ALVEOLAR DEADSPACE DURING HIGH FREQUENCY POSITIVE PRESSURE VENTILATION

Influence of ventilatory pattern

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SUMMARY

Pulmonary gas exchange was studied in association with high frequency ventilation and its relation to the duration of insufflation and end-expiratory pressure investigated. Alveolar deadspace, alveolar ventilation and the alveolar-arterial oxygen difference were obtained in cats receiving a constant minute ventilation. Alveolar deadspace increased with ventilatory frequency when a long insufflation time was used. A positive end-expiratory pressure (PEEP) decreased the alveolar deadspace in high frequency ventilation. Thus, with the low compressible volume ventilator, more efficient high frequency ventilation can be achieved with a short period of insufflation plus low PEEP.

Previous studies of high-frequency positive pressure ventilation (Jonzon et al., 1971; Jonzon, Sedin and Sjöstrand, 1973) demonstrated that adequate alveolar ventilation could be achieved with small tidal volumes and at low intratracheal and intrapleural pressures when a ventilator system with a low internal compliance and a small deadspace was used (Eriksson et al., 1977). These earlier studies, however, posed additional questions. For example, did the physiological and alveolar deadspaces alter when ventilation was altered from low to high frequency, and were the deadspaces influenced by the ventilatory pattern at high frequencies?

The aim of the present study was to investigate the changes in alveolar deadspace, alveolar ventilation and alveolar-arterial oxygen difference produced by different ventilatory patterns in association with high frequency ventilation. In addition, intratracheal and intrapleural pressures were measured and the transpulmonary pressure calculated to determine whether the deadspaces might be related to the degree of, or the variation in, the expansion of the lung.

In this study, the ventilatory frequency and the duration of insufflation were varied, while the minute ventilation was kept constant. This allowed arterial PCO₂ and PO₂ to change within the normal range, and avoided alterations in cardiac output and venous admixture (Prys-Roberts et al., 1967; Stone and Sullivan, 1970).

MATERIALS AND METHODS

Animals, anaesthesia and preparation

Five young healthy cats (weight 2.7–4.0 kg (mean 3.5 kg)) were used. Anaesthesia was induced with chloroform and maintained with an intermittent infusion of chloralose i.v. An endotracheal tube was inserted until its tip lay about 1 cm above the carina. A ligature was tied around the trachea to prevent leakage of gas around the tube. Dextrose solution 5.5% was infused slowly during the surgical preparation and the definitive experiment. Care was taken to maintain normal body temperature.

A catheter was inserted via the femoral artery to the thoracic part of the aorta for measurement of arterial pressure and withdrawal of blood samples. A second catheter was inserted via the femoral vein to a central vein for measurement of central venous pressure (CVP). The tip of this catheter was placed at the level of the right atrium. A third catheter was introduced through the rib-cage into the pleural space for measurement of intrapleural pressure. A fourth catheter was placed in the endotracheal tube for measurement of intratracheal pressure.

Measurements

Arterial pressure, CVP, intratracheal pressure and intrapleural pressure were measured with transducers (SE Labs/EMI/Ltd, GB). The signals were...
amplified with a six-channel amplifier (Elcomatic EM 760, GB) and recorded on an oscillograph (Oscillograph 6008, SE Labs Ltd, GB).

Expired gas was collected in a latex-rubber bag (Mischbeutel, E. Jaeger AG, GFR). The expiratory tidal volume was measured with a Krogh spirometer. Arterial \( PO_2 \), \( PCO_2 \), base excess (BE) and oxygen saturation were determined using an automatic analyser (ABL 2, Radiometer, Denmark).

Carbon dioxide concentrations were measured with a carbon dioxide analyser (Datex CD 101, Pediatric option, Datex Industries Oy, Finland). The sampling rate was 50 ml min\(^{-1}\) and gas was sampled via a catheter inserted 3 cm to the endotracheal tube. Sampling further down the endotracheal tube did not alter the results of the carbon dioxide measurements.

**Mechanical ventilation**

A fluidic-operated, constant-flow, time-cycled ventilator (Relog Infant Respirator G-19, Farum, Poland), giving a constant inspiratory flow of gas, was used. The ventilatory frequency and duration of insufflation (in per cent of the ventilatory cycle) can be set independently: ventilatory frequency can be varied between 50 and 120 b.p.m. in steps of 5 b.p.m. and the insufflation period in steps of 5\% from 20\% to 50\% of the ventilatory cycle. The static compliance of the respirator, patient circuit and humidifier ranges from 1.25 to 4.0 ml kPa\(^{-1}\), depending on the setting of the respirator (Rondio and Ryterski, 1978).

**Experimental programme**

During ventilation with the first respiratory setting (*vide infra*) in each animal, arterial blood-gas tensions were measured to ensure that alveolar ventilation was adequate. The minute ventilation was then kept constant. The order of ventilatory settings was varied between the cats to eliminate the effects of time-related changes in compliance. At each setting, the ventilation was maintained steady for at least 10 min and at the end of each period, acid–base status was determined, the concentration of carbon dioxide in end-expiratory and mixed expiratory gas was measured, and the various pressures recorded.

Two ventilatory frequencies (60 and 100 b.p.m.) and two periods of insufflation (20\% and 35\% of the ventilatory cycle), with and without a positive end-expiratory pressure (PEEP) of 0.5 kPa (\( P_e \)) were studied. With the shorter period of insufflation a PEEP of 0.25 kPa (\( P_{P2} \)) was used also.

**Calculations and analyses of data**

The anatomical deadspace was calculated from the equation:

\[
V_D^{an} = VT \frac{P_A - P_E}{P_A - P_{Ee}}
\]

where \( VT \) was the tidal volume, \( P_A \) the partial pressure of carbon dioxide in alveolar gas (kPa), and \( P_E \) and \( P_{Ee} \) the partial pressures of mixed expired gas and inspired gas (kPa) respectively.

The physiological deadspace was calculated as follows:

\[
V_D^{phys} = VT \frac{P_A - P_E}{P_A - P_{Ee}}
\]

where \( P_A \) was the partial pressure of carbon dioxide in arterial blood (kPa).

The alveolar deadspace was calculated as the difference between physiological and anatomical deadspace. Minute ventilation was calculated as a product of tidal volume and ventilatory frequency (\( f \)).

The alveolar ventilation was calculated from the relationship:

\[
V_A = \frac{VE CO_2}{CA CO_2}
\]

where \( VE CO_2 \) was the volume of expired carbon dioxide (litre min\(^{-1}\)) and \( CA CO_2 \) was the concentration of carbon dioxide in alveolar gas (\%)

Ventilation of the deadspace was calculated as a product of frequency and deadspace.

The alveolar–arterial \( PO_2 \) difference (\( PAO_2 - PaO_2 \)) was calculated from the measured \( PAO_2 \) and \( PaO_2 \). The latter was calculated from the alveolar air equation:

\[
PAO_2 = FI O_2 (PB - 47) - PA CO_2 / 0.8,
\]

where \( FI O_2 \) was the fraction of inspired oxygen, \( PB \) the barometric pressure and 0.8 was the anticipated respiratory quotient.

All gas volumes were corrected to BTPS. Statistically significant differences were determined by testing paired observations (Student's \( t \) test). Thus, the comparisons are intraindividual and no standard deviations or standard errors are given in the figures.

**RESULTS**

The anatomical and physiological deadspaces were smaller at a ventilatory frequency of 100 b.p.m. than at 60 b.p.m. (\( P<0.001 \)). The anatomical \( V_D / VT \) was
Alveolar deadspace

**Frequency.** $V_{D}^{alv}$ was larger at 100 than at 60 b.p.m. both with and without PEEP, but only when the insufflation period was 35% ($P < 0.05$).

**Insufflation.** At 60 b.p.m. $V_{D}$ was almost the same with insufflation periods of 35% and 20% of the ventilatory cycle. At 100 b.p.m. $V_{D}^{alv}$ was somewhat larger with insufflation periods of 35% (fig. 1), but the difference was not significant.

**PEEP.** At both ventilatory frequencies $V_{D}^{alv}$ tended to be less with a PEEP of 0.25 kPa ($P_2$) than without PEEP (60 b.p.m.: $P < 0.05$; 100 b.p.m.; $P < 0.08$).

Alveolar $V_{D}/VT$ (fig. 1; lower).

**Frequency.** Alveolar $V_{D}/VT$ was significantly greater at 100 than at 60 b.p.m. when the insufflation period was 35% both with and without PEEP ($P < 0.01$), but with an insufflation of 20% only without PEEP ($P < 0.05$).

**Insufflation.** During ventilation at 100 b.p.m. with PEEP ($P_2$), $V_{D}^{alv}/VT$ was greater at an insufflation period of 35% than at 20% ($P < 0.05$).

**PEEP.** $V_{D}^{alv}/VT$ was invariably less with than without PEEP, although the differences were small. A PEEP of 0.25 kPa ($P_2$) seemed to have the same effect as one of the 0.5 kPa ($P_2$) on $V_{D}^{alv}/VT$ (fig. 1; lower right).

Alveolar ventilation

The alveolar ventilation ($\bar{V}_{A}$) was less at 100 b.p.m. (fig. 2) than at 60 b.p.m. only when no PEEP was used (insufflation period of 20%: $P < 0.05$; 35%: $P < 0.01$).
Other variables

\((P_{AO2} - P_{A02})\) was greater during ventilation at 100 than at 60 b.p.m. except with use of 35% insufflation and no PEEP \((P<0.05; \text{fig. 2).}\)

The arterial \(P_{O2}\) was clearly lower \((P<0.05)\) at a ventilatory frequency of 100 b.p.m. combined with an insufflation period of 20% or 35% than at 60 b.p.m. and 20%. At a frequency of 60 b.p.m. and an insufflation period of 35%, \(P_{O2}\) was higher with a PEEP of 0.5 kPa than without a PEEP \((P<0.05)\).

The arterial \(P_{CO2}\) was always greater when ventilation was given at 100 b.p.m. than at 60 b.p.m. \((P<0.05)\) and when PEEP \((P_{2})\) was used.

When arterial blood-gas tensions were related to the duration of expiration it was found that \(P_{O2}\) was greater when the duration of expiration was longer, whereas \(P_{CO2}\) was smaller. The shortest expiratory time (100 b.p.m., insufflation period 35%) was accompanied by smaller variations in transpulmonary pressure than longer expiratory times (fig. 2). When PEEP was used the transpulmonary pressure differences were of the same order of magnitude.

At all settings, ventilation induced low maximal intratracheal pressures. These pressures did not exceed 1.0 kPa when no PEEP was used, or 1.2 kPa with PEEP 0.5 kPa. The maximal intrapleural pressures were always negative. No significant changes in arterial pressure, CVP or heart rate were observed in relation to the different ventilator settings.

DISCUSSION

This study shows that the alveolar deadspace was influenced by the ventilatory pattern in high frequency positive pressure ventilation. Thus, to maintain an unchanged alveolar deadspace when the ventilatory frequency is increased, the duration of insufflation should be shortened. The addition of PEEP reduces alveolar deadspace.

Our findings indicate that, for each frequency in high frequency ventilation, the choice of insufflation time (% of ventilation cycle), and of PEEP, has an important influence on the alveolar deadspace. No conclusion as to the best combination of frequency, insufflation period and PEEP when the lungs are of normal compliance can be drawn from the present study, but this could be investigated by keeping alveolar ventilation constant instead of minute ventilation. The best combination of ventilator settings might also be altered with changes in resistance or compliance (Nunn, 1977).

A ventilator with a low internal volume and a low compliance is required to generate a high flow rate during a short insufflation period (Eriksson et al., 1977). When using ventilator systems with a low compressible volume, Borg and colleagues (1981) found that \(V_{D}/V_{T}\) increased much less with increasing ventilatory frequency than when a ventilator with a larger compressible volume was used.

In the present study it was observed that \(V_{D}/V_{T}\) was always smaller when ventilation was given with PEEP. We expected that the alveolar ventilation would be less at the higher ventilatory frequency and smaller tidal volumes because of an increase in deadspace ventilation. However, a significantly lower alveolar ventilation at the higher ventilatory frequency was only obtained when PEEP was not used. In addition, the changes in transpulmonary pressure increased with PEEP. These changes with-
out PEEP were similar with all ventilator settings except 100 b.p.m. with 35% insufflation, which had the shortest expiratory time.

No previous study on alveolar deadspace in the ventilatory frequency range 60–100 b.p.m. has been reported and almost all studies with low frequencies have been made with longer insufflation periods. In one investigation, using ventilatory frequencies up to 40 b.p.m. and an insufflation period of 33%, Severinghaus and Stupfel (1957) found that the alveolar deadspace decreased slightly with faster ventilatory frequencies (up to 40 b.p.m.), increased with larger tidal volumes and decreased with larger lung volumes. Their finding concerning lung volume seems to be valid also for high frequency ventilation. Thus, in the present study, at a ventilatory frequency of 100 b.p.m. and an insufflation period of 20% (a setting that gives a functional residual capacity similar to that during spontaneous breathing (Jonzon, Rondio and Sedin, 1980)), the alveolar deadspace diminished when the lung was expanded with low PEEP. An increasing lung volume tended to result in less alveolar deadspace at all settings. This indicates that the lung and its alveoli were not hyperinflated with the given ventilation. Instead, to judge from \((P_{A\mathrm{O}_2} - P_{A\mathrm{O}_2})\), which increased with ventilatory frequency but was similar with both durations of insufflation, the frequency-related differences in alveolar deadspace were preferentially caused by hypoventilated or non-ventilated alveoli. Consequently, the unchanged alveolar deadspace with increasing frequency at short insufflation and PEEP must be caused either by a more favourable pulmonary perfusion or by more efficient gas mixing secondary to turbulence in conducting airways (Wattwil et al., 1983). Thus, low PEEP, which was invariably used in our early studies of HFPPV (Jonzon et al., 1971; Jonzon Sedin and Sjöstrand, 1973), is of great importance for ventilatory efficiency.

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REFERENCES


ESPACE MORT ALVEOLAIRE AU COURS DE LA VENTILATION A HAUTE FREQUENCE ET PRESSION POSITIVE

Influence du schema ventilatoire

RESUME

Nous avons étudié l'hématose au cours de la ventilation à haute fréquence et recherché l'influence respective de la durée d'insufflation et de la pression de fin d'expiration. Nous avons mesuré l'espace mort alvéolaire, la ventilation alvéolaire et la différence des pressions d'oxygène alvéolo-artérielle chez des chats soumis à une ventilation-minute constante. L'espace mort alvéolaire augmente avec la fréquence ventilatoire lorsqu'on utilise un temps d'insufflation long. Une pression positive continue (PEEP) diminue l'espace mort alvéolaire en ventilation à haute fréquence. Ainsi avec un respirateur ayant un faible volume compréssible, on peut obtenir une ventilation à haute fréquence plus efficace en associant à une faible durée d'insufflation une légère PEEP.
ALVEOLARTOTRAUM WÄHREND
HOCHFREQUENTER POSITIVER
DRUCKBEATMUNG
Einfluß des Beatmungstyps

ZUSAMMENFASSUNG

Bei hochfrequenter Beatmung wurde der pulmonare Gasaus-
thaushalt und die Relation zur Dauer der Insufflation und zum
endexspiratorischen Druck untersucht. Bei Katzen wurde bei
konstantem Minutenvolumen der alveolare Totraum, die alveol-
are Ventilation und die alveolo—arterielle Sauerstoffdifferenz
bestimmt. Der alveolare Totraum war erhöht, wenn bei hochfre-
quenter Beatmung eine lange Insufflationszeit gewählt wurde.
Ein positiver endexspiratorischer Druck (PEEP) verringerte den
alveolaren Totraum bei Hochfrequenzbeatmung. Eine effizien-
tere Hochfrequenzbeatmung kann also bei Anwendung eines
niedrig kompressiblen Volumenbeatmungsgerätes mit einer kur-
ze Insufflationszeit und einem niedrigen PEEP erreicht werden.