Positive End-Expiratory Pressure Has Little Effect on Carbon Dioxide Elimination After Cardiac Surgery

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We investigated the effects of positive end-expiratory pressure (PEEP) on carbon dioxide (CO_2) elimination in a cross-over study of 14 patients whose lungs were ventilated after cardiac surgery. They initially received either 7.5 cm H_2O PEEP or zero end-expiratory pressure and were then changed over to the other mode. We measured CO_2 minute elimination ($\dot{V}CO_2$) and "efficiency," a quantification of the shape of CO_2 single-breath test (SBT-CO_2), the plot of expired CO_2 against expired volume. $\dot{V}CO_2$ and efficiency (and therefore the shape of SBT-CO_2) were not significantly affected by PEEP. These results agree with findings in patients with acute lung injury, but are in contrast with those in an open-chest dog model, in which 7.5 cm H_2O PEEP caused a 19% decrease in $\dot{V}CO_2$ and significant changes in SBT-CO_2. **Implications:** During artificial ventilation, applying a positive pressure in expiration expands the lung and improves the uptake of oxygen, but there is a theoretical risk of reduced carbon dioxide elimination. We applied positive end-expiratory pressure to patients immediately after heart surgery and found that it has no effect on carbon dioxide elimination.

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Positive end expiratory pressure (PEEP) improves oxygenation in many situations: it recruits previously unventilated alveoli, increases end-expiratory lung volume, and reduces right-to-left intrapulmonary shunting. However, by increasing mean alveolar pressure, it may have negative effects on carbon dioxide (CO_2) elimination. In patients with acute lung injury and acute respiratory distress, 15 cm H_2O PEEP had little effect on CO_2 elimination (1). In contrast, in anesthetized, ventilated, open-chest dogs, Breen et al. (2) found that 7.5 cm H_2O PEEP reduced minute CO_2 elimination ($\dot{V}CO_2$) by 19%. The airway dead space increased by 24% and the physiological dead space by 12%.

Further, in dogs (2), but not in humans (1), there was a marked change in the shape of the plot of expired PCO_2 against expired volume, the CO_2 single-breath test (SBT-CO_2). The slope of Phase III of SBT-CO_2 (3), the "alveolar plateau," was increased, implying an increased spread of ventilation/perfusion (V/Q) ratios. Also, the transition between Phase II and Phase III became less distinct. [Phase II is the upstroke in expired PCO_2 between Phases I and III, representing the mixing of airway and alveolar gas, and the progressive recruitment of transit times (4).] A sloping Phase III implies a difference between $PETCO_2$ and mean alveolar PCO_2. The slope is positively correlated to the arterial-end tidal PCO_2 difference and the alveolar dead space fraction (5).

One of us (RF) had recorded SBT-CO_2 in many patients with PEEP, but had never witnessed the quantitative and qualitative changes in SBT-CO_2 demonstrated by Breen et al. (2) in dogs. We therefore performed a cross-over trial to investigate the effects of PEEP in humans. We made our measurements immediately after cardiac surgery—a situation in which PEEP is beneficial.

Methods

After local ethics committee approval, 15 patients (11 male) undergoing cardiac surgery gave informed consent to the study. None were current smokers. Their median age was 64 yr (range 50–76), height 170 cm (154–182), and weight 76 kg (62–100). They were premedicated with lorazepam 2–3.5 mg. Anesthesia was induced with fentanyl 2–3 mg. Pancuronium 8–11 mg was given to facilitate intubation, and their lungs were ventilated with 0.5%–1.0% enflurane in an air:oxygen mixture (FIO_2) 0.5. Cardiopulmonary bypass was conducted with a 2l crystalloid prime. Donor blood was

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given as necessary to achieve a postbypass hemoglobin of 8.5–9.5 g/dL.

After the chest drains had been inserted and the sternum wired, the patients' lungs were ventilated with a Servo 900C ventilator (Siemens-Elema, Solna, Sweden) set to volume control. The rate was fixed at 15 breaths/min, inspiratory:expiratory ratio 1:2, with no end-expiratory pause. The enflurane in air/oxygen continued throughout (Fio$_2$ 0.5). Minute volume was adjusted to produce a Petco$_2$ of approximately 4.0 kPa. The breathing circuit and the endotracheal tube cuff had been previously tested for leaks. Patients were sequentially assigned to initially receive either 7.5 cm H_2O PEEP or zero positive end-expiratory pressure (ZEEP).

Expired Pco$_2$ was measured by a Siemens-Elema 930 capnograph (Solna, Sweden) (6), an in-line device, which obtains power, expiratory flow, and timing signals from the Servo 900C ventilator. The flow signal from the ventilator is used to calculate expired CO_2 volume. The 930 can be connected to a computer to obtain SBT-CO_2 values (7,8). In addition to displaying Petco$_2$ and $\dot{V}$co$_2$, it shows the volume of Phase I of SBT-CO_2 (the non-CO_2 containing part of the tidal volume) and the volume of the combined Phases II and III, i.e., the CO_2-containing part of the tidal volume. The particular device we used was fitted with special components that give a quicker response time than the commercially available versions (9). The delay in the CO_2 signal is 12 ms, while that of the expiratory flow signal is 4 ms (9). In practice, these delays are of little significance, and compared with laboratory analysis of expired gas CO_2, there is no systematic error at adult tidal volumes (9–11). The CO_2 signal is linear in air/oxygen mixtures, as is the expired volume signal. The device does not compensate for compressed volume in the tubings. About 62%–68% of the compressed gas leaves the system before CO_2 reaches the transducer (10), and therefore, with knowledge of the tubing compliance and end-expiratory airway pressure, it is possible to estimate the effect of the remaining 33%–38% of compressed gas on the calculation of $\dot{V}$co$_2$.

The shape of SBT-CO_2 was quantified as "efficiency" (12), i.e., the volume of CO_2 in the breath, divided by the theoretical maximal volume in a breath of square wave form, expressed as a percentage. This square wave form is defined by Phase II plus Phase III volume (i.e., the CO_2 containing part of the tidal volume) and Petco$_2$ (Fig. 1). Results were analyzed by using Student's paired *t*-test; *P* values of < 0.05 were regarded as significant.

After sternal wiring was complete and when Petco$_2$ and $\dot{V}$co$_2$ were constant, these variables were noted from the capnograph display over 5–10 breaths. Also noted were Phase I volume and the combined volume of Phases II and III. The ventilator was then switched to the other mode, and new readings were taken when Petco$_2$ and $\dot{V}$co$_2$ had once again stabilized, after 2–5 min.

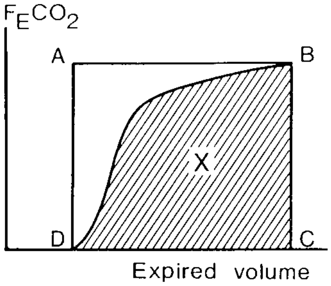

Figure 1. Efficiency is defined by the area under the curve, area X, divided by the theoretical maximal area under the curve, area ABCDA, expressed as a percentage. Efficiency in anesthetized, ventilated patients normally varies between 72% and 88%. Lower values are found in emphysema and asthma. Reproduced from Fletcher et al. (6) with permission from the *British Journal of Anesthesia*.

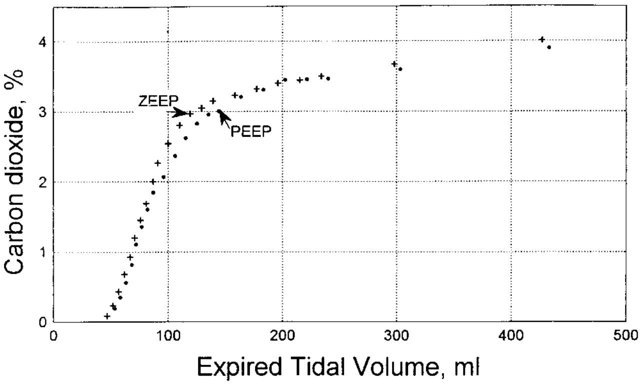

Figure 2. SBT-CO_2 tracings obtained from a patient after cardiac surgery during zero positive end-expiratory pressure (ZEEP) and 7.5 cm H_2O positive end-expiratory pressure (PEEP) ventilation. The data are compensated for compressed volume. The main difference between the two tracings is an increase in airway dead space of approximately 10 mL and a reduction in tidal CO_2 elimination of approximately 4% effected by PEEP. There is no obvious difference in Phase III slope or Phase I volume.

Results

Desaturation during ZEEP ventilation resulted in our abandoning measurement in one patient. In the remaining 14 patients, applying 7.5 cm H_2O PEEP decreased mean tidal volume by 4 mL. Mean $\dot{V}$co$_2$ decreased by 4 mL CO_2/min, i.e., 2% (not significant), with no change in Petco$_2$. Phase I volume, which is approximately half of the airway dead space (10), increased by 5 mL. The compliance of the tubings was 1.5 mL/cm H_2O, and therefore, it can be estimated that, at the airway pressures obtained, most of the 5 mL increase in Phase I volume during PEEP ventilation would be caused by compressed volume. There was no change in efficiency, and therefore, the shape of SBT-CO_2 cannot have changed significantly. Thus,

Table 1. Mean (SD) in the Two Modes, and Confidence Intervals (C.I.) for the Difference of the Means ($n = 14$)

	ZEEP	PEEP	95% CI of difference	P value
Phase I volume (mL)	86 (7)	92 (12)	1.2, 9.5	0.015
Phase II + III volume (mL)	384 (94)	375 (89)	−3, −15	0.007
Tidal volume (mL)	471 (97)	467 (97)	−0.3, −7.7	0.04
$\dot{V}CO_2$ (mL/min)	180 (50)	176 (46)	−8.0, 0.4	0.07
P_{ETCO_2} (kPa)	4.05 (0.26)	4.02 (0.21)	−0.13, 0.06	0.4
Efficiency (%)	76.6 (3.8)	77.6 (2.8)	−0.4, 2.3	0.14

ZEEP = zero end-expiratory pressure, PEEP = positive end-expiratory pressure, CI = confidence interval, $\dot{V}CO_2$ = carbon dioxide minute ventilation.

there can have been no important change in the volume of Phase II or the slope of Phase III. There was no difference in findings between those who began with PEEP and those who began with ZEEP.

Figure 2 was obtained from an on-line computer program, which became available after data from the 14 patients had been collected. It shows SBT-CO$_2$ tracings from a patient undergoing PEEP and ZEEP ventilation after cardiac surgery. The computer program takes signals from the ventilator and capnograph and compensates for compressed volume (7,8). The figure shows that Phase I volumes and Phase III slopes are similar, but there is an increase of approximately 10 mL in airway dead space with PEEP. The tidal volumes of CO$_2$ were 11.5 mL (ZEEP) and 11.0 mL (PEEP).

Discussion

In a setting in which it is clinically beneficial, we have shown that PEEP causes little qualitative or quantitative change in CO$_2$ elimination. A similar lack of effect of PEEP on the shape of SBT-CO$_2$ was reported in patients with acute lung injury and acute respiratory distress (1). These results contrast sharply with those obtained in open-chest dogs (2); PEEP reduced $\dot{V}CO_2$ by 19%, and produced a considerable change in the shape of SBT-CO$_2$, implying a greater spread of V/Q ratios.

PEEP reopens collapsed alveoli, increasing CO$_2$ clearance from such areas. It also dilates the conducting airways and may cause over-ventilation of some alveoli. The increased alveolar pressure may also distribute the pulmonary blood flow away from such areas, which should reduce CO$_2$ clearance. Presumably, in our cardiac surgery patients, any negative effects of PEEP on already ventilated alveoli were largely compensated for by the increase in CO$_2$ elimination from previously low and zero V/Q compartments.

The data in Table 1 were not compensated for airway pressure. With our system, 62%–68% of compressed gas volume passes through the ventilator flowmeter before any CO$_2$ arrives at the CO$_2$ transducer (10). We argued above that most of the 5-mL increase in Phase I volume during PEEP ventilation would be caused by compressed volume. The SBT-CO$_2$ shown in Figure 2, in which the data are compensated for airway pressure, supports this; there is little difference in Phase I volume between the two modes. Less than one quarter of the compressed volume passes through the flowmeter during Phase III (10); thus, the error in the measurement of $\dot{V}CO_2$ caused by PEEP is likely to be negligible. The small reduction in tidal volume with PEEP appears to be a feature of the Servo ventilator, as it occurs when ventilating a test lung.

We did not sample blood gases and, therefore, could not measure alveolar dead space or the arterial-end tidal P_{CO_2} difference. However, by monitoring $\dot{V}CO_2$ and P_{ETCO_2} continuously, we were able to demonstrate steady state at both measurements. Indeed, a striking finding of the measurement protocol was the lack of change in $\dot{V}CO_2$ on applying and removing PEEP; therefore, only a short time (two to five minutes) was needed to re-establish steady state after changing mode.

Although our protocol did not include plotting expired CO$_2$ against expired volume, we can be reasonably sure that PEEP did not cause any significant change in SBT-CO$_2$. Efficiency did not change, and therefore, Phase III slope could not have changed significantly, as Figure 2 confirms. [Efficiency in anesthetized patients varies between 72% and 88% (5), the greater values being associated with large tidal volumes in healthy patients. Values below 70% are seen in obstructive airway disease.] In dogs (2), PEEP affected the relationship between P_{ETCO_2} and mean alveolar P_{CO_2} by increasing Phase III slope. Our data suggest that, in cardiac surgery patients, the relationship is not significantly changed. Others have failed to show a change in Phase III slope when PEEP was applied to patients with lung injury (1).

The relationship between Phase III slope and commonly used indices of gas exchange have been explored in detail (5). The slope is caused by sequential emptying of lung regions with different V/Q ratios. The first alveoli to empty have the highest V/Q ratios and, therefore, the lowest P_{CO_2}; those emptying last have the lowest V/Q ratios and the highest P_{CO_2} (13). In addition, stratified inhomogeneity, caused by incomplete gas mixing within respiratory units (4), may play a part. Although both forms of mismatching could theoretically be worsened by PEEP, this was not demonstrated in our cardiac surgery patients. We chose to investigate these subjects because PEEP is usually beneficial in restoring the loss of lung volume effected by anesthesia and cardiac surgery. The present results and those of Blanch et al. (1) in patients with acute lung injury show no comparable changes to those in dogs (2). In clinical situations in which PEEP is commonly applied, PEEP appears to have little effect on CO_2 elimination.

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