EFFECT ON PULMONARY GAS EXCHANGE OF VARIATIONS IN INSPIRATORY FLOW RATE DURING INTERMITTENT POSITIVE PRESSURE VENTILATION

BY

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SUMMARY

Eleven patients anaesthetized with sodium thiopentone, tubocurarine and halothane and ventilated with constant volume intermittent positive pressure were the subjects of a study of the effects of changes in inspiratory flow rate on alveolar-arterial oxygen difference, physiological shunt and physiological deadspace. The physiological deadspace increased significantly when inspiratory flow rate was raised above 25 l./min, but no change was demonstrable in venous admixture effect. The magnitude of the values derived and the possible mechanisms involved are discussed. The existence of a series of negative feed-back loops, with respect to the reduction of alveolar-arterial oxygen gradient, by intermittent positive pressure, is suggested.

Flow and pressure patterns, delivered by various mechanical ventilators, differ widely (Fairley and Hunter, 1964) and have varying effects on cardiac output and on ventilation/perfusion relationships. The effect of inflation pattern on cardiac output was established in 1948 by Cournand and his colleagues, who showed that the lowest mean airway pressure was optimal. More recently, Bergman (1963) has shown, in dogs, that the highest mean airway pressures produce the best ventilation/perfusion circumstances.

This study was designed to indicate the effect of inspiratory flow rate during intermittent positive pressure ventilation on pulmonary gas exchange, preparatory to a comparison of the effects of different inspiratory flow patterns. Eleven patients, aged 18–62 years and weighing 111–200 lb., were studied (table 1). All were clinically normal from the cardiopulmonary standpoint. Ten comparisons of gas exchange were made, at inspiratory flow rates of 25, 50, 75 and 100 l./min, using constant volume ventilation at 15 b.p.m.

METHOD

Anaesthesia. Each patient was premedicated with papaveretum and hyoscine, anaesthesia being induced with sodium thiopentone and tubocurarine, following which a 9.5 mm cuffed endotracheal tube was introduced. Anaesthesia was maintained with 0.5 per cent halothane from a Fluotec vaporizer, using 70 per cent nitrogen and 30 per cent oxygen as a vehicle as a means of minimizing problems of denitrogenation associated with the use of nitrous oxide.

Ventilation was maintained with an Air-Shields Ventimeter-Ventilator, selected because of its ability to maintain a constant volume and its square inspiratory flow pattern (Fairley and Hunter, 1963), the latter simplifying the setting up of each mean inspired flow rate. The ventilator was connected to the endotracheal tube through an electronically activated non-return valve (fig. 1). This consists of two solenoid valves, opening and closing in such a sequence that no inspiratory gas crosses to the expiratory part of the valve and, similarly, no expiratory gas is rebreathed. One hundred per cent expiratory gas collection with zero leak was previously checked by pneumotachographs on both limbs of the valve. The valve sequence is as follows. At end-expiration, the inspiratory valve being closed, the inflation cycle starts and the initial rise in pressure is sensed by a transducer proximal to the closed valve. Immediately, the expiratory valve closes and then the inspiratory valve opens. Inflation is terminated by an adjustable time-delay circuit, set in action by the initial rise in pressure. At end-inflation,
both valves are momentarily closed before the expiratory side opens. This somewhat complex valve is considered an advantage in the collection of a true sample of expired gas during intermittent positive pressure ventilation, when the high pressures and flows may exaggerate errors created by leaks occurring across most non-return valves (Loehning, Davis and Safar, 1964).

**Flow** was measured by a Fleisch No. 2 pneumotachograph, placed between the non-return valve and the endotracheal tube, and was recorded on a Grass Model 5 Polygraph, using a Statham PM5 differential transducer, with the capacity of both sides balanced as described by Brown and Elam (1964).

**Airway pressure** was measured at a point between the pneumotachograph and endotracheal tube, using a Statham transducer.

**Expired gas** was collected in a 100-litre Douglas bag and its volume measured by a dry gas meter.

**Arterial samples** drawn from the radial artery, were analyzed in duplicate by Clarke oxygen, Severinghaus carbon dioxide and Metrohm pH electrodes using the Epsco amplification and null-balance readout system. Later in the series, oxygen tension was determined with the Radiometer electrode and amplification system. All values for any one patient were determined on the same electrode. A correction factor for blood-gas difference, determined by tonometry of samples of each patient’s blood, was applied to values obtained with the Clarke macro-electrode.

**Expired carbon dioxide tension** was determined by a Beckman LBII infra-red analyzer and **expired oxygen tension** by the oxygen electrode.

**Inspired oxygen tension** was measured by oxygen electrode, from duplicate samples drawn from tanks containing a mixture of nitrogen and oxygen, ignoring the tension changes produced by the subsequently added halothane.

**Procedure.**

In each patient the lungs were ventilated 15 times per minute to a tidal volume 1.5 times that predicted for his weight by the Radford nomogram. Having established an inspiratory flow rate of 25 l./min, inspiratory time was adjusted to give the necessary tidal volume, using a Wright Respirometer on the expiratory limb of the valve as an indicator. Expiratory time was then adjusted to give a respiratory rate of 15 per minute. After at least 20 minutes, at constant ventilation, expired gas was collected over a 5-minute period. Respiratory rate was recorded from the flow tracing on the Grass Polygraph. Arterial blood was drawn anaerobically during the middle of the gas collection period and analyzed immediately. Following each such half-hour period of ventilation and gas collection, the patient’s lungs were inflated vigorously, by hand, a further dose of tubocurarine administered and the next ventilatory pattern established (i.e. the same tidal volume and frequency but with the next inspiratory flow rate setting). In alternate patients, the order of gas collection was for flows of 25, 50, 75, 100 l./min and 100, 75, 50, 25 l./min, in an attempt to expose any differences dependent upon time, such as falling compliance or unsteady state for the anaesthetic agent, nitrogen or carbon dioxide.

**Calculations.**

Expired volume per minute was corrected to BTPS from the equation:

\[
VE_{BTPS} = VE_{ATPS} \left( \frac{PB - PHO (ambient temp)}{PB - PHO (patient temp)} \right) \left( \frac{273 + Tc pt.}{273 + Tc amb.} \right)
\]
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<th>Mean insp. press. (cm H₂O)</th>
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Table I
Total data for eleven individuals. Subject 5 was studied twice—once in each direction. Subjects 10 and 11 were excluded from statistical analyses.
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**Gastrectomy, hypotension**

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* *Inspired gas changed.*
The physiological deadspace was calculated from the modified Bohr's equation:

$$V_{D,phys} = \left( \frac{P_{A,O_2} - P_{E,O_2}}{P_{A,O_2}} \right) V_{E,ETP} - V_{D,MECH}$$

The alveolar oxygen tension was derived from the solution of a modified alveolar air equation:

$$F_{A,O_2} = F_{I,O_2} - \frac{P_{A,O_2}}{P_{E,O_2}} (F_{I,O_2} - F_{E,O_2})$$

substituting arterial carbon dioxide for alveolar carbon dioxide values. Physiological shunt* (Rahn and Farhi, 1964) was derived from the equation:

$$O_S = \frac{C_{a,o_2} - C'a_o}{C'O_2 - C'a_o}$$

assuming a constant A–V difference of 3.5 ml per cent (Nunn, 1964), i.e.

$$O_S = \frac{(S_a,o_2 \times (Hgb \times 1.34/100) + P_{a,o_2} \times 0.003) - [S'o_2 \times (Hgb \times 1.34/100) + P_{a,o_2} \times 0.003]}{[C_{a,o_2} - 3.5] - C'a_o}$$

$P_{a,o_2}$ was converted to the patient's temperature, using the Severinghaus nomogram (1958). $S_a,o_2$ was derived from $P_{a,o_2}$, measured at 37°C and using the Severinghaus nomogram after correction for pH. $S'o_2$ was derived from the same nomogram, using $P_{a,o_2}$ calculated from the alveolar air equation and corrected to 37°C.

The respiratory gas exchange ratio, $R$, was derived from the equation:

$$R = \frac{V_{CO_2}}{V_{O_2}} = \frac{F_E{CO_2} \cdot V_E}{(F_{I,O_2} \cdot V_I) - (F_E{O_2} \cdot V_E)}$$

Where $V_I = V_E \cdot \frac{1 - F_E{CO_2} - F_E{O_2}}{F_{I,O_2}}$

**RESULTS**

Table I shows results obtained from twelve studies in eleven patients. Patient 5 was studied on two occasions, once in each direction. Patients 10 and 11 were excluded from the statistical analyses as hypotension occurred during surgery and was likely to have produced a variable independent of flow change.

Figure 2 shows the comparison of alveolar-arterial oxygen gradients at various inspiratory flow rates, the arrows indicating the direction in which the study proceeded. All values are above normal, as with other published figures obtained during intermittent positive pressure ventilation (Campbell, Nunn and Peckett, 1958). The higher figures are from the older patients.

Figure 3 indicates the means of all ten studies. The same information quoted in terms of physiological shunt, is shown in figures 4 and 5. Figure 6 compares physiological deadspace changes, ex-

* The term "physiological shunt" refers to that quantity of blood which reaches the arterial circulation without attaining equilibrium with the alveolar oxygen tension, whether due to ventilation/perfusion disturbance, diffusion gradient or "true" venous admixture due to anatomical shunt. The total alveolar-arterial oxygen difference is quantitated as though due to admixture of mixed venous blood and is frequently termed the "venous admixture effect". Anatomical shunt may, to a large extent, be distinguished from total physiological shunt by raising the inspired oxygen level to 100 per cent. This was not considered practicable in this instance.
Alveolar-arterial oxygen tension differences plotted against mean inspiratory flow rate. Arrows indicate order in which studies proceeded. Dotted lines join values for subjects 10 and 11.

Fig. 2

Data shown in figure 2, expressed as physiological shunt.

Fig. 4

Physiological deadspace/tidal volume ratio plotted against mean inspiratory flow rate. Arrows indicate direction in which studies proceeded. Dotted lines join values for subjects 10 and 11.

Fig. 6

Mean values for ten studies. Vertical bars indicate ± two standard errors of the mean.

Fig. 3

Mean values for ten studies. Vertical bars indicate ± two standard errors of the mean.

Fig. 5

Mean values for ten studies. Vertical bars indicate ± two standard errors of the mean.

Fig. 7
Statistical analysis.

There is a considerable scatter of values between individuals, consequently initial comparisons were made on the basis of paired differences between values obtained at 25 and 100 l./min; "t" tests showed no significant differences in A—a Po$_2$ gradient or in physiological shunt, in this series. However, there was a very significant increase in deadspace/tidal volume ratio at 100 l./min (P<0.01). Further paired "t" tests showed a very significantly greater deadspace/tidal volume ratio, at all values above 25 l./min (P<0.01) but no difference between 50, 75 and 100 l./min.

A regression comparison between the two groups of patients in figure 6 showed no difference in slopes obtained by starting either at high flows or at low flows, suggesting that factors dependent upon time did not contribute to the findings.

DISCUSSION

The order of magnitude of values for A—a oxygen difference, physiological shunt and deadspace, confirm those found previously under anaesthesia (Bendixen et al., 1963; Askrog et al., 1964a; Nunn, 1964).

Values for physiological deadspace/tidal volume ratio were all higher than normal resting, spontaneously breathing adults (Larson and Severinghaus, 1962). The figures are even more significantly raised when considered in terms of normal values for the intubated patient. No allowance was made in these calculations for the fact that the upper airway was bypassed.

Halothane may contribute to this increase, by reducing pulmonary perfusion and by bronchodilatation. This increased effect, relative to other agents, is suggested by data for physiological deadspace, collected by Askrog and associates (1964a). The absence of any obvious time effect in this small series is in disagreement with Askrog and this may be explained by the intermittent maximum inflations in this instance.

The results obtained suggest a critical level for inspiratory flow rate, above which deadspace increases. This is presumably a function of the critical velocity for the inspiratory gases and the airway resistance. The existence of such a critical level, during intermittent positive pressure ventilation has been suggested by Watson (1962).

It might be assumed that the increasing deadspace with the higher inspiratory flow rates is due to an increase in the alveolar component, resulting from abnormal distribution of inspired air and the preferential ventilation of some alveoli in excess of perfusion. Since minute volume remained approximately constant, one must assume that increased ventilation of one part of the lung must be accompanied by decreased ventilation in another. The absence of any associated venous admixture effect, in this series, when deadspace increased, requires explanation. The explanation may lie in the small change in oxygen gradient to be anticipated, when compared with the relatively high initial gradients. Other factors, more potent than flow change, may have produced an overriding effect and may not be satisfactorily randomized in this study—in particular, the extent and effectiveness of the maximum inflations delivered at the end of each sampling period. Also, the relatively high tidal volumes and inspired oxygen level would tend to minimize changes in venous admixture effect.

The high figures for A—a oxygen difference and for physiological shunt are in agreement with those reported by Nunn (1964) for a series of spontaneously breathing patients anaesthetized with halothane. The figure of 3.5 ml, assumed in this and in Nunn's series for A—V oxygen difference, is probably low (Theye and Tuohy, 1965), in which case values for physiological shunt may be exaggerated. However, the figures obtained have obvious clinical implications, as shown by the fact that in this series of patients, breathing a mixture with a mean inspired oxygen tension of approximately 222 mm Hg, at an arterial carbon dioxide tension of 31.7 mm Hg, the arterial oxygen tension has a mean value of only 132 Tim Hg (table I). Two standard deviations would include arterial oxygen tensions as low as 79 mm Hg. Clearly, lower inspired oxygen tensions would be hazardous.

The aetiology of the high A—a oxygen difference is not clear from this study but ventilation/perfusion abnormality is most likely. The present data fail to reveal any dependency on inspiratory flow rate and, in the seven patients in whom it was recorded, there was no statistically significant correlation between mean airway pressure and A—a oxygen difference, as opposed to the findings,
in dogs, of Bergman (1963). This may be due to the small pressure range produced by the variations in flow used.

Attention has been drawn, in recent years, to the effect of tidal volume history (Hedley-White, Laver and Bendixen, 1964), intermittent sighs (Bendixen et al., 1964) and FRC (Severinghaus and Stupfel, 1957; Nunn et al., 1965) on the A–a oxygen gradient. However, this may be a relationship with a negative feed-back loop when produced by intermittent positive pressure ventilation, i.e. the factors producing the improvement may simultaneously produce offsetting effects. Thus, as FRC is increased and tidal volume raised, by intermittent positive pressure, mean intrathoracic pressure rises and cardiac output may fall. Any resulting increase in A–V oxygen difference would produce a fall in mixed venous tension. In addition, mixed venous and arterial oxygen tensions will be lowered by hypocarbia, due to the associated shift to the left of the oxygen dissociation curve (Rahn and Fenn, 1955). The effect of this on arterial levels would depend, in turn, on the extent of the initial physiological shunt and on the alveolar oxygen tension. Thus, in addition to factors recognized as controlling the A–a oxygen difference during spontaneous ventilation, intermittent positive pressure may introduce a negative feed-back relationship dependent upon mean intrathoracic pressure and its effect on cardiac output, and upon arterial carbon dioxide tension. The effect of this negative feed-back should be most evident when alveolar oxygen tensions are not markedly elevated and when an increased physiological shunt is present. An additional component in the anaesthetized patient is the effect of the agent on cardiac output and the consequences of this on mixed venous oxygen tension.

It is conceivable that one might approach 100 per cent negative feed-back under certain circumstances, with the result that A–a oxygen difference would remain unchanged. Indeed, with increasing hyperventilation by intermittent positive pressure in patients with an increased physiological shunt, it is possible to demonstrate an initial stability of A–a oxygen gradient and then, eventually, a paradoxical increase with arterial tensions falling below initial values (Fairley, 1966).

The magnitude of the values obtained in this series may well be peculiar to the circumstances of anaesthesia and hyperventilation. It is probable, however, that the changes produced by variations in flow rate are typical of a wider range of circumstances and that these changes may be exaggerated in patients with bronchopulmonary disease.

The two patients not included in the statistical analyses showed the effects of hypotension on A–a oxygen gradient and on deadspace, described by Askrog, Pender and Eckenhoff (1964b) and help underline the significance of this complication in pulmonary gas exchange.

ACKNOWLEDGEMENT
This project was supported in part by a grant from the Medical Research Council of Canada.

The authors wish to acknowledge the contribution made by Mr. O. Z. Roy, of the Radio and Electrical Engineering Division of the National Research Council of Canada, in developing the electronically actuated non-return valve.

REFERENCES


BRITISH JOURNAL OF ANAESTHESIA

L’EFFET SUR LES ÉCHANGES GAZÉUX PULMONAIRES DES VARIATIONS DU COURANT INSPIRATOIRE PENDANT LA VENTILATION EN PRESSION POSITIVE INTERMITTENTE

SOMMAIRE

Onze malades anesthésiés par le thiopentone sodique, la tubocurarine et l’halothane et ventilés avec un volume constant sous une pression positive intermitente ont été les sujets d’une étude sur les effets des changements de la vitesse du courant inspiratoire sur le gradient alvéolo-artériel de l’oxygène, le shunt physiologique et l’espace mort physiologique. L’espace mort physiologique a significativement augmenté quand la vitesse du courant inspiratoire était élevée au-dessus de 25 l./min., mais on n’a pu démontrer de changement dans l’effet de mélange veineux. On discute l’intensité des modifications des chiffres et le mécanisme possible qui est en cause. On suggère l’existence d’une série de boucles de feed-back négatif, en relation avec la réduction du gradient alvéolo-artériel de l’oxygène, par la pression positive interminente.

DIE WIRKUNG VON VERÄNDERUNGEN DER INSPIRATORISCHEN GAZFÜHR WAHREND DER BEATMUNG MIT POSITIVEM WECHSELDRUCK AUF DEN GASAUSTAUSCH DER LUNGEN

ZUSAMMENFASSUNG