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Lung Carbon Dioxide Elimination Corralates With Physiologic Dead Space Volume During Mechanical Ventilatory Support

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Abstract: Increased mean airway pressure (P_{aw}) predisposes to increased alveolar dead space volume and, hence, physiologic dead space volume (V_{Dphys}). This is the result of overdistending alveoli, converting Zone 2 and Zone 3 units to Zone 1 units. Lung carbon dioxide elimination (LCO_2) is a reflection of pulmonary capillary blood flow. It is hypothesized that as Zone 1 units form or V_{Dphys} increases, LCO_2 decreases proportionately and eventually $PaCO_2$ increases. The purpose of this study is to determine if LCO_2 correlates with V_{Dphys} during mechanical ventilation.

Six sheep (66.3 ± 6.5 kg), anesthetized with sodium thiopental and paralyzed using pancronium, had pulmonary artery and arterial catheters inserted, and were intubated and ventilated [Fraction of inspired oxygen of 1.0, controlled mechanical ventilation]. Acute lung injury was induced by tracheal instillation of hydrochloric acid (pH 2.5, 0.25 mL/kg). Continuous positive airway pressure (CPAP) levels of 5, 10 and 20 cm H_2O were randomly applied. Cardiac output was maintained nearly constant at all CPAP levels. Data from flow/pressure and infrared capnometer sensors, positioned between the endotracheal tube and the "Y" piece of the breathing circuit, were directed to a commercially available respiratory monitor (Novametrix), which provided real time display of P_{aw} and LCO_2 (area under the exhaled volume and CO_2 curve integrated over 1 min). V_{Dphys} and the physiologic dead space volume to tidal volume ratio (V_D/V_T), calculated using the single breath CO_2 elimination technique, were also displayed on the monitor. Data were analyzed using regression analysis; alpha was set at 0.05 for statistical significance.

Conclusion: CPAP increases Paw, which correlated positively with V_{Dphys}. LCO_2 correlated negatively and $PaCO_2$ correlated positively with V_D/V_T. At V_D/V_T of approximately 0.5, LCO_2 began decreasing and $PaCO_2$ increasing. LCO_2 is simple to measure, and real time data provides useful clinical information, i.e., a noninvasive inference of changes in V_{Dphys} and $PaCO_2$ following application of positive pressure.

Key Words: Lung carbon dioxide elimination, CPAP, Dead space

Introduction

P_{aw} that is increased during positive pressure ventilation predisposes to increased alveolar dead space volume (V_{Dalv}) and physiologic dead space volume. Increased P_{aw}, as a result of overdistending alveoli, converts some of the zone 3 and zone 2 alveolar units into zone 1 alveolar units, which creates alveolar dead space volume. Fletcher and colleagues reviewed the topic of dead space volume and described the application of single breath CO_2 analysis in patients with abnormal pulmonary perfusion (2). Recently a single breath CO_2 analysis station was developed by Arnold and colleagues, which allows on-line quantification of airway and alveolar dead space (1). Physiologic dead space, measured by this method, is significantly correlated with the method defined by Bohr-Enghoff (r^2=0.84, p = .0001).

During positive pressure ventilation, increased P_{aw} is used to increase oxygenation, but after a certain level of P_{aw}, V_D/V_T increases due to increased ventilation to high V/Q regions and a larger anatomic dead space. Currently, monitoring V_{Dphys} or V_D/V_T is not practical under clinical conditions for preventing this relationship between P_{aw} and dead space volume.

Physiologic dead space should be smallest when there is maximal recruitment of perfused or functional gas exchange units without overdistention of alveolar areas

contributing to dead space volume. LCO_2 is a reflection of pulmonary capillary flow and an easy tool for real-time monitoring. Therefore, in sheep with ARDS induced by hydrochloric acid, we examined whether LCO_2 could be a simple method of monitoring the changes in physiologic dead space volume.

Materials and Methods

Six sheep weighing 66.3 ± 6.5 kg (mean ± SD) were anesthetized with sodium pentobarbital, 25 mg/kg, intravenously and intubated with cuffed 8.5-mm endotracheal tubes. A continuous infusion of sodium pentobarbital was administered at a rate of 0.25 to 1.5 mg/min. The animals were ventilated by delivering a tidal volume of 15 ml/kg at a frequency of 12 breaths and a fractional O_2 concentration in dry inspired gas = 1.0 throughout the experiment. Airway pressure was transduced at the level of the endotracheal tube connector and recorded continuously on a six – channel recorder as well as computerized real time respiratory monitor. An 18-gauge catheter was placed into the carotid artery and a 7 – French, flow-directed, thermistor – tipped, oximetric catheter was threaded into the pulmonary artery via external jugular vein. The position of the pulmonary artery catheter was confirmed by transduced pressure tracings. Thermal dilution cardiac output was determined from a cardiac output computer (oximetric SO_2/CO computer, Abbott Laboratories) by averaging three measurements obtained after injection of iced saline at end exhalation. Arterial blood was drawn from the carotid artery catheter and mixed venous blood from the pulmonary artery catheter for analysis of pH, PO_2, PCO_2, SaO_2 and SvO_2. These values were corrected to body temperature and to ambient barometric pressure.

Data from flow/pressure and infrared capnometer sensors, positioned between the endotracheal tube and the "Y" piece of the breathing circuit, were directed to the respiratory monitor (Ventrak, Novametrix Medical Systems). This respiratory monitor provided real time display of P_{aw} and LCO_2, which is the area under the exhaled volume of the CO_2 curve integrated over 1 minute. V_{Dphys} and V_D/V_T, calculated using the single breath CO_2 elimination technique, were also displayed on the monitor.

After control measurements were completed, acute lung injury was induced by tracheal instillation of hydrochloric acid (pH of 2.5, 0.25 mL/kg). Fifteen minutes were allowed for stabilization, after which all measurements were repeated at 0 cmH_2O CPAP. Measurements were then taken after 15 minutes at 5, 10 and 20 cmH_2O of CPAP applied in random sequence.

All data are reported as means ± 1 SD. The data were analyzed using regression analysis, Kruskal-Wallis and Mann-Whitney U test. Changes were considered significant only if the probability of chance occurrence was less than 0.05.

Results

There was no significant change in PaO_2 or intrapulmonary shunt at incremental levels of CPAP. $PaCO_2$ showed statistical significance at a CPAP of 20 compared with 5. The results are summarized with CPAP 5, CPAP 10 and CPAP 20 in the Table. The correlation between P_{aw} and V_{Dphys} was significant ($p < 0.001$, $r = 0.8$) (Figure 1). LCO_2 correlated negatively with V_D/V_T ($p < 0.001$, $r = -0.8$) (Figure 2). $PaCO_2$ had a strong positive correlation with V_D/V_T ($p < 0.05$, $r = 0.7$) (Figure 3). When V_D/V_T reached 50%, LCO_2 began decreasing and $PaCO_2$ began increasing. Cardiac out put was 3.85 ± 0.7, 3.88 ± 0.7 and 3.31 ± 0.7 l/min respectively with a CPAP of 5, 10 and 20. There was no statistically significant difference between cardiac out put levels.

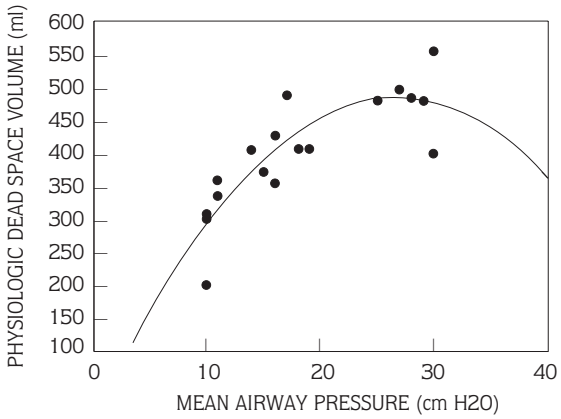

Figure 1. Correlation between P_{aw} and V_{Dphys} (r=0.8)

Discussion

Many attempts have been made to identify the appropriate level of CPAP to apply to a patient suffering from adult respiratory distress syndrome (ARDS). Each method has defined optimum in terms of a specific vari-

Table. Hemodynamic and respiratory parameters with changing CPAP levels.

Parameter	CPAP (5 cm H_2O)	CPAP (10 cm H_2O)	CPAP (20 cm H_2O)
	Mean ± SD	Mean ± SD	Mean ± SD
Paw (cm H_2O)	11.1 ± 1.9	16.6 ± 1.7*	28.1 ± 1.9*
LCO_2 (ml / min)	179.3 ± 13.4	170.6 ± 13.8#	167.1 ± 21.3
V_{Dphys} (ml)	312 ± 61.5	414 ± 42.5#	482 ± 49.2W
V_{Dana} (ml)	146.0 ± 72.1	226.0 ± 49.5	301.0 ± 63.2
V_{Dalv} (ml)	105.0 ± 70.4	167.3 ± 97.0	223.6 ± 119.9
V_D/V_T (ml)	0.5 ± 0.12	054 ± 0.11	0.59 ± 0.1
PH	7.32 ± 0.02	7.31 ± 0.03	7.29 ± 0.08
$PaCO_2$ (mm Hg)	44.0 ± 12.8	48.3 ± 9.6	48.3 ± 6.0#
PaO_2 (mm Hg)	78.6 ± 43.1	66.0 ± 27.3	69.3 ± 19.3
SaO_2	0.82 ± 0.22	0.79 ± 0.20	0.84 ± 0.11
$PETCO_2$ (mm Hg)	29.3 ± 3.8	32.1 ± 1.6	32.8 ± 3.7
$PETCO_2 – PaCO_2$	3.66 ± 1.5	22.3 ± 11.8#	16.1 ± 9.5#
SvO_2	0.62 ± 0.19	0.51 ± 0.21	0.56 ± 0.26
CO (l/min)	4.6 ± 2.0	3.9 ± 0.7	3.7 ± 0.9
Q_s/Q_t	0.59 ± 0.22	0.57 ± 0.12	0.53 ± 0.11
P_{AWP} (mm Hg)	8.5 ± 4.2	15.0 ± 8.2	12.1 ± 4.6

P_{aw}: Airway pressure, LCO_2: Lung carbon dioxide elimination rate, V_{Dphys}: Physiologic dead space, V_{Dana}: Anatomic dead space, V_{Dalv}: Alveolar dead space, V_D/V_T: Dead space to tidal volume ratio, SaO_2: Arterial oxygen saturation, $P_{ET}CO_2$: End-tidal carbon dioxide, SvO_2: Mixed venous oxygen saturation, CO: Cardiac out put, Q_s/Q_t: Shunt fraction, P_{AWP}: Pulmonary artery wedge pressure.
# $p < 0.05$, ¤ $p < 0.01$, * $p < 0.001$ (statistical difference with CPAP 5)

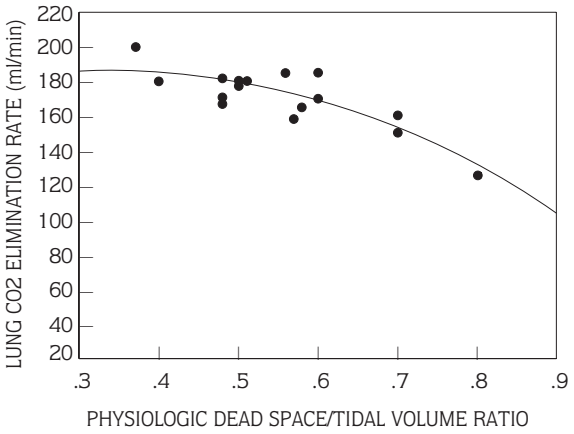

Figure 2. Correlation between LCO_2 and V_D/V_T (r=-0.8)

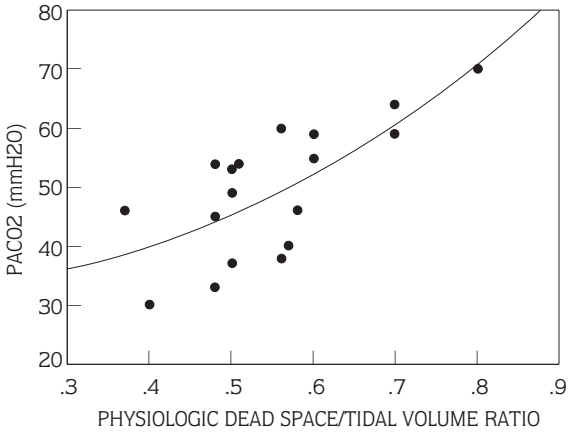

Figure 3. Correlation between $PaCO_2$ and V_{Dphys} (r=0.7)

able or group of variables measured (3-6). Sutter and colleagues found that increases in CPAP caused an increase in oxygen delivery (DO_2) up to a certain level, and, thereafter, further increases in CPAP caused a decrease in DO_2 (3). These conclusions were subsequently challenged. It was pointed out that if differences in patient characteristics are taken into account, no measurable physiologic variable is helpful in predicting the best CPAP in a given situation (7). Analysis of expired gas has been proposed as a useful noninvasive means of titrating end-expiratory positive pressure (4). In particular, computation of the arterial to end-tidal CO_2 gradient was found to be helpful in guiding positive end-expiratory pressure. It is reported that super syringe measurements of the volume-pressure curve assist with positive end expiratory pressure (PEEP) adjustments and that the best PEEP is pressure immedi-

ately above the inflection point (5,8). The best total compliance of the respiratory system has been shown in humans to correspond with maximal oxygen delivery and lowest V_D/V_T (3). Measurement of V_D/V_T was required for a ventilation system that can produce an inspiratory plateau or one that permits total obstruction of gas flow to reach a static equilibrium in the past but nowadays V_{Dalv} or V_{Dphys} can be measured with the single breath CO_2 elimination technique (1). It is shown that excessive positive pressure increases dead space (4). As dead space increases, the proportion of V_T that has actual contact with gas exchange units decreases and the PCO_2 of the V_T more closely resembles that of the PCO_2 of the atmosphere than that of the arterial blood. In other words, lung CO_2 elimination decreases with excessive positive pressure (4). Coffey and co-workers studied the mechanism of V_{Dphys} response to PEEP after oleic acid lung injury (9). They found that, following injury, V_D/V_TCO_2 increased. It decreased with 5 or 10 cmH_2O PEEP but increased progressively at higher PEEP levels. The increase in V_D/V_TCO_2 at 5 or 10 cmH_2O PEEP was due to reductions in shunt and midrange ventilation-perfusion heterogeneity. The increase in V_D/V_TCO_2 that occurred with higher PEEP levels was due to increased ventilation to high ventilation-perfusion regions and large anatomic dead space. Similarly, we found that as CPAP and P_{aw} increase anatomical dead space and V_D/V_T increases correspondingly LCO_2 decreases. In patients with cardiac surgery, positive end-expiratory pressure has little effect on LCO_2 (10). Since none of the patients had either acute lung injury or ARDS in this study our results are not comparable with their results.

Our results suggest that positive alveolar pressure during mechanical ventilation may increase V_D/V_T depending on the amount, and when V_D/V_T reaches 0.5, LCO_2 begins to decrease. While applying positive pressure or trying to find the best PEEP or CPAP in acute lung injury, LCO_2 can be used a good marker of increased dead space and can be used as parameter to titrate either PEEP or CPAP. The fact that applying higher levels of CPAP decreased the LCO_2 in all of the sheep studied is attributed to increased ventilation to high ventilation-perfusion regions and large V_{Dphys}.

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