ESTIMATION OF ARTERIAL Pco₂ DURING HIGH FREQUENCY JET VENTILATION

Studies in the Dog

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When healthy patients are anaesthetized and ventilated with normal tidal volumes at low frequencies, the arterial Pco₂ (Paco₂) may be estimated from the end-tidal Pco₂ (PETCO₂) by assuming an arterial to end-tidal Pco₂ difference (Paco₂ − PETCO₂) of 0.8–1.0 kPa (Nunn and Hill, 1960). However, during high frequency jet ventilation (HFJV), when small tidal volumes are delivered at frequencies between 1 and 10 Hz, (Paco₂ − PETCO₂) appears to increase as the frequency of ventilation increases (Capan et al., 1981; Rolly and Versichelen, 1983), so that the estimation of Paco₂ becomes inaccurate.

Whitwam and colleagues (1983), have shown that the normal (Paco₂ − PETCO₂) can be restored by the application of several large breaths during a brief interruption of HFJV at frequencies up to 1.7 Hz. The purpose of the present study was to determine whether the technique could be extended to frequencies up to 5 Hz, and to examine the restoration of the normal (Paco₂ − PETCO₂) in more detail.

MATERIALS AND METHODS

Five dogs of different breeds, weighing between 16 and 27 kg, were studied. After premedication with morphine 2–3 mg kg⁻¹, anaesthesia was induced with thiopentone 30 mg kg⁻¹ i.v. and pentobarbitone 2–3 mg kg⁻¹ i.v., and then maintained with a continuous infusion of pentobarbitone 1–2 mg kg⁻¹ h⁻¹. The trachea was intubated with a tracheal tube which had two narrow-bore tubes incorporated to its wall (Hi-Lo jet ventilation tube, Mallinkrodt, U.K.). The two small-bore tubes were used to monitor airway pressure and carbon dioxide concentration while the lungs were ventilated through the large lumen (9 mm i.d.) which was connected directly to the inflation valve of a Nuffield 200 ventilator (Penlon, U.K.).

During the periods of HFJV, the Nuffield 200 ventilator was replaced with a time-cycled solenoid-operated jet ventilator, driven by 100% oxygen supplied at 400 kPa. The pulses of oxygen from the ventilator were delivered to the centre of the lumen of the tracheal tube, through a 1.8-mm i.d. nozzle fixed into a T-piece connector (Bourgain, Mortimer and Sykes, 1986). To ensure

SUMMARY

The arterial to end-tidal Pco₂ difference (Paco₂ − PETCO₂) was measured in five anaesthetized dogs during controlled ventilation at 0.25 Hz (15 b.p.m.) and during high frequency jet ventilation at 1, 3 and 5 Hz. Because of the slow response of the infra-red carbon dioxide analyser, satisfactory recordings of end-tidal carbon dioxide could not be obtained at frequencies greater than 1 Hz. The interruption of high frequency jet ventilation by conventional ventilation resulted in approximately equal arterial and end-tidal Pco₂ values during the first breath, and restoration of the normal arterial to end-tidal Pco₂ difference by the third breath. It is concluded that, when high frequency jet ventilation at 1, 3 or 5 Hz is interrupted with normal tidal volumes at low frequencies, the arterial Pco₂ can be estimated from recordings of the end-tidal Pco₂.

that no carbon dioxide was entrained when the frequency was 1 Hz (Mortimer and Bourgain, 1983) a 15 litre min⁻¹ flow of oxygen was directed across the T-piece.

The carbon dioxide concentration was measured with an infra-red analyser (Gould Mark III), calibrated with oxygen and 10% carbon dioxide in oxygen, and the output displayed on a high frequency response ink-jet recorder (Siemens-Elema Mingograph). Gas was sampled at 180 ml min⁻¹ through silicone tubing 30 cm long and 2 mm i.d., connected directly to the inlet on the front panel of the instrument, thereby bypassing the water trap.

A femoral arterial catheter was used to permit monitoring of arterial pressure and to allow intermittent blood sampling. Arterial blood-gas analyses were performed with an automated blood-gas analyser (Radiometer ABL2, Denmark), the measurements being corrected to the temperature of the dog (38°C), which was monitored with a rectal thermistor and maintained at 38 ± 0.5°C by a heated table. Drugs and fluids were administered through a catheter placed in a femoral vein. The airway and arterial pressures were measured with Druck PDCR75 transducers and displayed on a heated stylus recorder (Lectromed M19, U.K.).

The preparation of each animal included the surgical removal of the upper and cardiac lobes of the left lung because the animals were also used for a separate study on the distribution of pulmonary blood flow. Blood loss during the surgical preparation was replaced with a colloid solution (Haemaccel Polygeline, Hoechst, U.K.); fluid balance during the investigation was maintained with 4% dextrose in 0.18% saline. Non-respiratory acidosis was corrected with an infusion of 8.4% sodium bicarbonate to maintain the base excess within ±1 mmol litre⁻¹. The arrangement of the equipment is shown in figure 1.

**Plan of investigation**

**First investigation.** Initially, the \( P_{a\text{CO}_2} \) was adjusted to 5.0 ± 0.2 (SD) kPa by altering the tidal volume using the inspiratory flow control of the Nuffield 200 ventilator, the inspiratory: expiratory (I:E) time ratio being 1:2 and the frequency 0.25 Hz. After conditions had stabilized, a control arterial blood sample was obtained and the end-tidal and minimal airway carbon dioxide concentrations recorded. High frequency jet ventilation was then applied, using an 1:E ratio of 1:2 and a frequency of 1, 3 or 5 Hz, the tidal volume being adjusted to produce a \( P_{a\text{CO}_2} \) similar to control conditions by varying the regulator governing the oxygen driving pressure. When
repeated blood-gas measurements indicated that conditions had been stable for 15–20 min, the measurements of blood and airway $P_{CO_2}$ were repeated. The T-piece was then rapidly disconnected from the tracheal tube and the Nuffield 200 ventilator reconnected without changing the previous settings, the airway carbon dioxide concentration being recorded continuously during the first eight breaths after the change of ventilator. High frequency jet ventilation was recommenced at a different frequency and another set of measurements obtained after 15–20 min. The inspired gas was 100% oxygen during all measurements and the three high frequencies were applied in random order, with two or more measurements at each frequency being obtained in each animal.

All the measurements of carbon dioxide concentration were recorded at ambient temperature and pressure saturated (ATPS). To compensate for the presence of water vapour in alveolar gas, each reading was corrected to body temperature and pressure saturated (BTPS) at 38 °C. The data have been compared by analysis of variance and paired $t$ tests where indicated. The significance level used was $P < 0.05$.

Second investigation. Since $(P_{a_{CO_2}} - P_{e_{CO_2}})$ may be influenced by the response characteristics of the capnograph and recorder (Jones, Robertson and Kane, 1979), a second study was carried out to define these—the capnograph and recorder being used in a manner similar to that in the animal study. The system described by Schena, Thompson and Crone (1984) was used for this purpose. A cylinder containing 5% carbon dioxide in oxygen was connected via a pressure regulator to a solenoid valve and the tip of the sampling tube from the carbon dioxide analyser placed in the outlet of the valve. Simultaneous recordings of the electrical signals supplied to the solenoid valve, and the capnograph response to a single step increase in carbon dioxide concentration from 0 to 5%, were obtained. Measurements were also made under pulsatile conditions cycling the solenoid valve at 1, 3 and 5 Hz, with an on-time of 55% of each cycle.

RESULTS

First investigation

There were no significant differences between $P_{a_{CO_2}}$ values at each of the frequencies of ventilation studied (table I), the mean value ($n = 51$) being $4.96 \pm 0.13$ (SD) kPa. During the control period of ventilation at 0.25 Hz, there was a significant difference ($P < 0.001$) of 0.98 kPa between the $P_{a_{CO_2}}$ and $P_{e_{CO_2}}$. The arterial to end-tidal $P_{CO_2}$ difference was similar in magnitude at 1 Hz, but at 3 Hz and 5 Hz, $(P_{a_{CO_2}} - P_{e_{CO_2}})$ increased (fig. 2). The minimal airway $P_{CO_2}$ was zero under control conditions, was just above zero at 1 Hz and increased progressively with frequency up to 5 Hz (table I, fig. 2).

The values of $P_{e_{CO_2}}$ observed during each of the first eight breaths after interruption of HFJV
The end-tidal $\text{PCO}_2$ during the first breath is significantly different from the control value ($P < 0.001$). There were no significant differences between the readings at 1, 3 and 5 Hz, so these have been combined. The $P_e'\text{CO}_2$ was significantly greater than the control values during the first breath ($P < 0.001$), variable in magnitude during the second breath and not significantly different from the control readings from the third breath onwards. Recordings of the expired carbon dioxide concentrations during the first three large breaths are shown in figure 4. The $P_e'\text{CO}_2$ recorded during the first breath after interruption of HFJV at all frequencies was $4.83 \pm 0.35$ kPa ($n = 35$). This was very similar to the measured arterial $\text{PCO}_2$ ($4.96 \pm 0.13$ kPa; $n = 51$). However, by the third breath after the interruption of HFJV, the $(P_a\text{CO}_2 - P_e'\text{CO}_2)$ was restored and the two values were significantly different ($P < 0.001$). Figure 5 shows that $(P_a\text{CO}_2 - P_e'\text{CO}_2)$ at the third breath was similar at 1, 3 and 5 Hz, when the $P_a\text{CO}_2$ was in the normal range.

Second investigation

The total delay time of the carbon dioxide measuring system was 635 ms. This comprised a transit time of 300 ms and 0–95% capnograph response time of 335 ms. Recordings of the 5% carbon dioxide mixtures sampled as step changes
at 1, 3 and 5 Hz are shown in figure 6. The peak value of 5% carbon dioxide was observed only at 1 Hz, the readings at 3 Hz and 5 Hz being less than 5%. During the periods when the solenoid valve was closed and room air was sampled, the minimum value of carbon dioxide recorded was 0.4% at 1 Hz, increasing to 2.8% at 3 Hz, and 3.8% at 5 Hz. Although the presence of carbon dioxide during this phase could be attributable to incomplete diffusion of carbon dioxide away from the sampling site, the damped response at frequencies greater than 1 Hz suggests that the limited frequency response of the analyser may have contributed to the observed results.

**DISCUSSION**

Our results indicate that, during HFJV at 1, 3 and 5 Hz, the $P_{a_{co}}$ may be estimated from measurement of the $P_{e_{co}}$ recorded during the first or third large breath.

When HFJV was interrupted with large tidal volume ventilation, we found that, during the first breath, $P_{a_{co_1}}$ and $P_{e_{co_1}}$ were similar and independent of the frequency of HFJV (4.96 ± 0.13 and 4.83 ± 0.35 kPa, respectively). Similar observations were made by Mihm and co-workers (1982) in a study in dogs in which HFJV at frequencies of 1.7–15 Hz was interrupted with a single breath of 15 ml kg⁻¹, or greater. They found a strong positive correlation between $P_{a_{co}}$ and $P_{e_{co}}$ ($r = 0.98; P < 0.001$) throughout the range of $P_{a_{co}}$ studied (3–10.7 kPa). Rearrangement of their linear regression equation yields the following relationship which may be used to predict the $P_{a_{co}}$:

$$P_{a_{co}} = 1.1 P_{e_{co}} - 0.3 kPa$$

Applying this equation to our results gives a predicted mean $P_{a_{co}}$ of 4.99 ± 0.38, whereas the measured mean $P_{a_{co}}$ was 4.96 ± 0.13 ($n = 35$). This extrapolation from the two studies suggests that the technique may be applicable over a wide range of combinations of frequency and tidal volume.

Our results also show that $(P_{a_{co_3}} - P_{e_{co_3}})$ measured during the third normal volume breath after interruption of HFJV at 1, 3 or 5 Hz, was similar to that measured during a more prolonged period of ventilation at normal tidal volume and frequency. Thus it may be used to provide an
alternative estimate of the \( P_{A\text{CO}_2} \). This confirms and extends the observation of Whitwam and colleagues (1983) at a frequency of 1 Hz.

The similarity between \( P_{A\text{CO}_2} \) and \( P_{E\text{CO}_2} \) during the first large breath was unexpected and not easy to understand. Two possible explanations must be considered, the first being concerned with the altered timing and depth of ventilation, and the second associated with a possible alteration in the distribution of ventilation and blood flow.

It can be predicted that the rate of transfer of carbon dioxide from the pulmonary capillary blood to the alveolus will depend on the difference in partial pressure of carbon dioxide between the blood and gas, and will thus be influenced by the cyclical dilution of alveolar gas during inspiration (Dubois, Britt and Fenn, 1951). It is well known that the input of carbon dioxide to the lung (pulmonary capillary blood flow) is influenced by cardiac systole and by changes in lung volume associated with breathing, but that the variations in flow are not great when the subject is resting (Lee and Dubois, 1955). However, in the dog, approximately 250 ml of fresh gas is transferred into the 1000 ml of gas in the functional residual capacity at each breath. If this change were to occur instantaneously, the alveolar gas would be diluted by 25\% (250/1000), that is from 6 kPa to 4.5 kPa. Although the degree of dilution is reduced by the fact that carbon dioxide continues to flow into the alveoli during inspiration, it is known that changes in the pattern of breathing can influence the carbon dioxide concentration in the alveolus and so influence the elimination of the carbon dioxide. For example, increasing the I:E ratio during mechanical ventilation decreases \( (P_{A\text{CO}_2} - P_{E\text{CO}_2}) \) and increases the efficiency of carbon dioxide elimination (Perez-Chada et al., 1983) whereas in exercise, when carbon dioxide production is very high, \( (P_{A\text{CO}_2} - P_{E\text{CO}_2}) \) may become negative (Jones, Robertson and Kane, 1979). High frequency jet ventilation results in a marked decrease in alveolar tidal volume so that the inspiratory dilution of alveolar gas is minimized, and it is possible that this may reduce \( (P_{A\text{CO}_2} - P_{E\text{CO}_2}) \) to values observed during the first breath. Although these time effects are probably of some importance, it is difficult to quantify their magnitude.

Another explanation for the existence of an increased \( (P_{A\text{CO}_2} - P_{E\text{CO}_2}) \) in anaesthesia is that there may be a zone of reduced perfusion in the non-dependent lung zones. Askrog (1966) has correlated the increase in \( (P_{A\text{CO}_2} - P_{E\text{CO}_2}) \) during anaesthesia with a decrease in pulmonary artery pressure. However, the magnitude of zone 1 depends on the relationship between alveolar pressure and pulmonary artery pressure, and it is conceivable that any reduction in mean alveolar pressure resulting from HFJV may decrease the number of poorly perfused alveoli and so may decrease alveolar deadspace. During this study, there were no significant differences in the relationship between mean airway pressure and mean pulmonary artery pressure at 1, 3 and 5 Hz.

The marked increase in \( (P_{A\text{CO}_2} - P_{E\text{CO}_2}) \) which was observed at 3 Hz and 5 Hz, has been reported previously with both open and closed ventilation circuits (Capan et al., 1981; Colgan, Teneyck and Sawa, 1983; Rolly and Versichelen, 1983). Various mechanisms have been suggested for this observation, including mismatching of ventilation and perfusion, mixing of inspired and expired gas in the airways, and inadequate capnograph response time. The results of our second study suggest that the last of these is the most important factor. It is concluded that the normal \( (P_{A\text{CO}_2} - P_{E\text{CO}_2}) \) is re-established by the third normal breath when HFJV at 1, 3 and 5 Hz is interrupted by ventilation at conventional frequencies and tidal volumes. Comparison with other studies suggests that this relationship may be maintained over a wide range of \( P_{A\text{CO}_2} \). Although the end-tidal plateau during the first breath is less reproducible than during the third breath, it appears to provide a close estimate of \( P_{A\text{CO}_2} \). However, it is important to determine whether this relationship is maintained in the diseased lung.

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**REFERENCES**


