THE EFFECT OF ARTIFICIAL VENTILATION ON FRC AND ARTERIAL OXYGENATION

1. RAPID SHALLOW VENTILATION AT NORMAL AND LOW ARTERIAL CARBON DIOXIDE TENSIONS*

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INTRODUCTION

FUNCTIONAL RESIDUAL CAPACITY (FRC) is reduced during anaesthesia¹ and in anaesthetized patients who are ventilated with intermittent positive pressure ventilation (IPPV).^{2,3} Rapid shallow ventilation (RSV) in which end-expiratory pressure remains slightly above zero may prevent this reduction.^{4,5} Jonzon *et al.*⁵ used high frequency ventilation in animals to promote stable cardiovascular conditions and Heijman, *et al.*⁴ used it for surgical patients.

Stone and Sullivan,⁶ however, found a significant increase in the alveolar to arterial oxygen tension gradient (AaDO₂) in dogs during high frequency ventilation, but in their studies carbon dioxide tensions (PaCO₂) were lower than those found in normal awake beagles in our own laboratory⁷ and that of Feigl and D'Alecy.⁸ Low PaCO₂ may cause a fall in cardiac output⁹ and a rise in oxygen consumption (VO₂),¹⁰ both of which might be expected to increase AaDO₂. Pichotka *et al.*¹¹ found an increase in AaDO₂ at a constant level of lowered alveolar CO₂ tension (PA_{CO₂}) and suggested that the AaDO₂ is mainly determined by PA_{CO₂}. Low regional PA_{CO₂} has also been associated with regional hypoventilation, decreased compliance and increased veno-arterial shunting.¹²

We therefore compared the effect of rapid shallow ventilation at a constant minute ventilation (VE) on arterial oxygenation in dogs maintained at normal and low PaCO₂ values for one to two hours. In some of the dogs we also compared FRC early and late during the period of artificial ventilation.

METHODS

Dogs were anaesthetized with intravenous pentobarbitone (30 mg/Kg), intubated with a cuffed tracheal tube and ventilated with air at a rate of 60–80/

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TABLE I
RAPID SHALLOW VENTILATION
PaO₂ and AaDO₂ (mm Hg)
(Mean and standard deviation)

	Normal PaCO ₂			Low PaCO ₂			
	Early	Late	Difference	Early	Late	Difference	
PaCO ₂	38 3	38 4	0	26 2	24 3	-2	φ _E φ _L
PaO ₂	88 8	91 4	+3	88 15	78 16	-10*	φ _L
PAO ₂	102 9	102 7	0	117 2	119 2	+2	φ _E φ _L
AaDO ₂	12 7	11 6	-1	29 14	41 17	+12*	φ _E φ _L

*Significant change from early to late within group.

φ_E, φ_L: Significant difference between normal and low PaCO₂ groups, early and late respectively.

minute using the Volgyesi ventilator,¹³ which enabled us to measure FRC.¹⁴ A catheter was placed in the femoral artery to collect arterial blood. Values for PaO₂, PaCO₂ and pH were measured and corrected for changes in the dog's temperature, which were minimized by placing the animal on a heated blanket. We measured volume and oxygen and carbon dioxide content of expired gas.

After PaCO₂ had stabilized at low values in one group of five dogs, and at normal values in a second group of five dogs, control measurements of PaO₂ and PaCO₂ were made and expired gas was collected and analysed. $\dot{V}O_2$ and carbon dioxide elimination ($\dot{V}CO_2$) were measured and respiratory exchange (R) and PA_{O₂} were calculated. FRC was then measured in four dogs of each group and rapid shallow ventilation was continued at the same VE and PaCO₂ for one to two hours, when the measurements and calculations were repeated.

In each group differences between the early control and late values were calculated and their significance was assessed by the paired t-test. The significance of the difference between values in the two groups of dogs at normal and low PaCO₂ levels was assessed by Student's t-test.

RESULTS

In the dogs ventilated at constant low PaCO₂, mean values for PaO₂ fell significantly during the period of rapid shallow ventilation. AaDO₂ increased significantly in these dogs and was significantly higher than in the dogs ventilated at normal PaCO₂, both at the beginning and end of the period of ventilation. Significant differences between the dogs at normal PaCO₂ and those at low PaCO₂ were also observed in the values for PaCO₂, PaO₂ (late values only), PA_{O₂} and f (Tables I, II, III).

No large change in mean FRC was observed during rapid shallow ventilation, in either group. FRC remained high in both groups (Table III).

TABLE II
RAPID SHALLOW VENTILATION
 $\dot{V}_{O_2}$ and R
(Mean and standard deviation)

	Normal PaCO ₂			Low PaCO ₂		
	Early	Late	Difference	Early	Late	Difference
$\dot{V}_{O_2}$ (ml STPD)	48 12	51 11	+3	55 16	55 17	0
$\dot{V}_{O_2}$ (ml/Kg STPD)	4.00 0.70	4.24 0.49	+0.24	5.68 2.48	5.68 2.51	0 0
R	.83 .17	.82 .18	-0.02	.83 .08	.80 .11	-0.03

No significant changes.

TABLE III
RAPID SHALLOW VENTILATION
Ventilation and FRC
(Mean and standard deviation)

	Normal PaCO ₂			Low PaCO ₂			ϕ_E	ϕ_L
	Early	Late	Difference	Early	Late	Difference		
f(/min)	73 1.2	74 1.9	+1	65 1.3	66 2.5	+1		
VT (ml ATPS)	70 16.2	69 14.1	-1	85 11.4	84 10.0	-1		
FRC (ml BTPS)	632 38	621 43	-11	457 173	527 216	+70		
FRC (ml/Kg BTPS)	50 5	49 4	-1	47 12	54 18	+7		

ϕ_E , ϕ_L : Significant difference between normal and low PaCO₂ groups, early and late respectively.

DISCUSSION

The principal differences between the two groups of dogs is a fall in PaO₂ which occurred in spite of a rise in PAO₂ in the hyperventilated dogs. A similar fall in PaO₂ and an increase in the AaDO₂ gradient has been reported during hyperventilation at a constant low PaCO₂ by Pichotka *et al.*¹¹ Stone and Sullivan⁶ also noted increasing AaDO₂ in dogs ventilated rapidly at PaCO₂ values lower than those measured in normal conscious dogs.^{7,8} This increase in AaDO₂ was not due to increased true shunt but could be associated with a fall in cardiac output (Q) or increased $\dot{V}_{O_2}$ or an increased maldistribution of ventilation in relation to perfusion.

The use of gas exchange equations at a tidal volume lower than deadspace volume may lead to error.¹⁵ The tidal volumes in the present study are sufficiently above deadspace volume to give reasonable values for R and PAO₂.

In our studies the raised PAO₂ resulting from hyperventilation was not paralleled by an increase in PaO₂. Khambatta and Sullivan¹⁶ also report that in dogs hyperventilated with air, PaO₂ does not rise as much as expected.

We were unable to differentiate between the effects of changes in cardiac

output, shunt, or venous admixture. Although $\dot{V}O_2$ was higher in hyperventilated dogs the difference between the two groups of dogs was not significant and the values for $\dot{V}O_2$ remained constant throughout the hyperventilation. It seems likely, therefore, that falls in cardiac output, or increasingly inappropriate distribution of ventilation in relation to perfusion, or both, were responsible for the decrease in arterial oxygenation. Low CO_2 levels in alveolar and bronchial gas may also have caused regional hypoventilation.¹²

The important consideration is that when $PaCO_2$ was normal no decrease in arterial oxygen tension or increase in $AaDO_2$ occurred during rapid shallow ventilation over the period studied. PaO_2 remained close to the average value for conscious dogs breathing air⁸ and during both the early and late measurements $AaDO_2$ was below the values measured by others in anaesthetized ventilated dogs.¹⁷ If we insert the blood gas values measured in these dogs at normal $PaCO_2$ into the ventilation shunt grid of Owen-Thomas¹⁸ the amount of venous admixture is in the region of 5 per cent of cardiac output, which is less than that usually measured in anaesthetized dogs^{16,21} or humans.²²

The mean values for FRC which we measured during rapid shallow ventilation (52.0 ml/Kg and 47.0 ml/Kg in the normal and low $PaCO_2$ groups respectively) are considerably higher than those in conscious, prone dogs (40.3 ml/Kg at mean $PaCO_2$ of 36 mm Hg¹⁹ and those for anaesthetized dogs breathing spontaneously (26.7 ml/Kg).²⁰

We conclude that over a period of 1 to 2 hours rapid shallow ventilation with air is able to maintain FRC at values slightly above those in conscious dogs and that if $PaCO_2$ is kept within normal limits, no fall in PaO_2 or increase in $AaDO_2$ will occur. This pattern of artificial ventilation may, therefore, be useful in preventing the falls in FRC and increases in venous admixture described with other techniques of artificial ventilation in anaesthetized subjects.

RÉSUMÉ

La capacité fonctionnelle résiduelle est diminuée durant l'anesthésie et chez les patients ventilés avec pression positive intermittente.^{2,3} Une ventilation rapide et superficielle avec maintien d'une pression légèrement positive en fin d'expiration peut prévenir cette réduction.^{4,5}

Nous avons étudié les effets d'une ventilation rapide et superficielle chez deux groupes de chiens. Le premier groupe de chien a été hyperventilé, la $PaCO_2$ étant maintenu basse de façon constante; les valeurs moyennes de la PaO_2 diminuèrent de façon significative durant le ventilation rapide et superficielle. Le second groupe fut ventilé de façon à maintenir le $PaCO_2$ à des chiffres normaux.

Les capacités fonctionnelles résiduelles ont été mesurées durant les périodes de ventilation superficielle et rapide et les chiffres moyens obtenus montrent une augmentation considérable par rapport à ceux que l'on retrouve chez les chiens conscients en position couchée et par comparaison avec ceux trouvés chez les chiens anesthésiés et respirant spontanément.

Nous concluons qu'une période d'une à deux heures de ventilation rapide et superficielle avec de l'air peut maintenir des capacités résiduelles fonctionnelles légèrement supérieures à celles trouvées chez des chiens conscients et nous concluons de plus que si les PaCO_2 sont maintenues normales il n'y aura pas de chute de la PaO_2 et le gradient alvéolo-capillaire n'augmentera pas. Ce genre de ventilation peut donc être utile pour prévenir des chutes de la capacité fonctionnelle résiduelle et les augmentations du shunt décrites avec les autres techniques de ventilation artificielle chez les malades anesthésiés.

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