PULMONARY SHUNTING DURING ANAESTHESIA IN MAN

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THE LITERATURE is replete with measurements of pulmonary shunting ($\dot{Q}_s/\dot{Q}_t$ with the $F_{I_{O_2}} = 1$) during anaesthesia.¹ In our laboratory we have found a mean shunt of 3.4 per cent in 43 anaesthetized healthy beagle puppies.²⁻⁴ Others have reported similar minimal shunting under anaesthesia in the dog.⁵⁻⁸ However, in the dog there is evidence of collateral pulmonary ventilation⁹ which would enhance pulmonary gas exchange and might attenuate the development of a large shunt. In man, almost all studies during anaesthesia demonstrate a markedly increased $\dot{Q}_s/\dot{Q}_t$. In surgical patients Marshall, *et al.*¹⁰ showed a 4.4 per cent shunt prior to anaesthesia and a 12.8 per cent shunt after induction suggesting that in man the anaesthetic state *per se* was responsible for abnormal shunting. However, more recently, Marshall, *et al.*¹¹ examined six healthy volunteers in their early twenties and reported that during anaesthesia the shunt fraction was less than 4 per cent. We have studied anaesthetized patients during anaesthesia and operation to determine whether in fact atelectasis with its consequent increase in shunting does occur in all who undergo general anaesthesia.

METHODS

$\dot{Q}_s/\dot{Q}_t$ was measured in 22 patients undergoing mostly major neurosurgical operations. Age varied between 23 and 61 years. All possessed good physique and were active until admission; however, thereafter some were sedated and on bed rest for days preoperatively. Some smoked cigarettes, but none gave a history or had physical findings indicative of pulmonary disease. Routine chest X-rays were all reported as normal. Extensive preoperative testing of pulmonary function was not attempted.

Halothane (0.5–2.0 per cent) and oxygen were the sole inhalational agents used for anaesthesia, and ventilation was controlled without positive end-expiratory pressure or resistance. The depth of anaesthesia, the degree of muscle relaxation and the P_{aCO_2} differed among patients, as premedication, induction and the course of anaesthesia were managed by anaesthetists apart from the study. Indwelling radial artery and right ventricular monitoring lines were inserted following induction and so preanaesthetic control measurement could not be obtained. Two shunt determinations were made during the course of operation, separated by as large

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an interval as was feasible. An attempt was made to sample only during periods when stable anaesthetic conditions had prevailed for at least 30 minutes, and similar anaesthetic techniques were employed. Blood pressure, heart rate, body temperature and in 12 patients dye-dilution cardiac output measurements were recorded at the time of blood gas sampling.

Arterial and mixed venous blood samples were drawn anaerobically into iced heparinized sterile glass syringes and analyzed expeditiously for P_{O_2} , P_{CO_2} and pH on an IL 313 analyzer. When a delay of more than ten minutes occurred between sampling and blood-gas analysis, a correction factor was employed to compensate for oxygen decay in the blood.⁴ A second adjustment was made upon the $P_{a_{O_2}}$ to compensate for the oxygen electrode-blood gas P_{O_2} difference.⁴ One pH and one CO_2 electrode sufficed for both arterial and venous blood, but two oxygen electrodes were used so that the high oxygen calibrating gas closely corresponded to the P_{O_2} of each sample. All determinations were done in duplicate. The temperature of the water bath was $37^\circ C$, but patient body temperature usually was not and so all blood gases were corrected for temperature.¹² The shunt fraction was calculated using the following formula:

$$\dot{Q}_s/\dot{Q}_t = (C\dot{c}_{O_2} - C\dot{a}_{O_2}) / (C\dot{c}_{O_2} - C\bar{v}_{O_2})$$

where $C_{O_2} = (1.39 \times \text{Hb} \times \text{per cent saturation}) + (\alpha \times P_{O_2})$, and $\dot{c}$, $\dot{a}$, and $\bar{v}$ represent end pulmonary capillary, arterial and mixed venous values, respectively. Oxygen content was therefore calculated and oxygen saturation derived from measured blood gases using the data of Severinghaus¹² and Roughton and Severinghaus.¹³ Oxygen solubility in blood (α) was corrected for temperature and haemoglobin,¹⁴ but P_{50} was not measured.

The Students t-test was used to determine statistical significance.

RESULTS

Data from individual patients are presented in Table I. They indicate the number of hours following induction when each determination of $\dot{Q}_s/\dot{Q}_t$ was made as well as the cardiovascular and pulmonary parameters measured simultaneously. For most patients the interval between was substantial, and the clinical conditions similar.

Pontoppidan, *et al.* stated that "In normal man at rest, $\dot{Q}_s/\dot{Q}_t$ varies from 2 to 5 per cent of the cardiac output."¹⁵ Our anaesthetized patients fell into two distinct groups; those whose shunt fractions were roughly in the normal range, and those that were markedly abnormal. The first 15 patients listed in Table I exhibited minimal pulmonary shunting with a mean of 3.9 ± 0.3 per cent (S.E.M.). The average age of these 15 patients was 37 years, ten years younger than those who had increased shunting and significantly younger as determined by variance analysis ($F = 4.6$, $P < 0.02$). Age correlated fairly well with $\dot{Q}_s/\dot{Q}_t$; the correlation coefficient was 0.57 ($P < 0.01$).

Shunt was unaltered by the passage of time under anaesthesia. Mean $\dot{Q}_s/\dot{Q}_t$ in the early period was 7.0 ± 1.1 per cent and in the later period, 6.9 ± 1.2 per cent (an insignificant difference). When examined separately, those with minimal

TABLE I

Surgical lesion	Age (years)	Early Period					Late Period						
		Hours after induction	MAP (torr)	HR (beats/min)	Qt (L/min)	Qs/Qt (%)	Paco ₂ (torr)	Hours after induction	MAP (torr)	HR (beats/min)	Qt (L/min)	Qs/Qt (%)	Paco ₂ (torr)
Herniated disc	23	1	74	79	3.2	1.9	29.8	5	88	68	2.5	2.3	30.2
Herniated disc	26	1	65	60	—	2.3	28.2	2	76	61	—	2.7	28.3
Acoustic neuroma	27	3	83	120	—	2.1	30.6	6	83	110	3.4	3.0	36.8
Cerebral aneurysm	31	2	70	95	4.2	3.1	18.3	8	60	95	3.4	1.5	24.7
Herniated disc	35	2	78	68	2.5	2.5	19.2	4	70	58	2.4	2.9	17.2
Cerebral aneurysm	54	4	50	106	—	3.3	22.3	6	100	80	—	3.0	27.5
Cerebral aneurysm	34	3	75	75	—	3.1	17.4	10	97	65	—	4.1	20.6
Pharyngeal carcinoma	40	1	68	60	3.1	2.3	28.3	2	80	70	4.0	4.3	47.1
Acoustic neuroma	52	2	87	90	—	4.6	11.7	4	83	103	—	2.4	12.3
Cerebral A-V malformation	26	3	73	90	5.3	4.7	19.0	6	77	105	6.8	3.1	33.1
Acoustic neuroma	52	4	97	120	—	5.1	33.5	6	88	120	—	5.1	36.0
Cerebral aneurysm	38	6	75	92	2.1	5.2	19.6	10	93	90	3.0	5.3	19.6
Cerebral aneurysm	31	6	68	55	2.6	6.3	29.2	10	68	58	2.5	4.9	24.6
Scalp melanoma	40	3	50	83	4.7	6.6	36.5	4	73	74	4.1	6.0	30.8
Cerebral aneurysm	39	3	72	82	—	6.4	36.6	6	83	90	—	6.4	43.8
Cerebral aneurysm	34	3	87	90	2.1	8.7	26.8	8	82	70	2.2	7.3	22.6
Cerebral aneurysm	31	3	93	110	2.6	10.9	31.2	8	80	80	2.0	7.7	28.8
Cerebral aneurysm	53	2	80	90	—	12.1	19.4	3	90	85	—	8.7	20.8
Acoustic neuroma	55	3	80	100	—	11.6	27.0	5	77	110	—	14.1	27.2
Tympanic hemangioma	47	1	87	62	2.2	12.3	32.7	3	107	85	2.9	15.2	33.7
Cerebral aneurysm	45	2	80	80	—	21.9	25.7	3	80	65	—	18.2	21.2
Suboccipital meningioma	61	2	93	85	—	17.1	27.5	4	107	95	—	22.5	27.3

shunting had a mean $\dot{Q}_s/\dot{Q}_t$ of 4.0 ± 0.4 per cent in the early period and 3.8 ± 0.4 per cent in the later one (insignificant statistically), and those with increased shunts had a mean $\dot{Q}_s/\dot{Q}_t$ of 13.5 ± 1.7 per cent in the early period and 13.4 ± 2.2 per cent in the later one (also statistically insignificant).

Many of the neurosurgical patients required hyperventilation to provide satisfactory operating conditions and so respiratory alkalosis was frequent, but the P_{aCO_2} did not correlate well with $\dot{Q}_s/\dot{Q}_t$ ($r = 0.05$) indicating that the level of ventilation did not affect the shunt.

The small size of the sample precluded an evaluation of the effect of smoking habits or preoperative sedation and bed rest upon the shunt.

DISCUSSION

Pulmonary disease states and the aging process both cause airway closure early during expiration and long before the resting lung volume is reached.¹⁵ Trapped distal airspace gas rapidly reaches equilibrium with the mixed venous blood, and if reexpansion of the alveoli which now requires a greater opening force does not occur, atelectasis ensues and pulmonary oxygen exchange is disturbed. When elderly or sick patients are anaesthetized their preexisting abnormal shunts usually increase because the functional residual capacity (FRC) decreases with anaesthesia and an even greater discrepancy between the resting lung volume and the closing volume (CV) develops.¹⁶ The data from our older patients merely confirm this fact. However, young and healthy patients with minimal preoperative $\dot{Q}_s/\dot{Q}_t$ have not been studied during anaesthesia until recently, and new evidence indicates that anaesthesia does not decrease FRC in these individuals.¹⁷ Nor does the volume of trapped gas in the lungs increase.¹⁸ In other words, throughout the respiratory cycle the FRC remains well above the CV during anaesthesia in the young and healthy, and so atelectasis and increased shunting ought not to occur. Clearly data from the first 15 patients in Table I demonstrate minimal pulmonary shunting during anaesthesia and operation. Marshall, *et al.* recently reported six more anaesthetized subjects in whom $\dot{Q}_s/\dot{Q}_t$ was found to be within the normal range.¹¹ Other papers which provide data from individual patients document that at least in some patients anaesthesia and operation were conducted without the development of pulmonary shunting.^{1,19-22} Certainly there is now adequate evidence in the literature so that one cannot dismiss normal values of $\dot{Q}_s/\dot{Q}_t$ during anaesthesia as merely erroneous measurement. More such reports would doubtless exist, if only $\dot{Q}_s/\dot{Q}_t$ did not require invasive measurements.

One situation where invasive monitoring is standard practice in the anaesthetized youngster is open heart surgery, but most of these patients have pre-existing pulmonary pathology. The pulmonary artery pressures can be normal pre-operatively, however, if cardiac failure and pulmonary overcirculation are absent. Table II contains data during anaesthesia and operation in five such patients. $\dot{Q}_s/\dot{Q}_t$ was measured after successful weaning from cardiopulmonary bypass just prior to closure of the median sternotomy. Blood pressures had returned to pre-bypass levels but cardiac output was not measured. The pleura were intact and the lungs were not compressed. Again the level of pulmonary shunting was minimal.

TABLE II

Surgical lesion	Age (years)	Minutes after bypass	Paco ₂ (torr)	Q _s /Q _t (%)
Pulmonary stenosis	1	15	35.6	4.6
Tetralogy of Fallot	2	45	23.4	1.5
Atrial septal defect	15	60	59.9	5.0
Atrial septal defect	19	30	21.2	6.4
Aortic stenosis	22	25	32.0	5.9

In the clinical environment in which these studies were done, it was not possible to determine $\dot{Q}_s/\dot{Q}_t$ pre-operatively; hence we do not know whether the shunt fraction increased, decreased or remained unchanged with the institution of anaesthesia. Nevertheless, as no one has suggested that anaesthesia decreases pulmonary shunting, normal levels during anaesthesia are very significant.

However, we do not mean to infer that most surgical patients do not develop increased pulmonary shunting during anaesthesia. This is indisputable and reasonable, for many patients are elderly or obese or have pre-existing cardiopulmonary dysfunction; moreover, the lungs are often compressed at operation. Objection is raised only when this observation is expanded and inferences are drawn about all anaesthetized patients. We therefore conclude that in the young and healthy individual abnormal pulmonary shunting should not be considered an unavoidable consequence or sequela of anaesthesia.

SUMMARY

$\dot{Q}_s/\dot{Q}_t$ was determined during halothane and oxygen anaesthesia in 22 patients. All were believed to have no pulmonary dysfunction. A surgical operation was in progress and respiration was controlled. $\dot{Q}_s/\dot{Q}_t$ was minimal in 15 of these patients (mean 3.9 ± 0.3 per cent), indicating that anaesthesia and operation can be conducted in man without a significant increase of pulmonary shunting. The level of shunting was increased in the other seven patients. In neither group was $\dot{Q}_s/\dot{Q}_t$ altered by the passage of time. Age varied between 23 and 61 years and was directly and significantly correlated to $\dot{Q}_s/\dot{Q}_t$ ($r = 0.57$, $P < 0.01$).

RÉSUMÉ

Chez 22 malades, les auteurs ont déterminé le $\dot{Q}_s/\dot{Q}_t$ au cours de l'anesthésie à l'halothane et à l'oxygène. Aucun de ces malades ne démontrait de dysfonction pulmonaire. Ces déterminations ont été faites au cours de la chirurgie et pendant que la ventilation était contrôlée. Chez 15 de ces malades, le $\dot{Q}_s/\dot{Q}_t$ était minime (3.9 ± 0.3 pour cent); ces résultats indiquent que chez l'homme l'anesthésie et la chirurgie ne produisent pas nécessairement une augmentation significative du shunt pulmonaire. Toutefois, le pourcentage de shunt était plus élevé chez les sept autres patients. Dans aucun des deux groupes le $\dot{Q}_s/\dot{Q}_t$ n'a été modifié par le temps. L'âge des malades variait entre 23 et 61 ans, et une corrélation directe et significative fut notée entre l'âge et le shunt ($r = 0.57$, $P < 0.01$).

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