OXYGEN BREATHING AND $\dot{Q}s/\dot{Q}T$ DURING POSTOPERATIVE PAIN RELIEF IN MAN

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POSTOPERATIVE HYPOXAEMIA following anaesthesia has been attributed mainly to two causes; venous admixture (shunt component) and variation in ventilation-perfusion ratios (distribution component). In order to assess quantitatively the role played by the latter factor, the method of breathing 100 per cent oxygen has been employed by many investigators.[1–5] This method is based on the fact that the diffusion and distribution components become negligible and $AaDo_2$ can then result only from direct venous admixture, either through normally existing channels or through direct pulmonary arterio-venous shunts, or perfusion of alveoli which cannot receive any of the inspired oxygen because they are atelectatic or completely occluded by exudate or thickened walls.

Griffo[6] reported that breathing pure oxygen at atmospheric pressure for several hours does not enhance the development of pulmonary atelectasis in normal subjects. Several workers,[7–9] however, have postulated that oxygen breathing of relatively short duration might lead to the development of diffuse pulmonary atelectasis. Such atelectasis has been suggested to result from the absorption of trapped gas distal to temporarily occluded airways.

The main purpose of the present study was to seek further evidence for or against these views by extending the observations to patients breathing air and 100 per cent oxygen during postoperative pain relief with meperidine or epidural analgesia and to determine the possibility of difference of the increase in $\dot{Q}s/\dot{Q}T$ between the epidural and meperidine analgesia groups.

METHODS

Thirteen geriatric patients of both sexes without known cardio-pulmonary disease and scheduled for upper abdominal surgery (cholecystectomy for cholelithiasis, gastrectomy for peptic ulcer except one hemicolectomy and one splenectomy) were chosen for this study. They were divided into two groups, the first consisting of 7 patients (5 males and two females) and the second, 6 patients (4 males and two females). Average values for age, weight and body surface area are shown in Table I.

Premedication consisted of atropine 0.5 mg subcutaneously, one hour prior to induction of anaesthesia. No narcotic drug was given. Anaesthesia was induced with thiamylal, followed by succinylcholine 40 mg to facilitate endotracheal intu-

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TABLE I

AVERAGE VALUES FOR AGE, WEIGHT, BODY SURFACE, DURATION OF OPERATION, MEASUREMENT TIME AFTER THE END OF OPERATION AND TIME FOR OXYGEN INHALATION FOR GROUP 1 AND GROUP 2

	Patients					Duration of Operation (min.)	First Series of Measurements Time (min.) after the End of Operation	Second Series of Measurements Time (min.) after the End of Operation	Duration of O_2 Inhalation (min.)	
Group	n	Males	Age (Years)	Weight (Kg)	Body Surface (m^2)				1st Series	2nd Series
group 1 Mean (± S.E.)	7	5	63.3 (1.6)	49.5 (4.8)	1.47 (0.07)	189 (17.3)	388 (44.6)	1121 (51.9)	19.7 (1.13)	18.0 (2.43)
group 2 Mean (± S.E.)	6	4	66.8 (5.5)	42.8 (3.2)	1.38 (0.05)	205 (21.4)	332 (57.8)	1051 (62.4)	20.5 (3.15)	17.9 (1.01)
Intergroup Difference										
P			NS	NS	NS	NS	NS	NS	NS	NS

Group 1: Subjected to epidural analgesia.
Gruop 2: Subjected to meperidine analgesia
NS = $P > 0.05$.

bation with a cuffed tube. Anaesthesia was maintained with nitrous oxide and halothane, supplemented with succinylcholine as needed for muscle relaxation. After recovery from anaesthesia, no special treatments other than routine postoperative measures were instituted. All patients lay supine and breathed room air.

In the first group, a polyethylene catheter introduced into the epidural space at the eighth or ninth thoracic intervertebral space prior to induction of general anaesthesia was retained in place for postoperative analgesia with mepivacaine or lidocaine 1 per cent solution. In the second group, meperidine was administered intravenously in intermittent doses of 10 mg until almost complete analgesia was obtained. Patients in group 1 were allowed to rest quietly in the recovery room for an average of 388 ± 44.6 (S.E.) minutes and those in group 2 rested 332 ± 57.8 minutes before observations were begun (Table I). A set of control measurements was then made under the conditions of sufficient analgesia, followed by oxygen inhalation through a mouthpiece and non-rebreathing valve system. Patients in group 1 breathed oxygen for 19.7 ± 1.13 minutes and those in group 2, for 20.5 ± 3.15 minutes before the other set of measurements was made (the first series of measurements). The second series of measurements were performed 1121 ± 51.9 minutes in group 1 and 1051 ± 62.4 minutes in group 2 postoperatively and consisted of the same procedures as those of the first series of measurements. Each set of measurements consisted of arterial blood pressure, heart rate, body temperature (rectal), expiratory volume, respiratory rate, carbon dioxide fraction of expired and end-expired gases, arterial blood gases, cardiac output, and oxygen uptake.

Details of the procedures and measurements as well as the errors in calculated pulmonary shunting coming from separate measurements of cardiac output and oxygen uptake have been reported and discussed in a previous paper.[10]

Calculations

Alveolar oxygen tension (P_{AO_2}) during breathing of air was estimated by applying the alveolar air equation.[11]

$$P_{AO_2} = P_{IO_2} - P_{ACO_2}\,[F_{IO_2} + (1 - F_{IO_2})/R]$$

R was measured and F_{IO_2} equalled 0.21. P_{ACO_2} was assumed to be equal to P_{aCO_2}. P_{AO_2} during breathing of oxygen was calculated as $P_{AO_2} = P_B - P_{H_2O} - P_{ACO_2}$ assuming P_{ACO_2} equalled P_{aCO_2}.

Physiologic shunt was calculated from the following equation during breathing of air: $\dot{Q}s/\dot{Q}T = (C\acute{c}o_2 - Cao_2)/(C\acute{c}o_2 - C\bar{v}o_2)$ and during breathing of oxygen:

$$\dot{Q}s/\dot{Q}T = 0.0031\ AaDo_2/[(Ca - C\bar{v}) + 0.0031\ AaDo_2]$$

Arterial to mixed venous oxygen content difference ($Ca - C\bar{v}$) was calculated from the formula:[12] ($Ca - C\bar{v}$) (vol. %) = 100 $\dot{V}o_2$/C.O. (both L/min) where $\dot{V}o_2$ = oxygen uptake and C.O. = cardiac output. Oxygen content in arterial blood (Cao_2) during breathing of air was measured by Van-Slyke Neill apparatus and that during breathing of oxygen was calculated from the following equation:

$$Cao_2 = 1.34\ Hb + 0.0031\ Pao_2$$

End-pulmonary capillary oxygen content ($C\acute{c}o_2$) during breathing air was calculated from the following equation:

$$C\acute{c}o_2 = So_2^{P_{AO_2}} \times 1.34\,Hb + 0.0031\,P_{AO_2}$$

and end-pulmonary capillary blood was assumed to have the same P_{O_2} as the calculated alveolar oxygen tension (P_{AO_2}). Oxygen saturation ($So_2^{P_{AO_2}}$) was calculated from the nomogram of Severinghaus[13] after correcting arterial oxygen tension for pH.

Results

Detailed data of the following parameters: blood pressure (B.P.), heart rate (H.R.), respiratory rate (f), minute volume (M.V.), expiratory volume (V_E), oxygen uptake ($\dot{V}o_2$), respiratory gas exchange ratio (R), respiratory dead space to tidal volume ratio (V_D/V_T), arterial carbon dioxide tension ($Paco_2$), arterial to end-tidal carbon dioxide tension difference ($(a - ET)\,Dco_2$), $\dot{Q}s/\dot{Q}_T$, pH and cardiac output (C.O.) in the epidural group and meperidine group were presented in Tables II and III.

Almost all the parameters exhibited no significant changes, however, $\dot{Q}s/\dot{Q}_T$ in the epidural group invariably exhibited a considerable increase, from a mean control value of 6.71 ± 1.17 to 10.85 ± 1.27 ($P < 0.001$) in the first series of measurements and from 7.87 ± 1.10 to 13.25 ± 0.98 ($P < 0.01$) in the second series of measurements, a 61.7 per cent increase in the first and 68.4 per cent increase in the second respectively. In the meperidine group, $\dot{Q}s/\dot{Q}_T$ increased from 7.53 ± 2.42 to 9.27 ± 1.05 per cent in the first series of measurements and from 7.37 ± 2.45 to 12.17 ± 2.43 per cent in the second series of measurements although these increases did not reach to the significant level. These changes in $\dot{Q}s/\dot{Q}_T$ were also shown graphically in Figure 1.

Discussion

Some investigators[14,15,6] have indicated that the magnitude of true shunt was not altered by breathing different oxygen concentrations; in other words, no significant collapse of alveoli occurred by denitrogenation.[16] This assumption became the theoretical basis to distinguish the distribution factor from total shunt by oxygen breathing.[3,5] The reasoning was that the existence of uneven distribution of ventilation to perfusion in the lung results in a minimal venous admixture provided the direct venous admixture contribution is constant at various oxygen tensions, and that the effect of diffusion barriers could be neglected under the condition of high oxygen concentrations.

Déry[17] *et al.* reported, however, an average decrease in functional residual capacity (FRC) during the first 30 minutes of oxygen breathing accompanied by decreasing Pao_2. These results are compatible with our data in which consistent rise in $\dot{Q}s/\dot{Q}_T$ was observed during oxygen breathing. These increases in $\dot{Q}s/\dot{Q}_T$ were observed in the long term study (second series of measurements) as well as just after the end of operation (first series of measurements). In analyzing the data obtained, the significant level of these changes was higher in the epidural group than in the meperidine group though the reverse was expected.[18]

TABLE II

VALUES FOR RESPIRATORY RATE, EXPIRATORY VOLUME, OXYGEN UPTAKE, PHYSIOLOGIC DEAD SPACE, V_D/V_T, ARTERIAL CARBON-DIOXIDE TENSION, ARTERIAL TO END-TIDAL P_{CO_2} DIFFERENCE, $\dot{Q}_S/\dot{Q}_T$ AND CARDIAC OUTPUT BEFORE AND DURING OXYGEN BREATHING (GROUP 1)

		No. of Observations	f (bpm)	M.V. (ml) (STPD)	V_E (ml) (STPD)	$\dot{V}_{O_2}$ ml/min /BSA (STPD)	V_D (Ml) (STPD)	V_D/V_T	P_{ACO_2} (torr)	(a-ET) D_{CO_2} (torr)	$\dot{Q}_S/\dot{Q}_T$ %	C.I. L/min/ BSA
The First Series of Measurement	Control (During Analgesia (Mean ± SE)	7	20.9 (1.0)	8213 (621)	394 (26)	174 (11.2)	169 (19.2)	0.43 (0.04)	34.3 (0.98)	3.82 (0.88)	6.71 (1.17)	2.66 (0.25)
	During Oxygen Inhalation (Mean ± SE)	7	22.5 (1.4)	9015 (1014)	406 (48)	168 (9.0)	187 (29.2)	0.45 (0.03)	34.2 (1.17)	4.98 (0.73)	10.85 (1.27)	2.60 (0.21)
	Mean of Differences (± SE)		1.6 (0.8)	802 (629)	12.1 (25.3)	−6.3 (5.6)	18.4 (19.2)	0.03 (0.02)	−0.06 (0.85)	1.16 (1.18)	4.14 (0.50)	−0.06 (0.22)
	P		N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	<0.001	N.S.
The Second Series of Measurement	Control (During Analgesia)	7	19.6 (1.1)	8221 (417)	431 (42)	158 (12.8)	200 (22.6)	0.47 (0.03)	34.9 (1.23)	3.72 (0.89)	7.87 (1.10)	2.64 (0.14)
	During Oxygen Inhalation	7	20.8 (1.0)	8556 (577)	423 (45)	156 (6.0)	217 (0.04)	0.50 (1.44)	33.3 (1.41)	4.00 (1.41)	13.25 (0.98)	1.72 (0.15)
	Mean of Differences		1.1 (0.7)	335 (303)	−7.9 (17.6)	−2.1 (10.7)	16.3 (18.7)	0.03 (0.04)	−1.54 (0.70)	0.29 (1.10)	5.38 (1.36)	0.08 (0.10)
	P		N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	<0.01	N.S.

Values for B.P., H.R., R, pH, P_{aO_2} and AaD_{O_2} were omitted from the table. N.S. = $P > 0.05$.

TABLE III

VALUES FOR RESPIRATORY RATE, EXPIRATORY VOLUME, OXYGEN UPTAKE, PHYSIOLOGIC DEAD SPACE, V_D/V_T, ARTERIAL CARBON-DIOXIDE TENSION, ARTERIAL TO END-TIDAL P_{CO_2} DIFFERENCE, $\dot{Q}_S/\dot{Q}_T$ AND CARDIAC OUTPUT BEFORE AND DURING OXYGEN BREATHING (GROUP 2)

		No. of Observations	f (bpm)	M.V. (ml) STPD)	V_E (ml) (STPD)	$\dot{V}_{O_2}$ ml/min /BSA (STPD)	V_D (ml) (STPD)	V_D/V_T	P_{aco_2} (torr)	(a-ET) D_{co_2} (torr)	$\dot{Q}_S/\dot{Q}_T$ %	C.I. L/min /BSA
The First Series of Measurement	Control (During Analgesia (Mean ± SE)	6	16.8 (1.2)	6347 (487)	379 (22)	140 (11.9)	200 (18.2)	0.52 (0.03)	40.1 (2.47)	6.24 (1.63)	7.53 (2.42)	2.54 (0.30)
	During Oxygen Inhalation	6	17.3 (1.4)	6668 (539)	383 (21)	148 (8.5)	190 (14.0)	0.50 (0.02)	38.6 (1.78)	6.62 (1.27)	9.27 (1.05)	2.36 (0.27)
	Mean of Differences		0.5 (0.3)	322 (237)	3.7 (9.9)	7.8 (6.2)	−9.5 (5.4)	−0.03 (0.01)	−1.52 (1.25)	0.38 (2.00)	1.74 (2.02)	−0.18 (0.11)
	P		N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
The Second Series of Measurement	Control (During Analgesia)	6	16.9 (1.5)	6090 (529)	366 (27)	140 (13.2)	175 (15.9)	0.47 (0.02)	39.1 (1.39)	4.13 (1.04)	7.37 (2.45)	3.07 (0.57)
	During Oxygen Inhalation	6	18.0 (1.7)	6820 (780)	381 (33)	148 (11.9)	201 (29.9)	0.52 (0.04)	39.3 (2.58)	5.51 (1.28)	12.17 (2.43)	3.44 (0.92)
	Mean of Differences		1.1 (0.5)	730 (360)	14.7 (16.3)	7.8 (5.7)	26.3 (24.8)	0.04 (0.05)	0.23 (1.51)	1.37 (1.23)	4.80 (2.61)	0.37 (0.37)
	P		N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.

Values for B.P., H.R., R, pH, P_{aO_2} and $AaDo_2$ were omitted from the table. N.S. = $P > 0.05$.

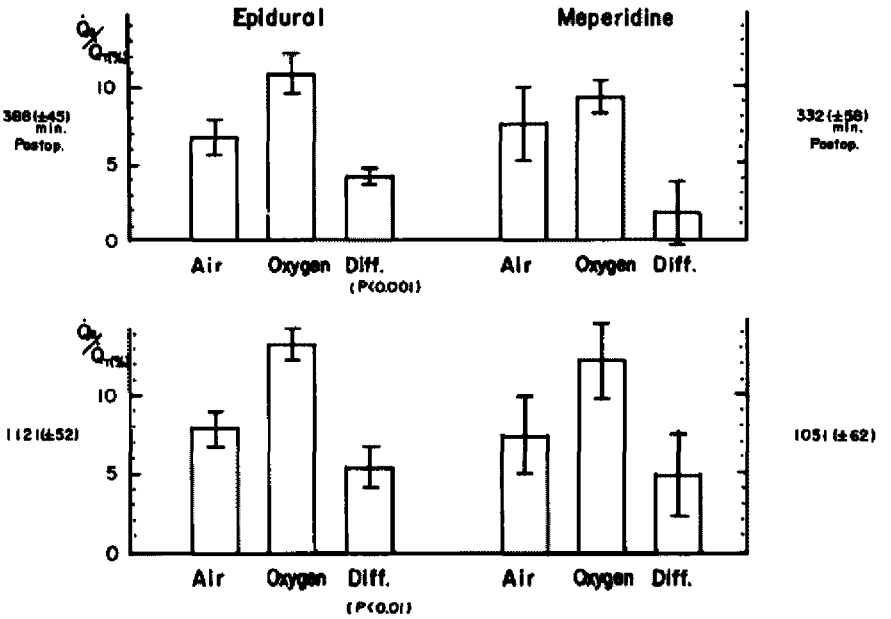

FIGURE 1. Calculated venous admixture during air and oxygen breathing and their differences in the epidural and meperidine groups both at the first (upper) and the second series of measurement (lower graph).

The reason for these changes was suggested to be attributable to the occurrence of atelectatic alveoli induced by oxygen breathing being facilitated by absence of the relatively insoluble alveolar nitrogen.[8] These changes were also suggested by Bendixen[9] in anaesthetized man. The question arises whether pure oxygen promotes development of atelectasis. It has been believed that atelectasis is most likely to occur in low $\dot{V}_A/\dot{Q}$ alveoli where the rate of O_2 uptake may exceed the flow of oxygen conveyed by ventilation.[8]

A redistribution of pulmonary blood flow is a further possibility. In order to explain an increase in shunt on this basis one would have to assume a redistribution favouring the shunt-producing gas exchange units; in other words, pulmonary vasodilatation of the hypoxic area. Although not likely to account for the entire change, this can neither be proved nor disproved.[5]

A third possible mechanism of increase in shunting might be expected from anatomical shunt during oxygen breathing. This seems most unlikely, however, in view of the fact that such anatomical shunt is less than one or two per cent of cardiac output in normal subjects and it has usually been assumed that the magnitude of the anatomical shunt does not vary with the alveolar oxygen tension, though the validity of this conclusion has never been established.[16]

In this study, oxygen breathing was carried out for about 20 minutes, which probably resulted in an incomplete nitrogen washout from hypoventilated alveoli. While the time for 90 per cent washout is one to two minutes for normal lung, in the lung units having $\dot{V}_A/\dot{Q}$ ratio of 0.1, the 90 per cent washout time is 5

minutes and at a ratio of 0.01, is 35 minutes.[8] Provided that the 98 per cent wash-out time for nitrogen is assumed to be 20 minutes for the postoperative patients, the actual alveolar oxygen tension would be 11 torr lower than that calculated from the alveolar air equation. This indicates that the actual $AaDo_2$ could be 11 torr smaller than that calculated above. If the error in $AaDo_2$ is 10 torr, the error in $\dot{Q}s/\dot{Q}T$ will be about -0.4 ± 0.03 (S.E.) per cent, calculated from the data of our patients. The size of this error is sufficiently small compared with changes in $\dot{Q}s/\dot{Q}T$ associated with inhalation of pure oxygen.

The procedures used in this study for calculating oxygen content in mixed venous blood result in some errors in $\dot{Q}s/\dot{Q}T$. The effects of these calculations on the accuracy of the results and the reliability of the methods were discussed in our previous paper.[10]

In conclusion, the oxygen breathing method to distinguish distribution component from true shunt (atelectasis) is difficult and erroneous during particular conditions such as the postoperative period.

RÉSUMÉ

L'hypoxémie post opératoire à la suite de l'anesthésie a été surtout attribuée à l'atélectasie ou aux anomalies ventilateur perfuseur au niveau du poumon. Pour distinguer les anomalies ventilateur perfuseur de l'atélectasie, nous avons donné à respirer de l'oxygène pur à des malades durant la période post-opératoire après une sédation adéquate avec de la meperidine ou une analgésie épidurale, le shunt pulmonaire a été mesuré avant et après inhalation de l'oxygène.

Après abolition de la douleur avec l'une ou l'autre méthode, le shunt Qs/QT a augmenté de façon significative lors de l'exposition à l'oxygène pur si on le compare à la respiration à l'air libre. Il n'y a pas eu de différence entre le groupe traité avec la mépéridine ou l'analgésie épidurale. D'après ces résultats, nous pouvons conclure que la différenciation entre les anomalies ventilateur perfuseur et l'atélectasie par la méthode de l'inhalation d'oxygène pur n'est pas possible dans tous les cas à cause de l'affaissement alvéolaire qui se développe par denitrogenation dans ces circonstances, de sorte que l'effet négatif de l'atélectasie miliaire peut être plus grand que l'effet positif de l'abolition des inégalités ventilateur perfuseur créée par l'inhalation d'oxygène pur. Nous avons aussi passé en revue les autres facteurs susceptibles d'augmenter le shunt durant l'inhalation d'oxygène pur.

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