A COMPARISON OF THE CARDIORESPIRATORY EFFECTS OF CONTINUOUS POSITIVE AIRWAY PRESSURE BREATHING AND CONTINUOUS POSITIVE PRESSURE VENTILATION IN DOGS

A. SCOTT, A. E. G. HILL, M. K. CHAKRABARTI AND B. CARRUTHERS

SUMMARY

Five patterns of ventilation have been compared in dogs: (1) spontaneous ventilation at ambient pressure (SV); (2) intermittent positive pressure ventilation (IPPV); (3) spontaneous ventilation at 0.98 kPa positive airway pressure, delivered from a non-rebreathing circuit incorporating a 5-litre reservoir bag and fresh-gas flow of twice the minute volume (CPAP (bag)); (4) spontaneous ventilation at 0.98 kPa positive airway pressure, with the reservoir bag replaced by a weighted bellows (CPAP (bellows)) and (5) IPPV with 0.98 kPa positive end-expiratory pressure (CPPV). CPAP significantly decreased the rate of respiration compared with SV. CPAP (bellows) resulted in a significant increase in tidal volume. Mean oesophageal pressure, right atrial pressure, pulmonary wedge pressure and pulmonary artery pressure increased during IPPV, CPAP (bag), CPAP (bellows), and CPPV compared with SV. There were no significant changes in mean systemic arterial pressure, cardiac output, \( \text{PaO}_2 \), \( \text{PaCO}_2 \), \( \text{CaO}_2 \), \( \text{CaO}_2 - \text{CV}_2 \), \( \text{PAo}_2 - \text{PaO}_2 \) or pulmonary venous admixture. Under the conditions of this study oxygen transport was not altered by positive airway pressure ventilation.

Spontaneous ventilation with an increased airway pressure was reported to be effective in the treatment of acute left ventricular failure in 1936 (Poulton, 1936). Subsequently, the development of positive pressure ventilation overshadowed its potential as a means of ventilatory support.

Since Gregory and others (1971) demonstrated the effectiveness of continuous positive airway pressure breathing (CPAP) in the treatment of neonatal respiratory distress syndrome, it has been used widely as an adjunct to positive pressure ventilation in the treatment of this disorder. Recent reports have advocated the use of continuous positive airway pressure breathing in adults with acute respiratory failure, characterized by a high degree of intrapulmonary shunting refractory to increased inspired oxygen concentrations (Civetta, Brons and Gabel, 1972; Garg and Hill, 1975; Glasser, Civetta and Flor, 1975; Greenbaum et al., 1976).

Studies of the effect of continuous positive pressure ventilation (CPPV) in both dogs (Uzawa and Ashbaugh, 1969; Jones and King, 1973) and man (Ashbaugh and Petty, 1973) have shown that, while CPPV increases arterial oxygen tension and decreases pulmonary shunting, the effects on cardiac output and therefore oxygen transport vary according to the conditions of the study.

In preliminary studies with adult subjects, CPAP delivered from a non-rebreathing circuit incorporating a 5-litre reservoir bag resulted in an inspiratory pressure pattern that was dependent on fresh-gas flow rate. Flow rates from two to three times the minute volume were required in order to maintain a positive airway pressure during inspiration. This resulted in both waste of gases and also high noise levels. When a weighted bellows, similar to that described by Pfitzner (1976), was substituted for the reservoir bag, a positive airway pressure was maintained during inspiration provided that the fresh-gas flow was at least equal to the minute volume (Askitopolou, personal communication).

In an attempt to elucidate the physiological consequences of positive airway pressure we have compared five patterns of ventilation: (1) spontaneous ventilation at ambient pressure (SV); (2) intermittent positive pressure ventilation (IPPV); (3) spontaneous ventilation at 0.98 kPa positive airway pressure delivered from a non-rebreathing circuit incorporating a 5-litre reservoir bag, and a fresh-gas flow rate approximately twice the minute volume (CPAP (bag)); (4) spontaneous ventilation at 0.98 kPa positive airway pressure when the reservoir bag was replaced by a weighted bellows (CPAP (bellows)) and (5) IPPV.
with 0.98 kPa positive end-expiratory pressure (CPPV).

METHODS

The experiments were performed on 11 crossbred dogs weighing 23–31 kg. Anaesthesia was induced with thiopentone 10–20 mg kg$^{-1}$ and pentobarbitone 5–7 mg kg$^{-1}$ and maintained with incremental doses of thiopentone 3–5 mg kg$^{-1}$ when required. The dogs were placed in the supine position and auffed endotracheal tube was inserted. The inspired gas was air.

The circuit used for positive airway pressure breathing is illustrated in figure 1. Airway pressure was maintained at 0.98 kPa greater than atmospheric by an underwater blow-off valve in the expiratory limb. A second underwater blow-off valve in the inspiratory limb of the circuit combined with a non-rebreathing valve allowed the pressure in the inspiratory limb of the circuit to be maintained very close to airway pressure without diluting the expired gas with excess fresh-gas flow. During spontaneous breathing the dog breathed through the non-return valve only. This valve has a flow resistance of 69 Pa at 30 litre min$^{-1}$ and a deadspace of 29 ml.

Positive pressure ventilation was provided by a volume preset ventilator (Cape) with a fixed 1 : 2 ratio of 1 : 2. The expired gas was separated from the gas compressed in the ventilator tubing by a collect valve (Sykes, 1969) and the positive end-expiratory pressure was generated by the same underwater blow-off valve used during CPAP. The frequency was set so that it was as close as possible to that during spontaneous breathing (minimum ventilator rate 10 b.p.m.). Tidal volume was adjusted to produce an end-tidal carbon dioxide tension in the range 4.7–5.3 kPa.

Cannulae for pressure measurements and blood sampling were placed in the aorta via the left femoral artery, in the right atrium via the left femoral vein, in the main pulmonary artery via the right jugular vein and a balloon catheter was passed into a pulmonary artery via the left jugular vein for pulmonary capillary wedge pressure measurements. Cannulae for dye injection and sampling during cardiac output measurements were placed in the pulmonary artery or right atrium via the right femoral vein and in the aorta via the right femoral artery. The position of all cannulae was checked from the pressure waveforms. Oesophageal pressure was measured inside a 10-cm latex balloon containing 0.2 ml air positioned cephalad to the point of maximum cardiac oscillations.

After completing the arterial and venous cannulations the dogs were maintained in each of the five patterns of ventilation for at least 30 min. The sequence in which the five patterns were applied was changed for each experiment (table I). Pressures were recorded continuously using strain-gauges, the outputs from which were fed to a six-channel, hot-wire chart recorder (Devices M19). The zero for intravascular pressures was taken as the level of the junction of the anterior third and the posterior two-thirds of the antero-posterior diameter of the chest. The strain-gauges were calibrated repeatedly against a column of saline. Airway and oesophageal pressures were calibrated separately against a column of water. Mean pressures were derived electronically.

Gas volumes were measured using a calibrated dry-gas meter or a spirometer. An infra-red analyser (Hartmann-Braun URAS 4) was used to measure end-tidal and mixed expired carbon dioxide concentrations. Mixed expired oxygen concentration was measured with a paramagnetic oxygen analyser (Servomex 101A). Duplicate gas samples were checked using oxygen and carbon dioxide electrodes.

Cardiac output was determined as the mean of duplicate measurements at the beginning of each gas collection. The indocyanine green dye-dilution method was used. The output from the densitometer (Gilford 103) was recorded and the cardiac output
calculated from co-ordinates of the resulting curve by the method of Simons and White (1976).

Arterial and mixed venous blood samples were obtained during the second half of each gas collection in heparinized syringes and were analysed immediately. Oxygen tension, carbon dioxide tension and pH were measured in duplicate on separate electrode systems (ABL-I and PHM-1, Radiometer, Copenhagen). The oxygen content of arterial and mixed venous blood was measured directly in an electrolytic cell following displacement of the oxygen from the haemoglobin by carbon monoxide (Lex-O₂-Con, Lexington Instruments Corp.). This method has been shown to be comparable with the Van Slyke method (Selman, White and Tait, 1975). Haemoglobin concentration was determined using the cyanmethaemoglobin method.

Correction factors for temperature, blood-gas factors of the oxygen electrodes and calculations using standard respiratory formulae were made with a digital computer and the program of Adams (1970). Arterial and mixed venous oxygen saturations were derived from the dissociation curve for dog haemoglobin (Rossing and Cain, 1966). Venous admixture (Qj&t) was calculated using the formula:

\[ (C \cdot O₂ - C \cdot V₂) / (C \cdot O₂ - C \cdot V₂) \]

\( Q_s / \hat{Q}_t \) was calculated using the formula:

\[ Q_s / \hat{Q}_t = \frac{C \cdot O₂ - C \cdot V₂}{C \cdot O₂ - C \cdot V₂} \]

An error may occur when oxygen content derived from oxygen tension is used in the shunt formula. Because of the change in slope of the oxyhaemoglobin dissociation curve between arterial and mixed venous points, a small shift of the curve will alter the mixed venous oxygen content more than the arterial content. This error can be minimized by inserting the measured arteriovenous oxygen content difference in the modified shunt equation.

Statistical analysis of the results was made in two stages. Initially, the differences between mean values were compared using two-way analysis of variance between dogs and between respiratory patterns. In a second separate analysis measurements in which there were significant \( P<0.05 \) differences between patterns of ventilation were compared using Student's \( t \) test for paired data values in each combination of the five patterns of ventilation.

RESULTS

Minimum airway pressure during inspiration was significantly \( P<0.001 \) less during CPAP (bag) than during CPAP (bellows). There were no significant differences between CPAP (bag) and CPAP (bellows) in any of the other indices of cardiovascular or respiratory function which were studied (tables II, III, IV).

Mean oesophageal pressure was least during spontaneous ventilation and increased during IPPV, CPAP and CPPV. Mean right atrial pressure tended to increase also with increasing oesophageal pressure, although the differences were on average 30% greater. Mean pulmonary artery pressure and mean pulmonary wedge pressure increased also, but in these instances the largest difference occurred between spontaneous ventilation and the other patterns of ventilation. Mean systemic arterial pressure and cardiac output, measured either by dye-dilution or using the direct Fick principle, did not differ significantly between the five groups (table II).

Minute volume was slightly greater during spontaneous ventilation than during the other patterns of ventilation. There was a marked difference between respiratory frequency during SV (15 b.p.m.) and

<table>
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<tr>
<th>Experiment No.</th>
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<th>Sample 2</th>
<th>Sample 3</th>
<th>Sample 4</th>
<th>Sample 5</th>
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<tbody>
<tr>
<td>1</td>
<td>SV</td>
<td>CPAP (bag)</td>
<td>CPAP (bell)</td>
<td>IPPV</td>
<td>CPPV</td>
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<td>IPPV</td>
<td>CPAP (bag)</td>
<td>SV</td>
<td>CPAP (bell)</td>
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<td>CPAP (bell)</td>
<td>CPPV</td>
<td>SV</td>
<td>CPAP (bag)</td>
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<td>CPPV</td>
<td>SV</td>
<td>CPAP (bell)</td>
<td>IPPV</td>
</tr>
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<td>CPAP (bell)</td>
<td>IPPV</td>
</tr>
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<td>CPAP (bell)</td>
<td>CPPV</td>
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<tr>
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<td>SV</td>
<td>CPPV</td>
<td>IPPV</td>
<td>CPAP (bell)</td>
<td>CPPV</td>
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TABLE II. Mean values (± SD) and number of observations of cardiovascular variables during five different patterns of ventilation. Mean values which differ significantly (P < 0.05) from other groups are indicated by the superscript: a = different from spontaneous ventilation; b = different from IPPV; c = different from CPAP (bag); d = different from CPAP (bellows); e = different from CPPV

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Cardiac output (litre min⁻¹)</th>
<th>( P_{AMT} ) (kPa)</th>
<th>( P_{PA} ) (kPa)</th>
<th>( P_{PCW} ) (kPa)</th>
<th>( P_{RA} ) (kPa)</th>
<th>( P_{DES} ) (kPa)</th>
<th>( P_{RA} - P_{DES} ) (kPa)</th>
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<td>Spontaneous ventilation</td>
<td>19.5 ± 3.3</td>
<td>1.63de</td>
<td>0.30e</td>
<td>0.02de</td>
<td>-0.30de</td>
<td>0.34</td>
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<td>11</td>
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<tr>
<td>IPPV</td>
<td>18.6 ± 3.2</td>
<td>2.18</td>
<td>0.61</td>
<td>0.22de</td>
<td>-0.13e</td>
<td>0.38</td>
<td>±0.13e</td>
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<td>CPAP (bag)</td>
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<td>0.69ae</td>
<td>0.47abo</td>
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<td>0.48</td>
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<tr>
<td>CPAP (bellows)</td>
<td>18.5 ± 3.7</td>
<td>2.40a</td>
<td>0.78</td>
<td>0.41abo</td>
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<tr>
<td>CPPV</td>
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<td>2.62a</td>
<td>0.90ae</td>
<td>0.61abd</td>
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<td>10</td>
<td>10</td>
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</tbody>
</table>

TABLE III. Mean values (± SD) and number of observations of respiratory variables during five different patterns of ventilation. Mean values which differ significantly (P < 0.05) from other groups are indicated by the superscript: a = different from spontaneous ventilation; b = different from IPPV; c = different from CPAP (bag); d = different from CPAP (bellows); e = different from CPPV

<table>
<thead>
<tr>
<th>Pattern</th>
<th>( V_E ) (litre min⁻¹)</th>
<th>( V_T ) (ml)</th>
<th>( f ) (b.p.m.)</th>
<th>( V_D/V_T ) (%)</th>
<th>( V_O_2 ) (mmol min⁻¹)</th>
<th>( V_CO_2 ) (mmol min⁻¹)</th>
<th>Oxygen flux (mmol min⁻¹)</th>
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<tr>
<td>Spontaneous ventilation</td>
<td>6.37 ± 2.11</td>
<td>464d</td>
<td>15.2ed</td>
<td>32e</td>
<td>7.60</td>
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<tr>
<td>IPPV</td>
<td>5.43 ± 1.04</td>
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<td>11.5ed</td>
<td>33e</td>
<td>7.73</td>
<td>6.17</td>
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<tr>
<td>CPAP (bag)</td>
<td>5.54 ± 1.64</td>
<td>612bde</td>
<td>8.9bde</td>
<td>29e</td>
<td>6.71</td>
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<td>CPAP (bellows)</td>
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<tr>
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<td>5.55 ± 1.0</td>
<td>480ed</td>
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</table>

CPAP (9 b.p.m.) and this was associated with a 30% increase in tidal volume during CPAP. Deadspace/tidal volume ratio was increased during continuous positive pressure ventilation only. Oxygen consumption, carbon dioxide production and oxygen flux (the product of arterial oxygen content and cardiac output) did not differ significantly between the five patterns (table III).

Alveolar oxygen tension, arterial oxygen tension and arterial oxygen content were all greatest during SV. The increased minute volume during SV was reflected in a smaller arterial carbon dioxide tension. Alveolar-arterial oxygen tension difference and venous admixture were also least during SV. None of these differences was statistically significant (table IV).
DISCUSSION

The most striking effect of CPAP was the 40% decrease in respiratory frequency. When initiating CPAP there was often quite prolonged breath-holding which could be terminated immediately by restoring airway pressure to atmospheric. This effect was increased by deeper anaesthesia. Breath-holding was so prolonged when chloralose was used as the anaesthetic agent that it proved impossible to initiate CPAP during chloralose anaesthesia. Stanley and others (1975), studying phrenic nerve activity in dogs, during positive pressure ventilation, have shown that cardiac output decreases when 0.98 kPa end-expiratory pressure was applied during CPAP (bellows). This compares with an increase of 32% of applied airway pressure during CPAP in normal infants (Powers and Swyer, 1975).

Mean oesophageal pressure increased by 25% of applied airway pressure during CPAP (bag) and 29% during CPAP (bellows). This compares with an increase of 32% of applied airway pressure during CPPV in normal infants (Powers and Swyer, 1975). These increases in mean intrathoracic pressure did not produce any significant change in cardiac output. Many studies in dogs under pentobarbitone anaesthesia have shown that cardiac output decreases during positive airway pressure breathing (Lenfant and Howell, 1960) or during CPPV (Colgan, Barrow and Fanning, 1971; Jones and King, 1973; Giordano and Harken, 1975).

Qvist and co-workers (1975), studying dogs ventilated with halothane, found that, although right atrial pressure increased following the addition of 12 mm Hg positive end-expiratory pressure, this increase was smaller than the increase in pleural pressure. There was therefore a net decrease in transmural right atrial pressure and this was associated with a decrease in cardiac output. Furthermore, a transfusion which restored transmural right atrial pressure to control value also reversed the decrease in cardiac output. They concluded that a decrease in net...
ventricular filling pressure was responsible for the observed decrease in cardiac output. On the contrary, in our experiment, right atrial pressure increased more than oesophageal pressure during the five patterns of ventilation.

Considerable differences occur in intrathoracic pressure measurements, depending on the site at which they are made and the position of the subject. In particular, pressure measurements in oesophageal balloons are known to be greater than pressures close to the left and right atria (Brookhart and Boyd, 1947; Coleridge and Linden, 1954). However, if mean right atrial pressure minus mean oesophageal pressure is an index of right ventricular filling pressure and mean pulmonary capillary wedge pressure minus mean oesophageal pressure is an index of left ventricular filling pressure, there was no significant change in the values of these indices between groups in our study. This probably accounts for our finding that cardiac output did not decrease during positive pressure breathing or ventilation. A possible explanation of the discrepancy between our findings and those of other workers may be the effect of different anaesthetic techniques used in the experiments. In dogs, thiopentone causes paralysis of sympathetic vasoconstrictor responses of a brief duration compared with pentobarbitone (Strandness et al., 1964). Jones and King (1973) have demonstrated, in dogs, that during pentobarbitone anaesthesia CPPV is associated with greater decreases in cardiac output than during chloralose anaesthesia. They attributed this finding to the preservation of peripheral vascular reflexes during chloralose administration enabling compensation for an increase in intrathoracic pressure to occur by increased peripheral venous tone.

There were small differences between mean values of alveolar and arterial partial pressures of oxygen in the five groups. Although the study was designed to keep arterial carbon dioxide tension as near to constant as possible, mean arterial carbon dioxide tension did vary slightly between the groups and this is sufficient to account for the observed differences in alveolar oxygen tension. Alveolar–arterial $P_{O_2}$ difference was very similar in all five groups and so the arterial oxygen tension reflected changes in alveolar oxygen partial pressure. None of these differences was statistically significant.

In man, the reported benefits of CPPV (Ashbaugh and Petty, 1973) and CPAP (Civetta, Brons and Gabel, 1972) in increasing arterial oxygen tension and reducing venous admixture occur only when improved distribution of ventilation–perfusion occurs. This will occur when FRC at ambient pressure is reduced to a value at which airway closure is occurring in dependent lung zones during all or part of normal tidal exchange. Under these circumstances an increase in FRC as a result of either CPAP or CPPV would be expected to improve ventilation to dependent lung zones because it is not accompanied by an increase in closing capacity (Abboud et al., 1975). However, in normal subjects where airway closure is taking place well below FRC, there will be no such redistribution of ventilation and no improvement in arterial oxygenation. In dogs complete occlusion of airways often does not prevent ventilation of lung units distal to the occlusion because of the availability of channels for collateral ventilation. Gas exchange in collaterally ventilated lung units may be relatively normal (Macklem, 1971) in addition, there is evidence that all terminal airspaces in dogs remain in continuity with the trachea until transpulmonary pressure has decreased to $-2$ cm $H_2O$ (Cavagna, Stemmier and Dubois, 1967).

Recently, Wagner and colleagues (1975) have shown that the pulmonary shunt fraction during IPPV in supine anaesthetized dogs is only 0.6% of cardiac output when measured by the inert gas infusion method which excludes shunt from the bronchial and Thébésian circulations. For these reasons normal dogs do not make a good model for comparing the effect of CPAP and CPPV on gas exchange.

We conclude that, under the conditions of this study, cardiac output is not affected by changes in intrathoracic pressure with 10 cm $H_2O$ positive end-expiratory pressure during either CPAP or CPPV. Any improvement in arterial oxygenation depends largely on the extent by which an increase in FRC improves ventilation/ perfusion matching in the lungs.

ACKNOWLEDGEMENTS

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REFERENCES


COMPARISON OF CPAP AND CPPV IN DOGS


COMPARAISON DES EFFETS CARDIORESPIRATOIRES DE LA RESPIRATION SOUS PRESSION POSITIVE CONTINUE DES PASSAGES D'AIR ET DE LA VENTILATION SOUS PRESSION POSITIVE CONTINUE SUR DES CHIENS

RESUME

On a compare cinq types de ventilation sur des chiens: (1) ventilation spontanée à la température ambiante (SV); (2) IPPV (ventilation sous pression positive intermittente); (3) ventilation spontanée sous une pression positive des passages d'air de 0,98 kPa, effectuée à partir d'un circuit de non-reinhalation comportant un sac réservoir de 5 litres et un débit de gaz frais de deux fois le volume/min (CPAP (sac)); (4) ventilation spontanée sous pression positive des passages d'air de 0,98 kPa, le sac réservoir étant remplacé par un soufflet lesté (CPAP (soufflet)) et (5) IPPV sous pression positive de 0,98 kPa à la fin de la période expiratoire (CPPV). La CPAP a diminué d'une manière significative le rythme de la respiration par rapport à SV. La CPAP (soufflet) a eu pour résultat de donner une augmentation importante du volume courant. La pression oesophagienne moyenne, la pression de l'oreillettre droite, la pression pulmonaire cunéiforme et la pression artérielle pulmonaire ont augmenté pendant l'IPPV, la CPAP (sac), la CPAP (soufflet) et la CPPV par rapport à la SV. Il n'y a eu aucune variation importante dans la pression artérielle systémique moyenne, dans le débit cardiaque, la PaO2, la PaCO2, la CaO2, (CaO2-CvO2) (PaO2-PaCO2) dans le mélangé veineux pulmonaire. Dans les conditions dans lesquelles cette étude a été effectuée, le transport d'oxygène n'a pas été modifié par la ventilation sous pression positive des passages d'air.
VERGLEICH DER KARDIORESPIRATORISCHEN WIRKUNGEN EINER KONTINUIERLICHEN ATMUNG MIT POSITIVEM LUFTWEGDRUCK MIT KONTINUIERLICHER POSITIVER DRUCKBELÜFTUNG BEI HUNDEN

ZUSAMMENFASSUNG

Fünf Belüftungsmethoden wurden bei Hunden untersucht: (1) spontane Belüftung bei Umgebungsdruck (SV); (2) intermittierende positive Druckbelüftung (IPPV); (3) spontane Belüftung bei positivem Luftwegdruck von 0,98 kPa, geliefert von einem nicht-rückgeatmeten Kreislauf mit einem 5 Liter-Reservoir und einem Frischgasstrom von doppeltminutenvolumen (CPAP (Sack)); (4) spontane Belüftung bei positivem Luftwegdruck von 0,98 kPa, wobei der Reservoirsack durch einen beschwerten Blasbalg ersetzt war (CPAP (Blasbalg)); und (5) IPPV mit einem positiven Endausatmungsdruck von 0,98 kPa. Durch CPAP wurde die Atmungsrate im Vergleich zu SV wesentlich verringert. CPAP (Blasbalg) führte zu einem wesentlichen Anstieg des Atemvolumens. Der mittlere Speiseröhrendruck, der Druck in der rechten Kammer, der Lungenkeldruck und der lungenarterielle Druck stiegen bei IPPV, CPAP (Sack), CPAP (Blasbalg) und CPPV, verglichen mit SV. Es gab keine wesentlichen Veränderungen bei mittlerem systemischen arteriellen Druck, im Herzminutenvolumen, bei $P_{aO_2}$, $P_{aCO_2}$, $CaO_2$ ($CaO_2-CvO_2$), $(P_{aO_2}-P_{aO_2})$, oder bei der lungenvenösen Beimischung. Unter den bei dieser Studie herrschenden Bedingungen wurde der Sauerstofftransport durch Belüftung mit positivem Luftwegdruck nicht verändert.

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COMPARACION DE LOS EFECTOS KARDIORESPIRATORIOS DE RESPIRACION CON PRESION POSITIVA CONTINUA EN LAS VIAS RESPIRATORIAS Y VENTILACION DE PRESION POSITIVA CONTINUA EN PERROS

SUMARIO

Se han comparado cinco tipos de ventilación en perros: (1) Ventilación espontánea a presión ambiental (SV); (2) Ventilación con presión positiva intermitente (IPPV); (3) Ventilación espontánea a 0,98 kPa de presión positiva en las vías respiratorias, entregado por un circuito de aspiración no repetida que incorpora una bolsa de depósito de 5 litros y una circulación de gas fresco con doble el volúmen-minuto (CPAP (bolsa)); (4) ventilación espontánea a 0,98 kPa de presión en las vías respiratorias, reemplazándose la bolsa de depósito por un fuelle contrapesado (CPAP (fuelle)) y un (5) IPPV con 0,98 kPa de presión expiratoria final positiva (CPPV). El CPAP disminuyó significativamente la rapidez de la respiración en comparación con el SV. El CPAP (fuelle) causó un aumento significativo en el volumen respiratorio. La presión esofágica media, presión atrial derecha, presión de acuñado pulmonar y presión arterial pulmonar aumentaron durante IPPV, CPAP (bolsa), CPAP (fuelle) y el CPPV estuvo al nivel de la SV. No se produjeron cambios significativos en la presión arterial general media, capacidad cardíaca, $P_{aO_2}$, $P_{aCO_2}$, $CaO_2$, ($CaO_2-CvO_2$), $(P_{aO_2}-P_{aO_2})$, o mezcla venosa pulmonar. Bajo las condiciones de este estudio no se vio alterado el transporte del oxígeno por la ventilación de presión positiva en las vías respiratorias.