THE EFFECT OF VARYING INSPIRATORY:EXPIRATORY RATIOS ON GAS EXCHANGE DURING ANAESTHESIA FOR OPEN-HEART SURGERY

BY

M. K. SYKES AND JEAN LUMLEY

SUMMARY
Measurements of physiological deadspace/tidal volume ratio (VD/VT per cent) and alveolar-arterial oxygen tension difference (A-aPo\(_2\)) were made during mechanical ventilation in patients undergoing open-heart surgery. The inspiratory:expiratory time ratios studied were 0.5:2.5 and 1.5:1.5 before perfusion and 0.5:2.5, 1:2 and 1.5:1.5 after perfusion. Pre-perfusion there were no significant differences in VD/VT or A-aPo\(_2\) between the two patterns of ventilation. Post-perfusion the A-aPo\(_2\) increased. Although there were no significant differences in A-aPo\(_2\) between any of the three ratios examined after perfusion, the VD/VT per cent was significantly less when the inspiratory time was 1.5 seconds than when it was 0.5 or 1.0 second.

The changes in lung function resulting from an alteration in the duration of inspiration during mechanical ventilation have been studied in dogs by Bergman (1963), in patients recovering from poliomyelitis by Watson (1962a) and in patients with normal lungs by Fairley and Blenkarn (1966) and Bergman (1967). The present studies were designed to show whether the alterations in lung function noted by these authors would be affected by the cardiopulmonary changes resulting from open-heart surgery involving total cardiopulmonary bypass.

METHODS
The eighteen patients studied had no evidence of right-to-left intracardiac shunting on pre-operative examination or cardiac catheterization. Premedication consisted of a quinalbarbitone suppository 2–3 mg/kg 2 hours before operation, followed by an intramuscular injection of pethidine 2 mg/kg and promethazine 1 mg/kg 1 hour pre-operatively. Sodium thiopentone, 100–250 mg, was used to induce anaesthesia and muscular relaxation was obtained with tubocurarine, 30–40 mg. Anaesthesia was maintained with nitrous oxide 70 per cent, oxygen 30 per cent and incremental doses of pethidine and tubocurarine as needed. Ventilation was controlled by an Engström ventilator (Engström, 1963), the respiratory rate being held constant at 20 b.p.m. The minute volume was calculated from the nomogram of Engström and Herzog (1959). During bypass the lungs were held inflated with an air-oxygen mixture (approximately 50 per cent oxygen) or were ventilated with a similar mixture at 2–6 l./min. In all cases, an end-expiratory pressure of 5–10 cm H\(_2\)O was maintained during bypass. At the end of bypass, the lungs were repeatedly inflated to pressures of 30–40 cm H\(_2\)O to re-expand any areas of atelectasis.

A Barnet Mk.II ventilator (Rochford, Welch and Winks, 1958) replaced the Engström ventilator during the periods of study. Gases from the Rotameters on the Engström were diverted into the Barnet ventilator and the spring on the positive pressure bellows of the latter was tightened to a maximum so that a pressure of 40 cm H\(_2\)O was available to drive the gases into the patient’s lungs. This pressure proved adequate to empty the positive pressure bellows completely in all patients studied. The gas flow rate into the patient was adjusted by the inspiratory flow rate control on the machine, so that inspiratory flow ceased just before the solenoid valve cycled to the expiratory position. This adjustment was made after each change of inspiratory time. The inspiratory times studied were 0.5 and 1.5 sec pre-perfusion and 0.5, 1.0 and 1.5 sec post-perfusion. Since
the respiratory rate was 20 b.p.m. the I:E ratios were 0.5:2.5, 1:2 and 1.5:1.5.

To separate the true expired gas from the gas compressed in the ventilator tubing a pressure-operated collect valve (Sykes, 1969) was inserted into the circuit in place of the patient Y-piece (fig. 1). This valve had a small deadspace (12 ml) and a low resistance to expiration (0.5 cm H₂O at 30 l./min flow). A Wright respirometer was interposed between the expiratory port of the collect valve and the Douglas bag. This enabled the circuit to be checked for leaks by comparing the expired volume with the Rotameter settings.

![Diagram of circuit used.](image)

It also provided a continuous check on the functioning of the collect valve. Further checks for leaks were carried out when the lungs were hyperinflated before each set of measurements. This was accomplished by occluding the expiratory tube leading to the Douglas bag until a pressure of 30–35 cm H₂O had built up in the circuit. Any leak in the circuit was then shown by a failure to maintain a horizontal pressure plateau during the expiratory phase. The inspired oxygen concentration was monitored continuously by a paramagnetic oxygen analyzer, and the end-expired carbon dioxide concentration was monitored by a rapid infra-red analyzer. The total sample volume was 150–200 ml/min, except during the period of measurement, when sampling was discontinued.

Measurements were made 30–70 minutes after induction of anaesthesia and 30–120 minutes after the end of bypass. The chest was open (median sternotomy), but the pleurae were in most cases closed. If the pleura was opened a drain was inserted and connected to a suction pressure of 3 cm Hg. Measurements were commenced when the end-expired carbon dioxide level had been constant for 10–15 minutes. The lungs were then hyperinflated three times in the manner described above and the Douglas bag was washed out twice with 20–30 litres of expired gas. The sampling tubes to the oxygen and carbon dioxide analyzers were disconnected and expired gas was collected for a period of 3–5 minutes. An arterial sample from the radial artery line was taken into a heparinized 5-ml plastic syringe during the middle 2–3 minutes of this period and, in eight patients post-perfusion, a simultaneous sample was obtained from a catheter in the pulmonary artery. A sample was also taken into a heparinized tube for hemoglobin estimation. At the conclusion of the gas collection the inspired oxygen concentration and end-tidal carbon dioxide concentration were again recorded. A new inspiratory: expiratory ratio was then selected. After ventilatory stability had been achieved, the lungs were hyperinflated and the measurements repeated.

The expired gas was collected in a plastic Douglas bag, and the contents were thoroughly mixed before withdrawing samples into 100-ml oiled glass and metal syringes with metal three-way taps soldered to their nozzles. The gas syringes were washed out with 100 ml of expired gas and the second 100 ml was retained for analysis. All gas analyses were performed on three gas syringes. The expired carbon dioxide concentration was determined by a Severinghaus carbon dioxide electrode and the expired oxygen concentration was determined on the paramagnetic oxygen analyzer and checked on the oxygen electrode. The expired gas volume was measured by passing the gas slowly through a previously calibrated dry gas meter and the temperature of the expired gas was noted. The patient's oesophageal temperature and the barometric pressure were also recorded.

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1 British Oxygen Gases Ltd., Great West Road, Brentford, Middlesex.
2 Model OA 101 Mk.II, Servomex Controls Ltd., Crowborough, Sussex.
4 Plysu Products Industrial Ltd., Woburn Sands, Bletchley.
5 National Welding Co., Richmond, California, U.S.A.
6 Nordgas Research model, Cape Engineering & Capecraft Ltd., Warwick.
Blood samples were analyzed for $P_{O_2}$, $P_{CO_2}$ and pH using a Radiometer E5046 oxygen electrode, the Severinghaus carbon dioxide electrode and a Radiometer capillary glass electrode standardized against NBS buffers. The output from the oxygen and pH electrodes was read on a Radiometer pH 27 meter and the output from the carbon dioxide electrode was read on an EIL Vibron electrometer, model C 33B.

The paramagnetic oxygen analyzer was checked for linearity against a Haldane gas analysis apparatus and found to be accurate to within 0.1 per cent oxygen.

The oxygen electrode was calibrated with white spot nitrogen, air and oxygen, and the accuracy of the blood-gas estimations was checked during the studies by equilibrating blood samples in a tonometer with gases containing 8.8–21 per cent oxygen. The standard deviation of the differences between the known gas and the measured blood-gas tension was 1.04 mm Hg. After measuring the oxygen tension on the electrode a blood-gas factor of 1.04 was applied (Adams and Morgan-Hughes, 1967).

The carbon dioxide electrode was calibrated with carbon dioxide-oxygen gas mixtures which had been analyzed on a Haldane apparatus. The mean error of the electrode as estimated with tonometered blood samples was +0.044 mm Hg and the 95 per cent confidence limits ±4 per cent of the reading.

The accuracy of blood pH determination was checked by comparison of the same blood sample on two different electrode systems (Adams, Morgan-Hughes and Sykes, 1967, 1968). When pH was determined twelve times in succession on the same blood sample, the standard deviation was 0.0023 pH units (mean pH 7.394). Additional checks on $P_{CO_2}$ and pH were obtained by plotting the results on the nomogram of Siggaard-Andersen (1962).

The standard deviation of twelve estimations of haemoglobin on the same blood sample (mean 15.4 g/100 ml) was 0.27 g/100 ml. Temperature corrections (Kelman and Nunn, 1966) were applied to the blood-gas tensions measured at 37°C.

Calculations.

All calculations were performed on an Elliot 4100 computer, utilizing a programme written in this department (Adams, in preparation). Physiological deadspace was calculated from the Enghoff (1938) modification of the Bohr equation:

$$V_{Dphys} = V_T[(P_{CO_2} - P_{E CO_2})/P_{CO_2}] - V_{Dapp}$$

where $V_{Dphys}$ = physiological deadspace; $V_T$ = tidal volume; $V_{Dapp}$ = apparatus deadspace; $P_{CO_2}$ = arterial carbon dioxide tension; $P_{E CO_2}$ = mixed expired carbon dioxide tension.

Alveolar oxygen tension was derived from the alveolar air equation (Nunn, 1963):

$$P_{A O_2} = P_{T O_2} - P_{CO_2}([(P_{T O_2} - P_{E O_2})/P_{CO_2}]$$

where $P_{A O_2}$ = alveolar oxygen tension; $P_{T O_2}$ = inspired oxygen tension; $P_{CO_2}$ = arterial carbon dioxide tension; $P_{E O_2}$ = mixed expired carbon dioxide tension; $P_{E O_2}$ = mixed expired oxygen tension.

The venous admixture effect was calculated from the formula:

$$(Qs/Qt)\% = \left[\left(C_{c'O_2} - C_{ao_2}\right)/\left(C_{c'O_2} - C_{vo_2}\right)\right] \times 100$$

where Qs/Qt = venous admixture expressed as a percentage of the cardiac output;

$C_{ao_2}$ = arterial oxygen content (vol%);

$C_{c'O_2}$ = end-pulmonary capillary oxygen content (vol%);

$C_{vo_2}$ = mixed venous oxygen content (vol%).

The solubility coefficient for dissolved oxygen was taken as 0.003 vol%/mm Hg (Finley et al., 1960). Oxygen capacity was taken as Hb(g) x 1.39 vol% (International Committee for Standardization in Haematology, 1965). When calculating $C_{ao_2}$ the arterial oxygen tension ($P_{ao_2}$) and pH were used to read the percentage oxygen saturation ($S_{ao_2}$) from the oxygen dissociation curve of Severinghaus (1966). Then

$$C_{ao_2} = (\text{capacity} \times [S_{ao_2}/100]) + (P_{ao_2} \times 0.003)$$

V. & A. Howe, 48 Pembridge Road, London W.11.

Electronic Instruments Ltd., Richmond, Surrey.

Elliot Automation Ltd., Portland Place, London W.1.
When calculating $C_{O_2}'$, the same formula was applied, but it was assumed that the end-pulmonary capillary blood had the same $P_{O_2}$ as the calculated alveolar oxygen tension. Thus any A–e' oxygen tension gradient was included in the calculated venous admixture effect.

When pulmonary artery blood samples could not be obtained, an arteriovenous oxygen content difference of 5 vol% was assumed (i.e. $C_{O_2} = C_{a_o} - 5$ vol% )

**RESULTS**

The means and standard deviation for each I:E time ratio studied pre- and post-perfusion are shown in table I.¹⁰

**VD/VT ratios** (fig. 2).

In the pre-perfusion period the VD/VT ratio was higher during ventilation with the 0.5:2.5 ratio than with the 1.5:1.5 ratio, although there was no significant difference on statistical analysis. Post-perfusion there was a significant reduction in VD/VT during ventilation with the 1.5:1.5 ratio as compared with the other two I:E ratios (P<0.05 in both comparisons), but there was no significant difference between the 1:2 and 0.5:2.5 ratios.

**A–aPo₂** (fig. 3).

There was no significant difference between the two ratios used in the pre-perfusion studies. However, there was a significant increase in the A–aPo₂ between the pre- and post-perfusion

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¹⁰ Full details of the measured and calculated data from individual patients can be obtained from M.K.S.

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<th>I:E time ratio</th>
<th>VT (ml)</th>
<th>$P_{a_o}$ (mm Hg)</th>
<th>$V_{co_2}$ (ml STPD)</th>
<th>VD/VT (%)</th>
<th>$P_{l_o}$ (mm Hg)</th>
<th>$P_{a_o}$ (mm Hg)</th>
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<th>A–aPo₂ (mm Hg)</th>
<th>Qs/Qt (%)</th>
<th>A–V difference (vol%)</th>
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* Assumed value.
values with I:E ratios of 0.5:2.5 and 1.5:1.5 (P<0.005). There was no significant difference between the three ratios in the post-perfusion period.

**DISCUSSION**

**VD/VT ratio.**

These studies confirm the findings of Watson (1962a), Fairley and Blenkarn (1966) and Bergman (1967) that VD/VT ratio is increased when the duration of inspiration is reduced. Such an increase could be due to an increase in anatomical deadspace or to an increase in alveolar deadspace.

The volume of the anatomical deadspace depends on the transpulmonary pressure at the end of inspiration (Shepard et al., 1957). This, in turn, depends on lung compliance and tidal volume. If tidal volume is constant, transpulmonary pressure will only increase if lung compliance falls. Dynamic compliance was not measured in the present studies but from the changes in end-inspiratory pressure recorded it may be inferred that there was a reduction in dynamic compliance during inflation with the 0.5 second inspiratory time (table I).

A reduction in dynamic compliance with short inspiratory times has also been noted by Watson (1962b): in one patient he found that dynamic compliance was reduced from about 50 ml/cm H$_2$O to 35 ml/cm H$_2$O when the duration of inspiration was reduced from 1.0 to 0.5 second. With a constant tidal volume of 500 ml this would increase inflation pressure from 10 to 14.3 cm H$_2$O. From the data on one subject provided by Shepard and associates (1957) it may be calculated that the increased transmural pressure would increase anatomical deadspace by about 15 ml.$^{11}$

An increase in anatomical deadspace could therefore account for part of the change in physiological deadspace noted in these studies. It is unlikely, however, that this explanation could account for all the increase in physiological deadspace for the reduction in dynamic compliance suggests that there were changes in the distribution of gas within the lung. It is thought that the reduction in dynamic compliance is due to maldistribution of inspired gas resulting from a scatter of time-constants in the alveolar units. When the duration of inspiration is reduced, pressure equilibrium between mouth and alveoli is not achieved in alveolar units with long time-constants and alveolar units with short time-constants are consequently over-ventilated. As a result the distribution of ventilation is uneven and ventilation: perfusion inequalities are accentuated. This would cause an increase in alveolar deadspace and venous admixture effect. Such an increase in alveolar deadspace is suggested by the observations of Bergman (1963) in dogs. He found that there was an increased arterial to alveolar carbon dioxide tension difference as inspiration was shortened. However, without more detailed measurements, it is not possible to say which of the two components of physiological deadspace was more affected in these patients.

Since the time available for making measurements in the pre-perfusion period was limited it was only possible to compare all three ratios in the post-perfusion period. The results show that a reduction in Vd/Vt ratio was only achieved with an inspiratory time of 1.5 seconds. A similar pattern was seen in two of the three conscious, but paralyzed, subjects studied by Watson (1962a) and in the anaesthetized patients studied by Bergman (1967). There is therefore now ample evidence to suggest that optimal distribution of inspired gas during mechanical ventilation is only achieved when the duration of inspiration approaches 1.5 seconds. This period may be less

$^{11}$ Shepard and associates (1957) state that the volume of anatomical deadspace equals $120 - 3.46 \times (P_{ao2} - P_{paco2})$. With a transpulmonary pressure of 10 cm H$_2$O the deadspace volume is therefore 154.6 ml, whereas with a transpulmonary pressure of 14.3 cm H$_2$O the volume is 169.5 ml.
when ventilation is spontaneous but is probably greater when the lung is diseased.

**A-aPo**

In common with Fairley and Blenkarn (1966) and Bergman (1967) we found no change in A-aPo with varying durations of inspiration. However, it is noteworthy that in all these three studies the inspired oxygen concentration was 30 or 100 per cent: this would raise the alveolar oxygen tension to levels which would ensure saturation of the end-pulmonary capillary blood emanating from all but extremely underventilated or collapsed alveoli. It is therefore unlikely that small changes in the distribution of inspired gas could be detected by this form of analysis. This fact probably also explains why Bergman (1963) obtained a marked variation in A-aPo with mean airway pressure in dogs ventilated with air, whereas he did not note this change in patients ventilated with additional oxygen (Bergman, 1967).

The increase in A-aPo after perfusion is a common finding. Its aetiology is unknown but it could be associated with multiple small emboli (Jones and Goodwin, 1965), pulmonary oedema (Stein et al., 1961; Said et al., 1964), or it could represent the earliest stages of the post-perfusion lung syndrome (Fordham, 1965; Hedley-Whyte et al., 1965; McClenahan, Young and Sykes, 1965). It is known that physiological deadspace is increased for periods of up to an hour after bypass (Pauca and Sykes, 1966) and it may be that the increased A-aPo represents mainly a ventilation/perfusion abnormality due to cardiopulmonary bypass or drugs given after the procedure (Fordham and Resnekov, 1968).

It is concluded from these studies that arterial oxygenation during anaesthesia for cardiopulmonary bypass is little affected by changes in inspiratory-expiratory time ratio when the inspired oxygen concentration is increased to levels which eliminate most of the changes due to ventilation-perfusion inequalities.

The small improvement in the efficiency of ventilation (as judged by the reduction in Vd/Vt ratio with the 1.5:1.5 inspiratory-expiratory time ratio) scarcely justifies the use of a ventilator with a variable duration of inspiration, particularly as the cardiac output may be impaired by the relatively short time allowed for cardiac filling when this ratio is used. Whether such an alteration in I:E ratio would become more important in patients with more serious lung disease has yet to be determined.

**ACKNOWLEDGEMENTS**

We wish to thank Miss B. Bird for technical assistance, Miss R. Boorne and Miss S. Jenkins for secretarial assistance, and the Department of Medical Illustration.

**REFERENCES**


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**FIFTH INTERNATIONAL CONGRESS ON ANAESTHESIOLOGY**

The Congress will take place in Kyoto, Japan, from October 2 to 8, 1972. The Belgian Professional Association of Specialists in Anaesthesia and Reanimation is to organize a three-weeks group tour from Brussels to the Far East, open to all anaesthetists of Western Europe and their families. The journey can thus be accomplished on the most advantageous terms. Booking is done on guaranteed periodical payments in advance.

For further particulars apply to

Dr. Et. Troch, Marcel de Backerstraat 2, Ekeren 2 (Antwerp), Belgium.

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**BRITISH JOURNAL OF ANAESTHESIA**

L’EFFET DE DIVERS RAPPORTS INSPIRATION: EXPIRATION SUR L’ÉCHANGE DE GAZ LORS DE L’ANESTHESIE POUR LA CHIRURGIE A COEUR OUVERT

**SUMMARY**

Chez des malades avec ventilation mécanique lors d’une opération à cœur ouvert, on a mesuré le rapport espace mort physiologique/volume courant (V<sub>D</sub>/V<sub>T</sub>)% et la différence de pression d’oxygène alvéolaire artérielle (A-aPo<sub>2</sub>). Les rapports temps d’inspiration: expiration étudiés étaient 0.5:2.5 et 1.5:1.5 avant la perfusion et 0.5:2.5, 1:2 et 1.5:1.5 après la perfusion. Il n’y avait donc pas de différence significative du V<sub>D</sub>/V<sub>T</sub>% et A-aPo<sub>2</sub> avant la perfusion entre les deux types de ventilation. Le A-aPo<sub>2</sub> augmentait après perfusion. Quoiqu’on ne notait pas de différence significative du A-aPo<sub>2</sub> entre les trois rapports étudiés après perfusion, le V<sub>D</sub>/V<sub>T</sub>% était significativement moins grand pour un temps d’inspiration de 1.5 secondes que de 0.5 et 1 seconde.

**ZUSAMMENFASSUNG**

Bei Patienten mit offenen Herzoperationen wurden während mechanischer Beatmung Messungen des physiologischen Verhältnisses zwischen Totraum und Atmungsvolumen (V<sub>D</sub>/V<sub>T</sub>%) sowie der Differenz von arteriellem und alveolärem Sauerstoffsdruck (A-aPo<sub>2</sub>) durchgeführt. Die untersuchten Zeitverhältnisse zwischen Aus- und Einatmung betrugen 0,5:2,5 und 1,5:1,5 vor Perfusion und 0,5:2,5, 1:2 und 1,5:1,5 nach Perfusion. Vor der Perfusion ergaben sich keine signifikanten Unterschiede in V<sub>D</sub>/V<sub>T</sub>% oder A-aPo<sub>2</sub> zwischen den beiden Beatmungsschemata. Nach der Perfusion stieg A-aPo<sub>2</sub> an. Obwohl zwischen keinem der drei Verhältnisse, die nach Perfusion geprüft wurden, signifikante Unterschiede in A-aPo<sub>2</sub> gefunden wurden, war doch bei einer Einatmungszeit von 1,5 Sekunden V<sub>D</sub>/V<sub>T</sub>% signifikant kleiner als bei 0,5 oder 1,0 Sekunden.