Hypoxaemia after partial gastrectomy

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After partial gastrectomy there is hypoxaemia which persists for many days. This occurs in patients without complications as well as in those who develop pulmonary collapse ; and although pulmonary mechanical function is much impaired as a result of the operation in both, in neither is post-operative hypoxaemia due to inadequate alveolar ventilation since the arterial (alveolar) CO_2 tension is normal or even reduced (Palmer and Gardiner, 1964). The degree of hypoxaemia does not strictly parallel the reduction in pulmonary mechanical function, and it is not apparently due to a reduction in diffusing capacity (Asmussen and Nielsen, 1960). It has been suggested that it arises because the inspired air and pulmonary blood do not meet in the correct proportions in the lung, *i.e.*, there is within the lung a disturbance of the normal ventilation to perfusion relationship (Nunn and Payne, 1962). Because arterial oxygen-tension measurement is a more sensitive method of detecting hypoxaemia at the upper end of the haemoglobin oxygen dissociation curve than oxygen-saturation measurements, we have measured arterial oxygen tensions directly and have studied the effect of ventilation to perfusion imbalance by measuring alveolar-arterial oxygen-tension differences and physiological dead-space to tidal-volume ratios in patients who had a partial gastrectomy.

METHODS

Ten patients who had been admitted to a surgical unit for elective surgery for chronic peptic ulcer were studied before the operation and for five days afterwards. During anaesthesia all the patients were paralysed and artificially ventilated with a Blease Pulmoflater. Eight were premedicated with omnopon, atropine, and scopolamine, and two were given omnopon and scopolamine only. Portable chest radiographs were taken during the period of study at daily intervals. Arterial blood samples were obtained from the femoral artery in a heparin-lubricated syringe. The blood was allowed to fill the syringe under its own pressure for one to two minutes, and during this period the patient was encouraged to relax and breathe normally.

The following analyses were carried out forthwith:

The percentage arterial oxygen saturation ($SO_2\%$) was measured in a Brinkman haemoreflector, calibration curves being constructed for each patient.

The arterial oxygen tension (PaO_2) was measured in a Clark oxygen electrode. The method was calibrated with oxygen/nitrogen gas mixtures which had been analysed with a Lloyd-Haldane gas analyser which is accurate for oxygen to 0.04%, and a correlation coefficient of 0.997 was obtained. The arterial carbon dioxide tension ($PaCO_2$) was measured in a Severinghaus electrode system. This was calibrated with carbon dioxide/oxygen gas mixtures and measured also with a Lloyd-Haldane gas analyser, which is accurate for CO_2 to 0.03%, and a correlation coefficient of 1.00 was obtained. The *p*H was measured in a capillary glass electrode reading to 0·005 *p*H unit.

Pulmonary ventilation was measured over 10 minutes with a respiration gasometer, the patients being semi-recumbent. The tidal volume (V_T) was derived from the respiratory rate (f). Of each expiration, 0·6% was sampled into a rubber bag attached to the gasometer, and this was analysed for carbon dioxide content (F_ECO_2) and oxygen content (F_EO_2) in the Lloyd-Haldane gas analyser. After the respiratory exchange ratio had been calculated, the alveolar oxygen tension (P_AO_2) was calculated from the equation:

$$P_AO_2 = P_IO_2 - P_ACO_2\left(F_IO_2 + \frac{1 - F_IO_2}{R}\right)$$

where P_IO_2 = the moist, inspired oxygen tension and F_IO_2 = the inspired oxygen content = 20·93% and P_ACO_2, the alveolar CO_2 tension, is taken to equal the measured arterial CO_2 tension (P_aCO_2).

Physiological dead-space (V_D) was calculated from the equation:

$$V_D = \frac{P_aCO_2 - P_ECO_2}{P_aCO_2} V_T - \text{Apparatus dead space}$$

where P_ECO_2 is the CO_2 tension of the expired air, the physiological dead-space to tidal-volume ratio being $V_D/V_T \times 100$.

RESULTS

For analysis the patients are separated into two groups. Group A is the uncomplicated group in which there were four men and one woman. The mean age was 52.2 years, and one patient was mildly bronchitic. Group B is the complicated group who developed clinical and radiological evidence of pulmonary collapse. There were four men and one woman. The mean age was 44.4 years, and one was asthmatic and three bronchitic.

Before the operation in both groups the mean blood gas tensions and the mean alveolar-arterial O_2 tension differences (normal <10 mm. Hg) were normal (Table). After the operation slight hypoxaemia developed, which was slightly more marked in the complicated group, and in both it was most evident during the first three post-operative days. It was accompanied by an increase in the alveolar-arterial O_2 tension difference, which was proportional to the degree of hypoxaemia. Physiological dead-space tidal-volume ratios were increased (normal <30%) before the operation, but in both groups this ratio increased more afterwards. $PaCO_2$ levels were in general slightly less after the operation in both groups, but arterial pH values did not change significantly in either group.

DISCUSSION

After partial gastrectomy, both in normals and in those who developed pulmonary collapse, the arterial oxygen tension was reduced, and this persisted for at least five days. The degree of hypoxaemia was only slightly greater in the complicated group, but the areas of pulmonary collapse were small, and no patient developed a severe post-operative chest infection. In both groups the hypoxaemia was associated with a proportionate increase in the alveolar-arterial oxygen tension gradient. Alveolar oxygen tensions were normal so that there was an adequate diffusing gradient for oxygen and, as the arterial CO_2 tensions were not raised, overall alveolar ventilation was adequate. Because there was no radiological evidence of pulmonary collapse in the normal group, we postulate that hypoxaemia arose because there was underventilation in some alveoli leading to increased alveolar-arterial oxygen tension gradients, so that pulmonary capillary blood was inadequately oxygenated, an effect which is equivalent to a right to left shunt. When pulmonary collapse was present in addition, there was blood flow through unventilated areas so that the shunt effect was increased and the desaturation was therefore greater.

The V_D/V_T ratios were slightly increased before the operation due, we think, to anxiety over-ventilation. The ratios were increased more, however, after the operation again due to overventilation, which indicates that there were alveoli in which ventilation was excessive relative to the amount of pulmonary blood flow. This might lead to hypocapnia, and after the operation there was a slight fall in mean arterial PCO_2 levels (especially in normals) from the pre-operative values. Recently, it has been suggested that premedication

TABLE

MINUTE VENTILATION, PHYSIOLOGICAL DEAD-SPACE TO TIDAL-VOLUME RATIOS, ARTERIAL OXYGEN SATURATION, BLOOD GAS TENSIONS, ALVEOLAR-ARTERIAL OXYGEN TENSION GRADIENTS, AND pH BEFORE AND AFTER PARTIAL GASTRECTOMY

After Operation (days)	Mean $\dot{V}$ l./min. B.T.P.S.	Mean f b.p.m.	Mean V_D (ml. B.T.P.S.)	Mean $\frac{V_D}{V_T} \times 100$ (%)	Mean $PaCO_2$ (mm. Hg)	Mean SaO_2 (%)	Mean PaO_2 (mm. Hg)	Mean PaO_2 (mm. Hg)	Mean $PAO_2 - PaO_2$ (mm. Hg)	Mean pH
Group A (Normals)										
Pre-op.	7·2	15·6	202	43·0	41·7	97·7	96·4	102·3	5·9	7·42
1	8·4	18·8	224	49·0	39·1	95·0	85·5	107·2	21·7	7·43
2	8·5	18·2	263	54·0	39·1	95·5	84·5	102·7	18·2	7·42
3	8·2	14·2	278	48·8	38·3	95·3	83·3	106·2	23·0	7·43
4	8·9	15·4	305	51·3	39·3	96·1	86·5	103·2	16·7	7·42
5	8·6	14·0	313	49·0	39·8	95·9	87·6	104·4	16·8	7·42
Group B (Patients with Pulmonary Collapse)										
Pre-op.	6·8	12·0	216	36·9	40·9	97·8	99·2	102·7	3·5	7·41
1	7·9	16·6	219	45·5	42·2	91·9	80·8	101·0	20·2	7·39
2	9·2	15·4	273	44·7	40·0	89·9	76·6	103·2	26·6	7·42
3	8·4	16·4	241	45·1	38·1	91·6	78·1	105·2	27·1	7·42
4	8·1	15·4	256	46·3	40·5	94·3	82·8	101·5	18·7	7·42
5	7·5	14·4	196	35·6	38·6	96·1	85·3	100·3	14·9	7·43

with atropine may be a factor in the development of post-operative hypoxaemia because desaturation was found after the administration of atropine and before the induction of anaesthesia (Tomlin, Conway, and Payne, 1964). The cause of this is not clear. Atropine causes a 30% increase in anatomical dead-space (Severinghaus and Stupfel, 1955), so that if the tidal volume remained the same or became less, alveolar ventilation would be reduced, and this might lead to hypoxaemia. But it would also result in hypercapnia, and this was not found by Tomlin and his colleagues ; instead, the mean carbon dioxide tensions were somewhat reduced. We think that the disturbance in pulmonary mechanical function which follows abdominal operations irrespective of the type of premedication used, *viz.*, reduced diaphragmatic movement and rapid shallow breathing in the expiratory position (Palmer and Gardiner, 1964) and perhaps also the inability to take periodic deep breaths (Bendixen, Hedley-Whyte, and Laver, 1963), is of major importance in the causation of hypoxaemia, because as a result of these changes regional hypoventilation of the lung bases readily arises leading to low ventilation to perfusion ratios. The areas of lung with high ventilation to perfusion ratios are, therefore, in the upper parts of the lung.

These changes in blood gases are slight and are unlikely to be of much significance to patients in good health, even when small areas of atelectasis develop after the operation. They are likely to be of greater significance in older patients and in those with respiratory, cardiac or cerebrovascular disease.

SUMMARY

The effect of partial gastrectomy on arterial oxygen saturation, blood gas tensions, alveolar-arterial O_2 tension gradients, and physiological dead-space/tidal volume ratios was studied in 10 patients. Five had no complications, but five developed clinical and radiological evidence of pulmonary collapse. The changes were similar in both groups, but were slightly more marked in the complicated group. There was slight arterial hypoxaemia lasting at least five days, especially during the first three post-operative days, but there was no evidence of overall alveolar hypoventilation as the arterial P_{CO_2} levels were normal. In both groups, the hypoxaemia was associated with a proportionate increase in the alveolar-arterial oxygen tension gradient, and physiological dead-space/tidal-volume ratios were also increased somewhat before and after the operation, presumably due to overventilation. It was concluded therefore that hypoxaemia after operations arises as a result of disturbances of the normal ventilation to perfusion relationships within the lung. These changes are unlikely to be of significance to patients in good health but may be of significance to older patients and when there is cardiac, respiratory or cerebrovascular disease.

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