pH/blood gas analyzer, CIBA Corning, Japan). Blood gas were corrected for rectal temperature and hemoglobin concentration. Rectal temperature was measured continuously by electrical thermometer.

Concentration of CO_2 in tracheal gas ($P_{ET}CO_2$) was measured continuously, using an infrared CO_2 analyzer (Capnogard, NOVAMETRIX, USA). Gas was sampled from the catheter secured into an endotracheal tube adapter. The peak, end-expired, fractional concentrations of CO_2 were read from the monitor at the same time with blood sampling. During anesthesia electrocardiography was used continuously for monitoring the animal's condition. Heart rate was obtained from the ECG tracing.

The alveolar dead space to tidal volume ratio (Vd/Vt) was calculated, using Nunn and Hill's arrangements of the Bohr equation where the fraction of end-tidal gas from unperfused spaces in the lung is indicated by;

$$Vd/Vt=(PaCO_2-P_{ET}CO_2)/PaCO_2$$

This arrangement assumes that the unperfused alveolar spaces contain gas which is free from CO_2 and that the PCO_2 of the gas in the properly perfused al-

veoli is constant and equal to $PaCO_2$, neither of which assumption is strictly correct[11].

Statistical analysis

The values of blood pressure, heart rate, arterial pH, $PaCO_2$, $P_{ET}CO_2$, $PaCO_2$-$P_{ET}CO_2$ and Vd/Vt ratio were compared with respect to the operative positions and the modes of ventilation by ANOVA and student's t-test using SAS GLM. The relationship between $PaCO_2$ and $P_{ET}CO_2$ was analyzed using simple linear regression.

Result

Systolic, diastolic, and mean arterial blood pressure of mechanical ventilation were greater than those of spontaneous ventilation regardless of position (Table 1). In systolic blood pressure, there was no difference between dorsal and lateral recumbency in mechanical ventilation but in spontaneous ventilation. In diastolic and mean blood pressures there was no difference between dorsal and lateral recumbency but between spontaneous and mechanical ventilation. There was one case of cardiac arrythmia in dorsal recumbency in our study, but the horse did not show that in lateral recumbency. Heart rate had no significant change during anesthesia. Arterial pH in mechanical ventilation was higher than that in spontaneous ventilation ($P<0.05$).

$PaCO_2$ and $P_{ET}CO_2$ in each ventilatory regimen were not different significantly between dorsal and lateral recumbency (Table 2). However $PaCO_2$ and $P_{ET}CO_2$ in spontaneous ventilation decreased significantly with the start of mechanical ventilation.

The regression equations for both the dorsal (n=36) and the lateral (n=35) groups are statistically significant ($p<0.0001$) and both of them show similar high correlation coefficiency (Fig 1, 2).

Table 1. Circulatory measurements during anesthesia in dorsal and lateral recumbency

mode of ventilation	Blood presssure (systolic,mmHg)	Blood pressure (diastolic,mmHg)	Blood pressure (mean,mmHg)	Heart rate (m^{-1})	Arterial pH
Dorsal recumbency					
Spontaneous Ven.	91.50 ± 12.58^{a}	54.00 ± 14.07^{a}	63.75 ± 14.66^{a}	38.00 ± 8.29^{a}	7.22 ± 0.04^{a}
Mechanical Ven. I*	106.75 ± 8.26^{b}	70.50 ± 4.93^{b}	82.25 ± 6.02^{b}	35.75 ± 1.50^{a}	7.38 ± 0.03^{b}
Mechanical Ven. II**	109.75 ± 12.34^{b}	73.75 ± 15.39^{b}	85.00 ± 13.74^{b}	36.50 ± 2.65^{a}	7.41 ± 0.02^{b}
Lateral recumbency					
Spontaneous Ven.	78.75 ± 14.86^{c}	47.75 ± 9.91^{a}	57.25 ± 11.15^{a}	34.75 ± 5.91^{a}	7.20 ± 0.08^{a}
Mechanical Ven. I	98.75 ± 3.59^{b}	$62.26\pm3.30^{a,b}$	75.00 ± 4.32^{b}	32.50 ± 3.00^{a}	7.38 ± 0.10^{b}
Mechanical Ven. II	101.75 ± 2.06^{b}	$60.50\pm5.92^{a,b}$	77.00 ± 3.65^{b}	35.75 ± 6.08^{a}	7.39 ± 0.10^{b}

[a-c]Means with different superscripts are significantly different within the same column ($p<0.05$).
*repiratory rate is 8/min, airway pressure is 30 mmHg, inspiration time is 2.0 seconds
**repiratory rate is 8/min, airway pressure is 30 mmHg, inspiration time is 2.5 seconds

Table 2. Result of Capnograph and Blood gas analysis in both positions

Mode of ventilation	$PaCO_2$ (mmHg)	$P_{ET}CO_2$ (mmHg)	$PaCO_2$-$P_{ET}CO_2$	Vd/Vt
Dorsal recumbency				
Spontaneous ven.	59.25 ± 7.81^{a}	48.25 ± 4.27^{a}	11.00 ± 4.40^{a}	0.181 ± 0.054^{a}
Mechanical Ven. I	36.25 ± 6.19^{b}	27.00 ± 2.16^{b}	9.25 ± 4.03^{a}	0.247 ± 0.062^{a}
Mechanical Ven. II	33.50 ± 8.19^{b}	24.50 ± 4.04^{b}	9.00 ± 4.55^{a}	0.257 ± 0.078^{a}
Lateral recumbency				
Spontaneous ven.	60.75 ± 6.13^{a}	44.50 ± 2.08^{a}	16.25 ± 4.65^{a}	0.246 ± 0.054^{a}
Mechanical Ven. I	35.50 ± 8.89^{b}	26.50 ± 2.89^{b}	9.00 ± 7.62^{a}	0.230 ± 0.138^{a}
Mechanical Ven. II	35.00 ± 8.49^{b}	25.50 ± 3.32^{b}	7.50 ± 5.80^{a}	0.206 ± 0.117^{a}

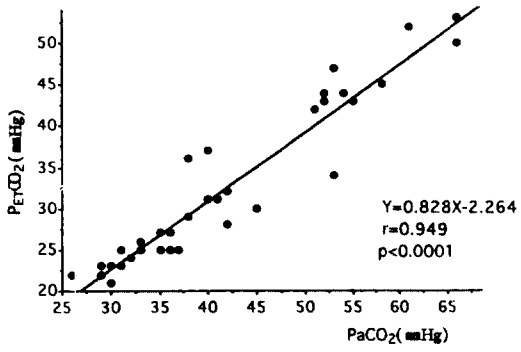

Fig 1. Scatter plot and regression line observed for $P_{ET}CO_2$ (mmHg) on $PaCO_2$ (mmHg) in dorsal recumbency during anesthesia.

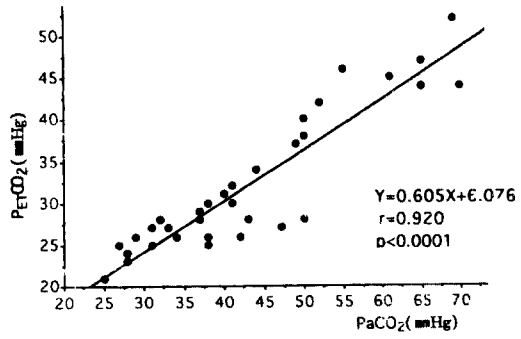

Fig 2. Scatter plot and regression line observed for $P_{ET}CO_2$ (mmHg) on $PaCO_2$ (mmHg) in lateral recumbency during anesthesia.

Discussion

Based on our observations, $P_{ET}CO_2$ was a trend indicator of $PaCO_2$ in either dorsal or lateral recumbency in halothane-anesthetized horses. There was a linear increase of $P_{ET}CO_2$ with increasing $PaCO_2$ similarly in both positions.

Theoretically, $P_{ET}CO_2$ predicts $PaCO_2$ accurately because the driving pressure for CO_2 at the arterial end of the pulmonary capillary to the alveoli is great. However despite the close correlation, $P_{ET}CO_2$ did not give the actual value of $PaCO_2$, only rather to indicate whether the value of $PaCO^2$ increase or not. The difference between $P_{ET}CO_2$ and $PaCO_2$ was 7~20 mmHg through anesthesia.

In one study, when a horse is placed in lateral recumbency, lung volume is reduced and there is an increase in asynchronous ventilation[14,15]. It is theorized that this is because of mechanical compression of the lower lung with a decrease in efficiency of ventilation, that is by an increase in dead space[7,20] and when anesthesia extended over 150 minutes horses in lateral recumbency had a statistically significantly great value of $PaCO_2$-$P_{ET}CO_2$ and Vd/Vt ratio[3]. Reduction in cardiac output causing pulmonary hypotension and in perfusion of nondependent lung regions were reported to be the major mechanism for an increase in $PaCO_2$-$P_{ET}CO_2$[1,13,19]. However the result of that study was applied in long-term recumbency and we found that in short-term anesthesia ($\leqq$90 minutes) there is no increasing of $PaCO_2$-$P_{ET}CO_2$ and Vd/Vt ratio in lateral recumbency as well as in dorsal recumbency especially during mechanical ventilation.

Slight increase of $PaCO_2$, $P_{ET}CO_2$ and $PaCO_2$-$P_{ET}CO_2$ during spontaneous breathing showed that they were hypoventilated. Geiser *et al* reported that the average of $PaCO_2$ during supported ventilation was significantly lower than during spontaneous ventilation at 1-2 hours of anesthesia[1]. When we started the mechanical ventilation they decreased significantly without change of Vd/Vt ratio. The Vd/Vt ratio provides a measure of the functional volume of lung not participating in CO_2 elimination. Its increase can be the major disturbance of gas change. In recent studies, the ateletiatic area in dependent lung regions was not smaller during mechanical than during spontaneous ventilation[12] and the same was true for this study. These findings indicate hypoventilation can be avoided when ventilation was controlled mechanically with high ventilation-perfusion ratios and there was no significant increase in alveolar dead space.

To continue cardiac function well in anesthetized horses, especially to prevent hypotension, dobutamine in lactated Ringer's solution were infused. One study in which dobutamine was administered to 200 horses for treatment of hypotension reported a 28% incidence of cardiac arrythmias, which included sinus bradycardia, second-degree atrioventricular block, premature ventricular depolarizations and isorhythmic

dissociation.

In conclusion, our study demonstrates that $P_{ET}CO_2$ can be a parameter of $PaCO_2$ in dorsal and lateral recumbency under adequate control of ventilation in halothane anesthetized heavy-breed horses. Further study with operating any surgery and longer duration is required.

Conclusion

The correlation between end-tidal partial pressure of CO_2 ($P_{ET}CO_2$) and arterial partial pressure of CO_2 ($PaCO_2$) was studied in four halothane-anesthetized heavy-breed horses(700~750 kg). They anesthetized 2 times in lateral recumbency and dorsal recumbency. The anesthesia was maintained under three different ventilatory modes; spontaneous ventilation, mechanical ventilation I (inspiration time 2.0 seconds, airway pressure 30 mmHg, respiratory rate 8 min^{-1}) and mechanical ventilation II (inspiration time was 2. 5 seconds, airway pressure 30 mmHg, respiratory rate 8 min^{-1}).

In both dorsal and lateral recumbency, there was a strong correlation between $PaCO_2$ and $P_{ET}CO_2$ (dorsal; r=0.949, lateral; r=0.920, $p<0.01$). In spontaneous and 2 modes of mechanical ventilation, The present study indicates that $P_{ET}CO_2$ is representative of $PaCO_2$ regardless of ventilation and position.

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