Laboratory Investigations

Changes in PETCO$_2$ and pulmonary blood flow after bronchial occlusion in dogs

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The use of PETCO$_2$ in detecting accidental bronchial intubation was investigated. The PETCO$_2$ was measured in six mongrel dogs after occluding the left mainstem bronchus in three conditions; pentobarbital anaesthesia, 0.8% halothane insufflation together with pentobarbital anaesthesia, and simultaneous left pulmonary artery and bronchial airway occlusion with intravenous pentobarbital anaesthesia. An external flow probe measured left pulmonary artery blood flow. The PETCO$_2$ decreased after bronchial occlusion during pentobarbital (35 $\pm$ 3 vs 30 $\pm$ 5 mmHg) and halothane-pentobarbital (30 $\pm$ 6 vs 25 $\pm$ 6 mmHg) conditions (P < 0.05). However, within three minutes of bronchial occlusion, the values of PETCO$_2$ had returned to their pre-occlusion values. After five minutes of bronchial occlusion pulmonary artery blood flow in the non-ventilated lung decreased (P < 0.05) during pentobarbital (770 $\pm$ 533 ml $\cdot$ min^{-1} vs 575 $\pm$ 306 ml $\cdot$ min^{-1}) and halothane-pentobarbital (495 $\pm$ 127 ml $\cdot$ min^{-1} vs 387 $\pm$ 178 ml $\cdot$ min^{-1}) conditions. Simultaneous bronchial and pulmonary artery occlusion prevented any changes in PETCO$_2$. It was concluded that accidental one-lung ventilation results in small and transient decreases in PETCO$_2$. A redistribution of blood flow from the non-ventilated to

Key words

ventilated lung occurs which restores PETCO$_2$ to the original values observed with two-lung ventilation.

L'utilisation de la PETCO$_2$ pour détecter l'intubation bronchique accidentelle a été évaluée. La PETCO$_2$ a été mesurée chez six chiens bâtards après avoir obstrué la bronche souche gauche sous trois conditions : une anesthésie avec pentobarbital, une anesthésie avec pentobarbital et insufflation d'halothane à 0,8%, et une anesthésie avec pentobarbital et obstruction simultanée de l'artère pulmonaire gauche et de la voie aérienne bronchique gauche. Une sonde à débit externe a été utilisée pour mesurer le débit sanguin de l'artère pulmonaire gauche. La PETCO$_2$ diminuait après l'obstruction bronchique lors de l'anesthésie avec pentobarbital (35 $\pm$ 3 vs 35 $\pm$ 5 mmHg) et l'anesthésie avec halothane-pentobarbital (30 $\pm$ 6 vs 25 $\pm$ 6 mmHg) (P < 0,05). Cependant, moins de trois minutes après l'obstruction bronchique, les valeurs de la PETCO$_2$ étaient revenues à leurs valeurs préocclusion. Cinq minutes après l'occlusion bronchique, les débits sanguins de l'artère pulmonaire du poumon non ventilé diminuaient (P < 0,05) durant l'anesthésie au pentobarbital (770 $\pm$ 533 ml $\cdot$ min^{-1} vs 575 $\pm$ 306 ml $\cdot$ min^{-1}) et à l'halothane-pentobarbital (495 $\pm$ 127 ml $\cdot$ min^{-1} vs 387 $\pm$ 178 ml $\cdot$ min^{-1}). L'occlusion simultanée de l'artère pulmonaire et de la bronche prévenait tous les changements de la PETCO$_2$. En conclusion, la ventilation accidentelle d'un seul poumon conduit à une légère diminution transitoire de la PETCO$_2$. Une redistribution du débit sanguin du poumon non ventilé vers le poumon ventilé se produit, ce qui rétablit la PETCO$_2$ à sa valeur originale observée lors de la ventilation à deux poumons.

Accidental misplacement of a tracheal tube into the mainstem bronchus most commonly occurs at the time of intubation but may also take place at any time during anaesthesia.[1] Prompt diagnosis of accidental bronchial intubation and subsequent one-lung ventilation is required

to avoid the potential complications of atelectasis and hypoxaemia. Bronchial intubation may not always be detected by bilateral chest auscultation or increased airway pressure.[1,2] More recently, continuous $PETCO_2$ monitoring has been suggested as a rapid and reliable method in the diagnosis of bronchial intubation.[2] Theoretically, inadvertent bronchial intubation should cause an increase in dead space ventilation[3] and thereby decrease $PETCO_2$ and increase the $PaCO_2$–$PETCO_2$. In the absence of specific experimental data, different authors have conjectured that the expected decrease in $PETCO_2$ may occur suddenly[2] or more gradually.* However, one case report of accidental bronchial intubation described an increase rather than a decrease in $PETCO_2$.[4] There are parallels between accidental bronchial intubation and one-lung ventilation during thoracotomy. Henghan *et al.*[5] found that the $PaCO_2$–$PETCO_2$ was not affected on changing from two-lung to one-lung ventilation.

In order to assess the utility of $PETCO_2$ monitoring in the detection of bronchial intubation, we studied the changes in $PETCO_2$ using an open chested canine model and measured concomitant changes in pulmonary blood flow. It was speculated that changes in $PETCO_2$ associated with bronchial intubation would be transient as the non-ventilated lung gradually became more hypoxic. Hypoxic pulmonary vasoconstriction should reduce blood flow to the non-ventilated lung thereby decreasing the intrapulmonary shunt.[6,7] It was also expected that with increased blood flow to the ventilated lung, calculated dead space ventilation would subsequently return to baseline values.

Methods

Animal preparation

All experiments were approved by the University Animal Care Committee which adheres to the Canadian Council of Animal Care guidelines. Six mongrel dogs (20–25 kg) were anaesthetized with thiopentone (25 mg · kg^{-1}) and pentobarbital (15 mg · kg^{-1}). The trachea was intubated

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ABBREVIATIONS
Arterial carbon dioxide pressure = $PaCO_2$ (mmHg)
End-tidal carbon dioxide pressure = $PETCO_2$ (mmHg)
Arterial to alveolar carbon
       dioxide gradient = $PaCO_2$–$PETCO_2$ (mmHg)
Shunt fraction = $\dot{Q}s/\dot{Q}t$ (%)
Dead space ratio = Vd/Vt

with a 10 mm tracheal tube positioned above the carina. The lungs were ventilated (Harvard ventilator) with 100% oxygen, with a tidal volume (V_T) of 10 ml · kg^{-1} and a frequency adjusted between 15–25 min^{-1} and then fixed at the start of each experiment to maintain the arterial PCO_2 between 30 to 40 mmHg. Anaesthesia was maintained throughout the experiment by a constant infusion of pentobarbital (1 mg · kg^{-1} · hr^{-1}) and muscle relaxation was maintained with pancuronium (0.1 mg · kg^{-1} · hr^{-1}). A catheter, inserted via the femoral vein, was first advanced under continuous pressure monitoring into the right ventricle and then pulled back into the atrium to ensure proper positioning. The right atrial catheter was used to measure right atrial pressure (PRA), inject cold dextrose solution (4° C) for cardiac output (CO) determinations and administer fluids and drugs. A second catheter was inserted via the femoral artery into the ascending aorta and was used to measure systemic arterial pressure (PSA) and for withdrawal of blood for arterial blood gas analysis (pHa, $PaCO_2$, PaO_2). A pulmonary artery thermodilution catheter (Baxter Edwards Model 93-131-7F Irvine, CA USA) was also inserted via the right external jugular vein and positioned in a branch of the pulmonary artery under continuous pressure monitoring. This catheter was used for measurement of mean pulmonary artery pressure (PPA), pulmonary capillary wedge pressure (PWP), and measurement of core body temperature (temp). Cardiac output was measured by thermodilution (Edwards Cardiac Output Computer Model 9520A) after injection of 5 cc of 4° C dextrose (5%) into the right atrial catheter. Blood samples withdrawn through this catheter were analyzed for mixed venous blood gases (pHv, $PvCO_2$, PvO_2). All vascular catheters were connected to pressure transducers (Statham) using low compliance tubing and signals were continuously recorded on an eight channel recorder (Hewlett Packard). Peak inspiratory airway pressures (PINSP) were measured at a port near the tracheal tube which was also connected to an airphase transducer by low compliance tubing.

The pleural cavity was entered through the left sixth intercostal space. Umbilical tape was positioned around the left main pulmonary artery and by tightening the tape the artery could be completely occluded. The pulmonary artery catheter position was verified by palpation of the left pulmonary artery and repositioned if found within the occluded segment. A 25 mm external flow probe (Carolina Instruments) was placed on the main left pulmonary artery to allow for continuous measurement of left lung blood flow. A bronchial blocker (#7 Fogarty Balloon Catheter) was then positioned, under bronchoscopic guidance, in the left main bronchus. Proper positioning of the bronchial blocker was verified by directly observing that the respiratory excursions of the left lung stopped after inflating the

Fogarty catheter balloon. As well, on direct visual inspection, the left lung become gradually atelectatic following balloon inflation. Following the surgical preparation heparin was administered as a 5,000 U intravenous bolus followed by 1,000 U · hr^{-1}. At the conclusion of the experiment all animals were sacrificed by thiopentone overdose.

Calculations

Arterial and mixed venous blood gases were analyzed at 37° C (Corning 162-2) using appropriately calibrated electrodes. The values were corrected for core body temperature[8] and haemoglobin saturations were then calculated using a standard nomogram.[11] Alveolar PO_2 and intrapulmonary shunt (Qs/Qt) were calculated using standard equations.

The CO_2 was sampled from the distal airway through a 1 mm diameter tube. The tube was inserted via the tracheal tube until resistance was met and then pulled back one centimeter. The PETCO$_2$ measurements were made using a side stream infrared capnometer (Puritan-Bennett Datex CO_2 Monitor) with a 150 ml · min^{-1} sampling rate. The capnometer was calibrated before each experiment with 5% CO_2 reference gas. The capnometer measured the percent CO_2 present in the airway and these percentage values were subsequently converted to pressures (mmHg) by multiplication with the same barometric pressure used to calibrate the blood gas analyzer. An instrument correction factor was calculated to account for water vapour differences in the dry reference gas and wet sample gas.[10] Measured values of PETCO$_2$ at atmospheric temperature were also corrected to body temperature which was measured throughout the experiment.[9] The signal from the capnogram was recorded on a single-channel recorder (Puritan-Bennett Datex Recorder). The ascending, plateau and descending limbs of the signal in periods were compared immediately before and immediately after bronchial balloon inflation and deflation. Alveolar dead space ventilation (Vd/Vt) was calculated from the Enghoff modification of the Bohr equation where Vd/Vt = (PaCO$_2$–PETCO$_2$/PaCO$_2$.[10] The PETCO$_2$ was utilized rather than mixed expired CO_2 in order to approximate better the alveolar dead space alone rather than the combined alveolar and anatomical dead space.[11]

Experimental protocol

Following the surgical preparation the animals were placed in the supine position and were allowed to stabilize until values of PaCO$_2$ and PETCO$_2$ varied by less than 5% over a ten-minute period. The following haemodynamic variables (PSA, PRA, PWP, PPA, CO) and gas exchange variables (Qs/Qt, pHa, PaO$_2$, PaCO$_2$, pHv̄, Pv̄O$_2$, Pv̄CO$_2$). Haematocrit (Hct), Pinsp, and temperature was also measured. All these values constituted observations at
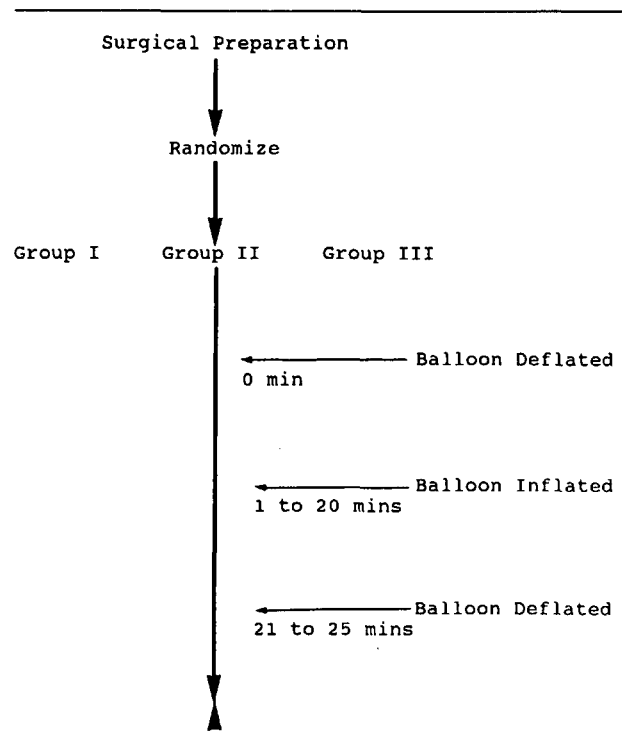

FIGURE 1 Group I refers to intravenous pentobarbital anaesthesia, Group II refers to 0.8% halothane insufflation and intravenous pentobarbital and Group III refers to simultaneous bronchial and pulmonary artery occlusion during pentobarbital anaesthesia. Balloon was placed within the left mainstem bronchus. *Denotes significant difference ($P < 0.05$) comparing baseline (period 0) and 1 minute (Period 1).

time 0. As well, PETCO$_2$ was continuously sampled and recorded for later analysis and calculation of PaCO$_2$–PETCO$_2$. Following all measurements, the bronchial blocker in the left main bronchus was rapidly inflated with 5 ml of air. All measurements were then repeated at one, three, and five minutes after occlusion (time 1, 3, and 5 measurements respectively). Twenty minutes after bronchial occlusion measurements were repeated (time 20). The bronchial blocker was then rapidly deflated. The measurements were again repeated at 21, 23, and 25 min following bronchial occlusion (time 21, 23, and 25 measurements respectively).

All animals were studied under three experimental conditions, the order of which was randomized (Figure 1). At the beginning of each experimental condition, the entire lung received one standardized inflation with a volume of 20 ml · kg^{-1}. Also a positive end expiratory pressure of 10 cm H_2O was applied for 30 sec to ensure similar alveolar recruitment as verified by visual inspection. The animals were studied under pentobarbital anaesthesia as outlined above. The animals were also studied during a condition of bronchial airway occlusion with concomitant left main pulmonary artery occlusion. During these studies flow to

TABLE I Haemodynamic and blood gas values during pentobarbital anaesthesia

	0	1	3	5	20	21	23	25
PSA	183 ± 23	172 ± 19	172 ± 19	172 ± 19	172 ± 19	180 ± 17	180 ± 17	180 ± 17
PINSP*	8 ± 2	14 ± 3†	14 ± 3†	14 ± 3†	14 ± 3	13 ± 3	12† ± 3	12† ± 2
PaO₂*	338 ± 90	248 ± 78†	194 ± 65†	192 ± 81†	202 ± 77	217 ± 89	214 ± 79	199 ± 75
Pv̄O₂*	53 ± 10	51 ± 6	48 ± 7†	47 ± 8†	48 ± 7	47 ± 7	48 ± 8	49 ± 7
PPA*	24 ± 3	29 ± 4†	30 ± 4†	30 ± 4†	30 ± 4	28 ± 4	27† ± 4	27† ± 4
CO	3.29 ± 0.43	3.48 ± 0.56	3.48 ± 0.48	3.58 ± 0.23	3.81 ± 0.90	3.57 ± 0.72	3.42 ± 0.58	3.63 ± 0.74
Flow*	770 ± 533	828 ± 569†	637 ± 375	575 ± 306†	445 ± 269	442 ± 264	462 ± 236	468 ± 227
Q̇s/Q̇t	19 ± 9	24 ± 7	28 ± 6†	30 ± 7†	27 ± 7	25 ± 6	26 ± 7	28 ± 6
Vd/Vt*	0.03 ± 0.09	0.22 ± 0.16†	0.08 ± 0.16	0.06 ± 0.13	0.08 ± 0.12	0.09 ± 0.12	0.08 ± 0.13	0.08 ± 0.15

PSA = mean blood pressure (cm H_2O), PINSP = peak inspired airway pressure (cm H_2O), PaO_2 = arterial partial pressure of oxygen (mmHg), PvO_2 = venous partial pressure of oxygen (mmHg), CO = cardiac output (L · min⁻¹), PPA = pulmonary artery mean pressure (cm H_2O), Flow = left pulmonary artery flow (ml · min⁻¹), Qs/Qt = shunt fraction, Vd/Vt = ventilatory dead space. 0, 1, 3, 5, 20, 21, 23, 25 refer to times in minutes after initial time 0 measurements. Bronchial airway balloon was deflated at times 0, 21, 23, and 25 and inflated at times 1, 3, 5, and 20.
*Signifies $P < 0.05$ for each group by ANOVA.
†Signifies $P < 0.05$ for each group comparing time 0 with times 1, 3, or 5 and comparing time 20 with times 21, 23 or 25.

the left main pulmonary artery was simultaneously stopped and restarted when ventilation to the left main bronchus was stopped and restarted. The final experimental condition was identical to the initial conditions except halothane was insufflated throughout, including the stabilization period before recording of any data. No attempt was made to standardize haemodynamic variables between conditions. We used an Ohio vaporizer to generate the concentration of halothane added to the inspired gas. The concentration of halothane in the inspired gas was measured using a SARA mass spectrometer. Similarly, expired halothane concentrations were measured during the subsequent conditions to ensure that there was no residual halothane present. During both the halothane and bronchial occlusion periods pentobarbital was constantly infused.

Statistics

All values of blood gases, haemodynamic and ventilatory variables were compared using a one-way analysis of variance (ANOVA). Where the F statistic showed a significant difference, paired t tests were used to determine which periods were different. Sidak's multiplicative inequality was used to correct for the number of comparisons made between periods.[12] This correction causes the requisite t value to increase as the number of comparisons increases. Therefore the number of comparisons was prospectively limited in these experiments. Initial measurements before bronchial occlusion (time 0) were compared with all subsequent measurements. Similarly, measurements at the termination of bronchial occlusion (time 20) were compared to all subsequent measurements. Statistical comparisons were only made between variables within the same experimental condition.

Results

Values of temperature (range 36.9 ± 0.7° C to 37.6 ±

0.7° C), haematocrit (range 31 ± 2% to 33 ± 2%), pHa (range 7.34 ± 0.03 to 7.40 ± 0.04), PaCO₂ (range 32 ± 5 mmHg to 39 ± 3 mmHg), Pv̄CO₂ (range 33 ± 5 mmHg to 36 ± 6 mmHg), PRA (range 9 ± 2 cm H_2O to 10 ± 2 cm H_2O), or PWP (range 12 ± 2 cm H_2O to 13 ± 2 cm H_2O) did not change over time during any of the experimental conditions. Tables I, II, and III show selected values of haemodynamic and gas exchange variables over time during the three experimental conditions (pentobarbital, halothane-pentobarbital and pulmonary artery occlusion-pentobarbital respectively). During halothane-pentobarbital, inspired halothane concentrations were stable with values of 0.8 ± 0.2%, 0.8 ± 0.3% and 0.8 ± 0.3% during time 0, 1 and 21 respectively. Halothane could not be detected in expired gas during the other experimental conditions. Although CO was not identical between different conditions, it remained constant within any one condition.

The PINSP increased following bronchial balloon inflation ($P < 0.05$) and decreased following bronchial balloon deflation ($P < 0.05$) under all three conditions. Values of PPA followed similar patterns to values of PINSP. Values of PSA, however, tended to decrease following balloon inflation and increase with balloon deflation reaching significance under conditions of halothane-pentobarbital. Values of PaO₂ decreased following bronchial occlusion ($P < 0.05$) except during simultaneous pulmonary artery occlusion. After bronchial balloon deflation values of PaO₂ did not change.

The PETCO₂ decreased immediately with balloon inflation from values of 35 ± 3 mmHg to 30 ± 5 mmHg and from 30 ± 6 mmHg to 25 ± 6 mmHg under pentobarbital and halothane-pentobarbital conditions respectively. Similarly, with balloon inflation PaCO₂–PETCO₂ increased immediately from 1.2 ± 3.4 mmHg to 8.9 ± 6.2 mmHg and from 2.0 ± 2.6 mmHg to 9.1 ± 4.2 mmHg

TABLE II   Haemodynamic and blood gas values during halothane-pentobarbital anaesthesia

	0	1	3	5	20	21	23	25
PSA*	138 ± 26	127 ± 22†	125 ± 22†	125 ± 22†	172 ± 22	180 ± 31	180 ± 31	180 ± 31
PINSP*	8 ± 2	12 ± 3†	12 ± 3†	12 ± 3†	12 ± 3	12 ± 3	12 ± 3	11 ± 3
PaO₂*	341 ± 74	241 ± 127	185 ± 72†	198 ± 84†	203 ± 88	206 ± 76	209 ± 82	201 ± 73
PvO₂	45 ± 8	41 ± 6	42 ± 8	43 ± 9	41 ± 9	42 ± 10	41 ± 10	40 ± 8
PPA*	23 ± 2	28 ± 3†	28 ± 3†	28 ± 3†	28 ± 3	26 ± 3	26 ± 3	25 ± 3†
CO	2.21 ± 0.54	2.31 ± 0.83	2.57 ± 0.91	2.24 ± 0.73	2.02 ± 0.66	2.02 ± 0.51	2.09 ± 0.73	2.10 ± 0.69
Flow*	495 ± 127	507 ± 162	480 ± 213	383 ± 178†	365 ± 156	358 ± 176	362 ± 180	382 ± 193
Qs/Qt*	17 ± 5	24 ± 9	25 ± 4†	27 ± 9†	26 ± 7	27 ± 6	25 ± 7	24 ± 6
Vd/Vt*	0.06 ± 0.08	0.27 ± 0.12†	0.09 ± 0.06	0.06 ± 0.08	0.08 ± 0.04	0.07 ± 0.12	0.11 ± 0.05	0.11 ± 0.09

PSA = mean blood pressure (cm H_2O), PINSP = peak inspired airway pressure (cm H_2O), PaO₂ = arterial partial pressure of oxygen (mmHg), PvO₂ = venous partial pressure of oxygen (mmHg), CO = cardiac output (L · min⁻¹), PPA = pulmonary artery mean pressure (cm H_2O), Flow = left pulmonary artery flow (ml · min⁻¹), Qs/Qt = shunt fraction, Vd/Vt = ventilatory dead space. 0, 1, 3, 5, 20, 21, 23, 25 refer to times in minutes after initial time 0 measurements. Bronchial airway balloon was deflated at times 0, 21, 23, and 25 and inflated at times 1, 3, 5, and 20.
*Signifies $P < 0.05$ for each group by ANOVA.
†Signifies $P < 0.05$ for each group comparing time 0 with times 1, 3, or 5 and comparing time 20 with times 21, 23 or 25.

TABLE III   Haemodynamic and blood gas values with bronchial airway and pulmonary artery occlusion during pentobarbital anaesthesia

	0	1	3	5	20	21	23	25
PSA	183 ± 8	170 ± 15	167 ± 15†	167 ± 15†	167 ± 15	173 ± 18	173 ± 18	177 ± 10†
PINSP*	9 ± 3	13 ± 3	14 ± 3†	14 ± 2†	14 ± 2	12 ± 1	11 ± 2†	11 ± 2†
PaO₂	382 ± 79	301 ± 123	353 ± 110	338 ± 103	359 ± 102	315 ± 132	287 ± 131	293 ± 115
PvO₂	52 ± 7	53 ± 9	50 ± 7	51 ± 9	49 ± 12	52 ± 12	49 ± 11	49 ± 10
PPA*	26 ± 3	31 ± 2†	31 ± 2†	31 ± 2†	31 ± 2	29 ± 4	29 ± 4	28 ± 4
CO	3.49 ± 0.73	3.45 ± 0.45	3.44 ± 0.52	3.28 ± 0.66	3.14 ± 0.62	3.26 ± 0.57	3.22 ± 0.48	3.34 ± 0.46
Flow	607 ± 238	—	—	—	—	545 ± 192	435 ± 180	428 ± 188
Qs/Qt*	17 ± 9	24 ± 13†	17 ± 7	18 ± 9	16 ± 7	21 ± 13	21 ± 11	21 ± 10
Vd/Vt	0.02 ± 0.05	0.03 ± 0.12	0.02 ± 0.1	0.04 ± 0.08	0.07 ± 0.05	0.07 ± 0.09	0.03 ± 0.05	0.06 ± 0.03

PSA = mean blood pressure (cm H_2O), PINSP = peak inspired airway pressure (cm H_2O), PaO₂ = arterial partial pressure of oxygen (mmHg), PvO₂ = venous partial pressure of oxygen (mmHg), CO = cardiac output (L · min⁻¹), PPA = pulmonary artery mean pressure (cm H_2O), Flow = left pulmonary artery flow (ml · min⁻¹), Qs/Qt = shunt fraction, Vd/Vt = ventilatory dead space. 0, 1, 3, 5, 20, 21, 23, 25 refer to times in minutes after initial time 0 measurements. Bronchial airway balloon was deflated at times 0, 21, 23, and 25 and inflated at times 1, 3, 5, and 20.
*Signifies $P < 0.05$ for each group by ANOVA.
†Signifies $P < 0.05$ for each group comparing time 0 with times 1, 3, or 5 and comparing time 20 with times 21, 23 or 25.

during pentobarbital and halothane-pentobarbital conditions respectively (Figures 2 and 3). As well, values of Vd/Vt increased with bronchial occlusion. The changes in PETCO₂, PaCO₂–PETCO₂ and Vd/Vt were transient and returned to time 0 values within three minutes of balloon inflation. Left lung blood flow showed an early increase within one minute after balloon inflation followed by a sustained decrease. Under conditions of simultaneous pulmonary artery and bronchial balloon inflation, no changes were seen in Vd/Vt, PETCO₂ or PaCO₂–PETCO₂ (Figure 4). With deflation of the bronchial balloon, values of PETCO₂ or PaCO₂–PETCO₂ did not change with deflation of the bronchial balloon during any of the three experimental conditions.

Discussion

These experiments were designed to assess the utility of PETCO₂ in detecting a misplacement of the tracheal tube

into the bronchus. We had previously noted that in a closed chest model, the decrease in PETCO₂ with bronchial intubation was transient and unlikely to be clinically useful.[13] These experiments in an open chest model also demonstrate that PETCO₂ decreases transiently with the onset of single-lung ventilation. The PETCO₂ returns to pre-occlusion values as blood flow is diverted away from the non-ventilated lung.

Model

These studies were performed in animals with their pleural spaces open to atmosphere. In this way the left main bronchus was selectively occluded. The complete absence of respiration in the left lung following bronchial balloon inflation was observed. As well, under conditions when the pulmonary arterial flow was not concurrently occluded, progressive reabsorption of oxygen occurred, with the left lung becoming atelectatic in a patchy distribution and then
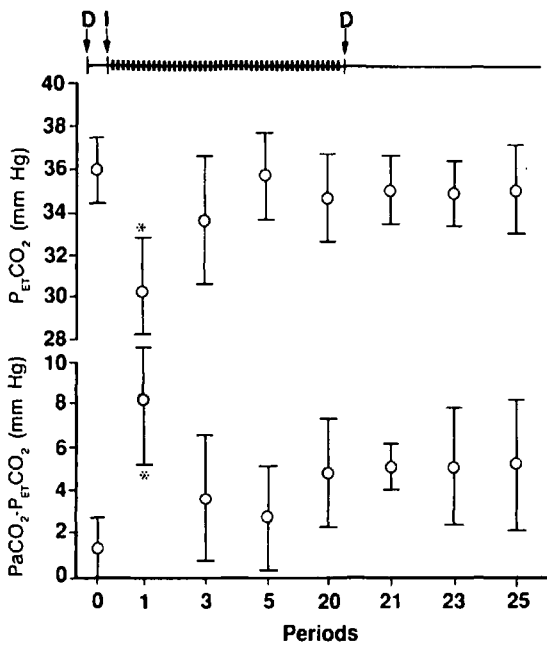
FIGURE 2 Changes in $P_{ET}CO_2$ and $PaCO_2-P_{ET}CO_2$ during pentobarbital anaesthesia. Onset of bronchial balloon inflation (I) with reference to time 0 at which baseline measurements were made with bronchial balloon deflated (D). Shaded area represents total period of bronchial balloon inflation. Note that measurements at time 20 were made just prior to bronchial balloon deflation. $P < 0.05$ comparing time 0 with times 1, 3, and 5 or time 20 with times 21, 23 and 25.
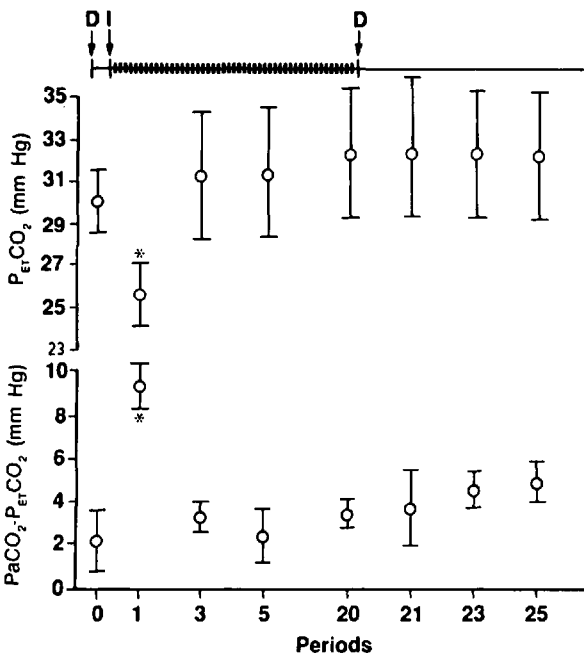

FIGURE 3 Changes in $P_{ET}CO_2$ and $PaCO_2-P_{ET}CO_2$ during halothane-pentobarbital anaesthesia. (See legend Figure 1.)
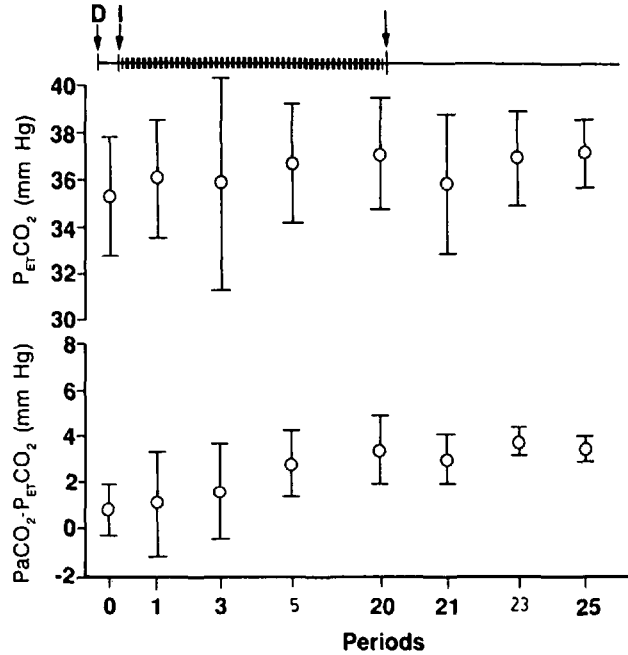

FIGURE 4 Changes in $P_{ET}CO_2$ and $PaCO_2-P_{ET}CO_2$ during pentobarbital anaesthesia with simultaneous pulmonary artery and bronchial occlusion.

finally becoming completely atelectatic. When the left pulmonary artery flow was obstructed, the left lung appeared to remain partially inflated but, more importantly, respiratory excursions in the left lung were also absent following the bronchial airway balloon inflation. These events following bronchial airway balloon inflation were temporally related to decreases in arterial PO_2 during pentobarbital and halothane-pentobarbital conditions. Deflation of the bronchial balloon did not reverse the atelectasis nor improve the depressed values of PaO_2.[14] Corresponding to the visual changes noted in the lobe, peak inspiratory airway pressure immediately increased with bronchial airway balloon inflation which is compatible with diversion of the tidal volume to the right lung. On balloon deflation airway pressure tended to decrease but since the atelectasis was not reversed, the airway pressure did not return to pre-occlusion values.

The changes in $P_{ET}CO_2$ which we found were not a reflection of changes in arterial PCO_2 which remained stable over the duration of the experiment within each condition. Cardiac output within each condition also remained stable so that CO_2 delivery to the lung should have been constant. Finally, values of pH were stable over the duration of the experiments. Therefore, we are confident that the changes in $P_{ET}CO_2$ and $PaCO_2-P_{ET}CO_2$ which we observed were related to the effects of bronchial occlusion and were not confounded by other independent variables.

We noted that cardiac output decreased in the halothane-pentobarbital condition compared with the other two conditions. This lower cardiac output is unlikely to have altered either $PETCO_2$ or $PaCO_2-PETCO_2$ without simultaneous changes in $PaCO_2$. That is, elimination of $PaCO_2$ was dependent upon minute ventilation (fixed at the start of the experiment) and efficiency of carbon dioxide exchange through the lung. As both Vd/Vt and $\dot{Q}s/\dot{Q}t$ were similar in the halothane-pentobarbital and pentobarbital conditions during baseline periods carbon dioxide exchange should be similar.

We found that the baseline left pulmonary artery flow was lower than expected and accounted for approximately 20% of the measured cardiac output rather than the 40–45% expected.[15] It is possible that our surgical manipulations produced subclinical atelectasis in the left lung and this caused a reduction in baseline flow. However, the left lung was inflated after each period of bronchial occlusion until no atelectatic areas were apparent on gross inspection. Our calculated shunt fraction breathing oxygen at the initiation of each condition was always less than 20% indicating that ventilation and perfusion were reasonably matched. Rather this apparent decreased left pulmonary artery flow might represent a difficulty in comparing two different methods of blood flow determination (i.e., electromagnetic flow probe and thermodilution). The lower than expected flow does not influence our conclusions, however, since we were interested in relative rather than absolute changes in blood flow. If anything, if flow to the left lung were increased then the time to complete reabsorption of oxygen would be reduced. This in turn would result in more rapid diversion of flow away from the occluded lung with a resultant more rapid correction of the $\dot{V}/\dot{Q}$ mismatching.

Hypoxic pulmonary vasoconstriction

The immediate increase in pulmonary artery pressures with balloon inflation was unlikely to be related to hypoxic pulmonary vasoconstriction since the alveoli were still filled initially with 100% oxygen. Similarly the immediate decrease in pulmonary artery pressure with balloon deflation was unlikely to be related to reduction in hypoxic pulmonary vasoconstriction as the lobe failed to re-expand. In fact there was an initial transient increase in flow to the left lung with balloon inflation and this is not consistent with the action of hypoxic pulmonary vasoconstriction. We speculate that changes in the transmitted airway pressure could account for our observed immediate changes in pulmonary artery pressure. With increased airway pressure in the right lung following inflation of the bronchial balloon, there would be increased compression of the alveolar vessels causing diversion of flow away from the right lung. Concurrently, there would be a

reduction in alveolar pressure in the left lung thereby reducing alveolar vessel compression and enhancing flow. As the left lung became atelectatic, there would be recruitment of hypoxic pulmonary vasoconstriction with diversion of flow away from the left lung to the well ventilated right lung. We believe that this sequence of events best explains our observations of flow distribution to the lungs. Following deflation of the bronchial balloon the opposite sequence would occur with an initial decrease in flow to the left lung due to transmitted airway pressure increasing followed by an increased flow if hypoxic pulmonary vasoconstriction were relieved. However, following bronchial balloon deflation, the atelectatic lung failed to re-expand and therefore the left pulmonary artery blood flow and PaO_2 did not return to baseline values.[14] Finally, our primary anaesthetic agent, pentobarbital, was infused in similar amounts through the experiment and has not been found to independently alter hypoxic pulmonary vasoconstriction.[16]

Expired carbon dioxide

We noted that $PETCO_2$ decreased and $PaCO_2-PETCO_2$ increased with the onset of single lung ventilation. These changes were only transient; i.e., they could be detected within one minute of bronchial occlusion and were reversed within three minutes. The decrease in $PETCO_2$ is likely secondary to an increase in alveolar dead space ventilation[17] (high $\dot{V}/\dot{Q}$ units) produced when the total tidal volume is diverted to the single lung. By utilizing the values for $PETCO_2$ rather than mixed expiratory CO_2 in the calculation of Vd/Vt in the Bohr equation we were able to quantify this increase in alveolar dead space.[11] We speculate that as vascular reflexes are recruited, pulmonary blood flow is diverted away from the non-ventilated lung. This improves $\dot{V}/\dot{Q}$ mismatch and the alveolar dead space ventilation decreases thus returning $PETCO_2$ toward pre-occlusion values. In the presence of simultaneous bronchial airway occlusion and pulmonary artery occlusion, $\dot{V}/\dot{Q}$ matching is maintained and there is not a time delay until hypoxic pulmonary vasoconstriction diverts blood flow away from the non-ventilated lung. This was shown by the shunt fraction which did not increase under conditions of simultaneous bronchial and pulmonary artery occlusion but did increase under pentobarbital and halothane-pentobarbital conditions. We did not see any changes in $PETCO_2$ under conditions of concurrent bronchial and pulmonary artery occlusion.

With the onset of single-lung ventilation, the increase in alveolar dead space ventilation also caused a transient increase in $PaCO_2-PETCO_2$.[17] The $PaCO_2-PETCO_2$ increased primarily because the $PETCO_2$ decreased and secondarily to a small increase in the $PaCO_2$. The $PaCO_2$ tended to be less effected by single-lung ventilation than

was the $PETCO_2$ since $\dot{V}/\dot{Q}$ mismatch tends to have only a small effect on CO_2 elimination.[6]

We did not find any differences in the capnograph waveform which were superimposable during all of the experimental conditions. This waveform was similar to the expected results for expired CO_2 during expiration.[8] We were thus confident that our measurements of $PETCO_2$ represented a reasonable sampling of alveolar CO_2 during expiration.

In conclusion, we found that changes in $PETCO_2$ were associated with diversion of pulmonary blood flow away from the non-ventilated lung. This occurred whether the flow diversion occurred due to hypoxic pulmonary vasoconstriction or whether the flow diversion occurred due to mechanical obstruction of the left pulmonary artery. Although the change in $PETCO_2$ is statistically significant, it is of a relatively small magnitude and, more importantly, transient. It could be easily overlooked in a clinical setting after tracheal intubation and dual lung ventilation had been established. These experiments may actually overestimate the time until the effect on $PETCO_2$ is reversed. Left lung blood flow was relatively low which would tend to maintain aerated alveoli for a longer period. As well, 100% oxygen was used for ventilation. In the more common clinical setting of nitrous oxide and oxygen mixtures, the alveolar O_2 would decrease to low levels even more rapidly and allow hypoxic pulmonary vasoconstriction to divert flow over even a shorter time period. If, in addition, the $PETCO_2$ samples are intermittently measured by a central mass spectrometer servicing several operating rooms, the changes might be missed. Changes in PPA and PINSP were more prolonged than the changes in $PETCO_2$. It is possible that these haemodynamic and airway pressure changes which have been traditionally emphasized are still more clinically useful in detecting inadvertent bronchial intubation.

Acknowledgements

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