Failure of Shallow Ventilation to Produce Pulmonary Shunting in the Anesthetized Dog

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Ten anesthetized, paralyzed dogs were ventilated mechanically with a constant shallow tidal volume and a respiratory rate of 60 breaths/min for four hours. V_D/V_T was found to be 74 per cent, and mean pH_a and $Paco_2$ were 7.40 and 31.7 mm Hg. $\dot{Q}_S/\dot{Q}_T$ averaged 3.6 per cent, with no significant variation throughout the study. Two additional groups of five dogs each were studied in the same manner; however, respiratory rates were maintained at 30 and 15 breaths/min, respectively, with V_T adjusted so that pH_a and $Paco_2$ remained essentially the same as in the first group. Minimal pulmonary shunting was again observed. Correlation analysis of results in all 20 animals showed no significant association between V_T and $\dot{Q}_S/\dot{Q}_T$. (Key words: Pulmonary shunting; $A\text{-}aDo_2$; Hypoxia; Anesthesia; Cardiac output; Alveolar ventilation; Hyperinflation of the Lungs; Ventilatory pattern; Tidal volume.)

INCREASING alveolar–arterial oxygen tension differences ($A\text{-}aDo_2$) during general anesthesia often have been attributed to ventilatory patterns deficient in deep breaths.[1–5] Large tidal volumes and periodic hyperinflation of the lungs have been reported to impede the development of this impairment in oxygenation.[1,2,6] Pulmonary right-to-left shunting ($\dot{Q}_S/\dot{Q}_T$) more accurately reflects arterial oxygenation than $A\text{-}aDo_2$, but is examined less frequently because the determination requires sampling of mixed venous blood from a catheter in the right heart. Oxygen content of central venous blood often approximates that for mixed venous blood, but the possibility of inadequate mixing is always present. Either method of determination far exceeds the accuracy of calculating $\dot{Q}_S/\dot{Q}_T$ from an assumed arteriovenous O_2 content difference which, in fact, adds no further information to the $A\text{-}aDo_2$. Even so, reported results of studies of $\dot{Q}_S/\dot{Q}_T$ during anesthesia vary widely,[3,7,8,9] and there seems to be no study relating true pulmonary shunting to the magnitude of tidal volume (V_T) or to alveolar ventilation. The purpose of the present study was to measure the effect of deliberate shallow ventilation on pulmonary shunting.

Methods

Healthy pedigreed beagle dogs were anesthetized with single intravenous doses of pentobarbital (Nembutal), 30 mg/kg. A 0.1 per cent succinylcholine infusion provided continuous paralysis. Following endotracheal intubation with a cuffed tube, the lungs were ventilated mechanically with a volume-limited ventilator. Oxygen was delivered to the dog via a nonrebreathing system. Absence of contamination of the inspirate by room air was verified by measuring end-expired N_2 with a Med-Science Electronics 305 AR Nitralyzer. The dogs were supine, and breathing was not restricted. Body temperature, monitored with an esophageal thermistor, remained between 36 and 37 C throughout the study.

There were three groups of dogs. The first ten animals were ventilated at 60 breaths/min. Five dogs comprised each of the second and third groups, with respiratory rates controlled at 30 breaths/min for one group and 15 for the other. During each experiment the rate was kept constant and the V_T adjusted at the beginning to maintain end-expired CO_2 tension at 20 to 25 mm Hg (Beckman LB-1 infrared CO_2 analyzer). Once such conditions were attained, no further adjustments of the respirator were made. Each study took four hours. After induction of anesthesia and onset of constant controlled breathing, two hours of

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TABLE 1. Results in Ten Dogs Ventilated at 60 Breaths/min

	2 hours	3 hours	4 hours
pH_a	7.40 ± 0.017	7.40 ± 0.022	7.40 ± 0.029
P_{aCO_2} (mm Hg)	30.9 ± 1.52	31.6 ± 2.19	32.6 ± 2.62
$V_D/V_T \times 100$	73.9 ± 1.86	76.1 ± 2.39	72.5 ± 2.93
V_T (ml/kg)	9.8 ± 0.94	9.8 ± 0.93	9.7 ± 0.98
A-aD_{O_2}* (mm Hg)	39.7 ± 8.8	51.6 ± 12.5	76.6 ± 17.5
$\dot{Q}_S/\dot{Q}_T \times 100$	3.18 ± 0.62	3.28 ± 0.61	4.30 ± 0.95
$C_{aO_2} - C\bar{v}_{O_2}$** (vol %)	3.53 ± 0.45	4.34 ± 0.52	4.47 ± 0.62
Cardiac index*** (l/m²/min)	5.4 ± 0.57	4.2 ± 0.52	4.1 ± 0.45

Values are means ± SE.
* $P < 0.01$ for hours 2–4 and 3–4.
** $P < 0.001$ for hours 2–3 and $P < 0.02$ for hours 2–4.
*** $P < 0.005$ for hours 2–3 and $P < 0.02$ for hours 2–4.

TABLE 2. Results in Five Dogs Ventilated at 30 Breaths/Min

	2 hours	3 hours	4 hours
pH_a	7.43 ± 0.029	7.46 ± 0.025	7.46 ± 0.021
P_{aCO_2} (mm Hg)	28.8 ± 1.1	28.4 ± 1.5	28.3 ± 1.3
$V_D/V_T \times 100$*	63.2 ± 4.0	52.3 ± 3.5	51.3 ± 5.3
V_T (ml/kg)	16.1 ± 1.9	15.6 ± 1.9	15.2 ± 1.8
A-aD_{O_2}** (mm Hg)	37.1 ± 9.8	64.6 ± 21	61.3 ± 16
$\dot{Q}_S/\dot{Q}_T \times 100$	2.42 ± 0.71	3.54 ± 1.04	3.07 ± 0.89
$C_{aO_2} - C\bar{v}_{O_2}$*** (vol %)	4.64 ± 0.60	5.02 ± 0.67	5.63 ± 0.61
Cardiac index (l/m²/min)	3.55 ± 0.50	3.15 ± 0.38	3.06 ± 0.39

* $P < 0.02$ for hours 2–4.
** $P < 0.05$ for hours 2–4.
*** $P < 0.02$ for hours 2–4 and $P < 0.05$ for hours 3–4.

TABLE 3. Results in Five Dogs Ventilated at 15 Breaths/Min

	2 hours	3 hours	4 hours
pH_a	7.40 ± 0.032	7.38 ± 0.021	7.39 ± 0.024
P_{aCO_2} (mm Hg)	33.4 ± 2.4	34.4 ± 2.0	34.5 ± 2.4
$V_D/V_T \times 100$	49.9 ± 1.8	41.5 ± 1.6	41.3 ± 2.4
V_T (ml/kg)	20.8 ± 3.4	18.7 ± 1.5	19.4 ± 1.5
A-aD_{O_2}* (mm Hg)	40.0 ± 18	53.5 ± 16	52.2 ± 22
$\dot{Q}_S/\dot{Q}_T \times 100$	3.14 ± 1.3	3.88 ± 1.2	3.30 ± 1.4
$C_{aO_2} - C\bar{v}_{O_2}$ (vol %)	3.58 ± 0.14	4.06 ± 0.45	4.41 ± 0.40
Cardiac index (l/m²/min)	4.37 ± 0.63	4.43 ± 0.68	3.85 ± 0.78

* $P < 0.01$ for hours 2–3 and $P < 0.02$ for hours 2–4.

stabilization preceded the initial measurements, which were repeated at three and at four hours. Minute volume and tidal volume were measured with a 13.5 l Collins Respirometer. Mixed expired gas was collected and analyzed for CO_2 with the Scholander apparatus.[10] Catheters were inserted into the pulmonary artery[11] and aorta through the jugular vein and femoral artery, respectively. Arterial and mixed venous blood samples were drawn anaerobically in heparinized glass syringes and iced immediately. An Instru-

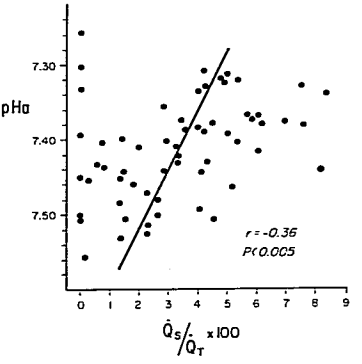

FIG. 1. Relationships between $\dot{Q}_S/\dot{Q}_T$ and pH_a during anesthesia with constant-volume ventilation.

mentation Laboratory gas analyzer (Model 113) with a modified Clark O_2 electrode,[12] a modified Severinghaus CO_2 electrode,[13] and a glass electrode for pH determination was employed in a constant-temperature water bath at 37 C. Both arterial and mixed venous O_2 tensions were corrected by tonometry,[14] and an additional correction was made for the loss of oxygen during the interval before analysis.[15] All determinations were made in duplicate. Cardiac output was determined by the dye-dilution technique,[16] with the dye curve recorded on a Beckman Cardiodensitometer. Standard formulae were used to calculate $\dot{Q}_S/\dot{Q}_T$, A-aDo_2, physiologic dead-space (V_D/V_T), cardiac output ($\dot{Q}$) and cardiac index.

Results

Table 1 shows data from the first group of ten dogs ventilated at a mean respiratory rate of 60.6 breaths/min with a V_T of 9.8 ml/kg body weight and a V_D/V_T of 74.2 per cent. pH_a averaged 7.40 and $Paco_2$, 31.7 mm Hg. At the 2nd, 3rd and 4th hours, $\dot{Q}_S/\dot{Q}_T$ values were 3.2, 3.3, and 4.3, respectively. A paired comparison revealed no significant differences from hour to hour. A-aDo_2 for the same interval showed significant increases from 40 to 52 to 77 mm Hg ($P < 0.01$), while cardiac index decreased from 5.4 to 4.2 to 4.1 l/m²/min ($P < 0.005$) as A-aDo_2 rose.

In the ten dogs ventilated at 30 (five dogs) and at 15 (five dogs) breaths/min (tables 2 and 3), $\dot{Q}_S/\dot{Q}_T$ values averaged 3.2 per cent, with an insignificant hourly variation. $Paco_2$ and pH_a for these groups were essentially unchanged.

Although the mean pH_a and $Paco_2$ levels were similar in all groups, individual variation from study to study provided an opportunity for further analysis. The scatter in figures 1 and 2 is large, but statistical correlation of the data disclosed statistically significant associations berween $\dot{Q}_S/\dot{Q}_T$ and both pH_a ($r = -0.36$; $P < 0.005$) and $Paco_2$ ($r = 0.299$; $P < 0.025$). However, there was no significant correlation between $\dot{Q}_S/\dot{Q}_T$ and V_T ($r = -0.087$; $0.6 > P > 0.5$ (fig. 3).

Discussion

During the past decade constant-volume respiration has been linked to the development of atelectasis,[17] so anesthetic techniques employing large tidal volumes or periodic sighs have become accepted practice. Reports that A-aDo_2 increases during anesthesia and that this increase can be reversed by hyperinflation of the lungs imply that large tidal volumes

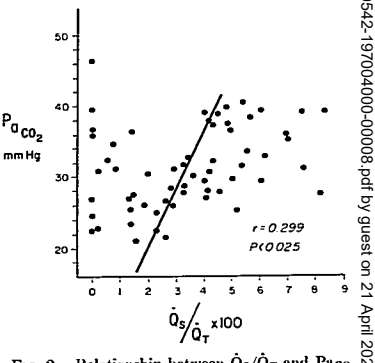

FIG. 2. Relationship between $\dot{Q}_S/\dot{Q}_T$ and $Paco_2$ during anesthesia with constant-volume ventilation.

are necessary.[1,2,6] In man these studies have been undertaken during surgical procedures, in which unrestricted thoracic expansion cannot be guaranteed and the patients' preexisting pulmonary status can only be estimated.[1,2,5,6,7,18] Under these conditions the observed impairment of oxygenation may be unrelated to the pattern of respiration. Similarly, animal experiments have been carried out with a chest tube compressing the lung,[4] or pulmonary shunting has been increased by intermittent negative pressure on the airways.[19,3] Poyart and Nahas showed that a healthy unmedicated dog (pH 7.40) has a $Paco_2$ of 32 mm Hg,[20] so studies of $\dot{Q}s/\dot{Q}T$ in the dog with $Paco_2$ above this value measure the effects of constant-volume hypoventilation.[4] Few studies of oxygenation during anesthesia have emphasized actual values of pH_a and $Paco_2$. Adequacy of alveolar ventilation is reflected by pH_a and $Paco_2$, and in the present study, when these values were normal, $\dot{Q}s/\dot{Q}T$ was minimal (figs. 1 and 2). There appears to be more correlation between pH_a and $\dot{Q}s/\dot{Q}T$ than between $Paco_2$ and $\dot{Q}s/\dot{Q}T$, but the explanation for this is not clear. In any case, there was a significant statistical correlation between respiratory acidosis and increased $\dot{Q}s/\dot{Q}T$, inviting criticism of studies of subjects with elevated $Paco_2$ levels.[1,2,6]

Several studies have shown that A-aDo_2 in the anesthetized patient does not always increase with time.[6,7,15,21] Others report minimal $\dot{Q}s/\dot{Q}T$ during anesthesia.[7,8,9] The data presented here demonstrate that $\dot{Q}s/\dot{Q}T$ remains minimal and stable without hyperventilation or hyperinflations. When the dogs (table 1) were ventilated deliberately with rapid shallow respiration, V_D/V_T was 74.2 per cent. $Paco_2$ and pH_a were maintained at levels which coincided with those reported in the normal unanesthetized state,[20] while $\dot{Q}s/\dot{Q}T$ showed no significant variation from hour to hour. Moreover, we found no correlation between depth of breathing and $\dot{Q}s/\dot{Q}T$ (fig. 3), indicating that V_T alone is not a primary determinant of arterial oxygenation.

Kelman *et al.*[22] predicted that if $\dot{V}E$, $\dot{V}o_2$ and $\dot{Q}s/\dot{Q}T$ all remained constant, then $\dot{Q}$ would vary inversely with A-aDo_2. This hypothesis was examined by Prys-Roberts *et al.*,[23,24] who altered cardiac output in patients by varying

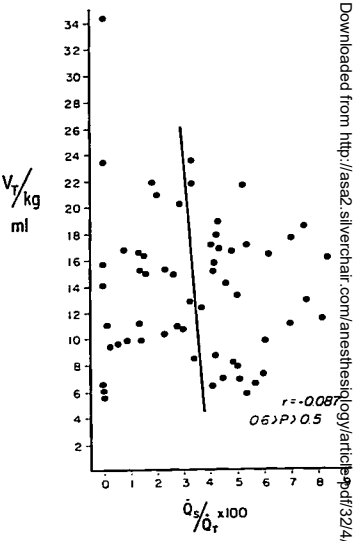

FIG. 3. Relationship between Qs/Q_T and tidal volume during anesthesia with constant-volume ventilation.

FIo_2 and noted a reciprocal relation with A-aDo_2. Similar results were reported by Michenfelder *et al.*[25] Philbin *et al.*[26] studied patients after cardiopulmonary bypass, in whom cardiac output reached very low levels. They found that lower cardiac indices were associated with decreased oxygenation, and that the relationship was augmented by increasing degrees of pulmonary shunting. Our findings point up the essential difference between $\dot{Q}s/\dot{Q}T$ and A-aDo_2, the former being less influenced by the state of the circulation. Cardiac output in our 20 dogs decreased during anesthesia (tables 1, 2 and 3). Gilmore also observed a decreasing $\dot{Q}$ in the spontaneously-breathing dog which received a single anesthetizing dose of pentobarbital.[27] The fall in $\dot{Q}$ in our animals coincided with an increasing A-aDo_2, with a relatively constant $\dot{Q}s/\dot{Q}T$.

In conclusion shallow constant-volume ventilation failed to produce significant pulmonary

shunting in the anesthetized dog during a four-hour period. Nor did shunting correlate with the tidal volume used. Therefore, we suggest that, with an intact lung–thorax system, neither hyperventilation nor hyperinflation may be necessary during uncomplicated anesthesia and operation. Furthermore, with an intact lung–thorax system the adequacy of alveolar ventilation is more important than the magnitude of tidal volume in maintaining arterial oxygenation.

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