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Hypoxaemia after left thoracotomy for benign oesophageal disease

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ABSTRACT Arterial blood gases were measured before and after operation in 14 patients undergoing conservative oesophageal surgery via a left thoracotomy. All the patients had a preoperative partial pressure of oxygen (Po_2) of > 10 kPa, and none gave a history of chronic respiratory disease. All exhibited a fall in Po_2 values after operation, the mean maximum reduction being 31%. The overall pattern of hypoxaemia was similar to that previously reported after pulmonary resection,[1] and upper abdominal surgery,[2] characterised by the greatest reduction in Po_2 on the first two postoperative days, followed by a gradual return towards preoperative values. In addition, there was a marked similarity in the degree of hypoxaemia observed by Parfrey *et al*[2] and by ourselves. These findings are attributed to similar changes occurring in pulmonary function in both series of patients, caused at least in part by different factors associated specifically with the two surgical approaches involved. In our experience, left thoracotomy, even without pulmonary resection, is associated with significant postoperative hypoxaemia. Knowledge of this may assist in the selection of patients for surgery and in their subsequent management.

It is generally acknowledged that arterial hypoxaemia occurs commonly after operation. The cause of the hypoxaemia appears to be a combination of factors rather than any single mechanism.[3] One factor which has been shown to affect the severity of hypoxaemia is the site of operation.[4 5] In this report, upper abdominal incisions are associated with more profound hypoxaemia than incisions in the lower abdomen. Though there have been reports of hypoxaemia after pulmonary resection,[1 6 7] there appear to be no published data regarding arterial oxygen levels after thoracotomy for non-cardiopulmonary disease. We have measured the degree and duration of hypoxaemia after conservative oesophageal operations, so as to provide an index of the disturbance caused by thoracotomy and routine handling of the lung. It is hoped that a knowledge of this may help in the preoperative selection of patients, lead to improved management of the period of hypoxaemia, and encourage methods of minimising it.

Methods

Fourteen patients undergoing left thoracotomy, none of whom gave a history of chronic respiratory disease, were studied. Preoperative pulmonary function was assessed by measuring the forced vital capacity (FVC), and the one second forced expiratory volume (FEV_1), using a Vitalograph machine.

Arterial blood samples were obtained by direct femoral artery puncture and collected into heparinised syringes. The partial pressures of oxygen and carbon dioxide (Po_2 and Pco_2 respectively), were measured within five minutes using a pH blood gas analyser (ABL 1 Radiometer Copenhagen). Samples were taken on the day before operation in all cases. In 11 cases samples were obtained on the first six days after operation, while in the remainder sampling was performed on the first and third days only. Patients had been breathing room air for a minimum of 20 minutes before arterial puncture, and had received no opiate analgesia for four hours.

Anaesthesia was induced with thiopentone or methohexitone, and a Robertshaw double lumen endotracheal tube passed. Nitrous oxide, supplemented in four cases with halothane, was used to maintain anaesthesia. Thoracotomy was via the sixth left intercostal space, with posterior division of the seventh rib. After the pleural cavity had been opened, the left lung was collapsed and it

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Table 1 *Age, sex, smoking habits, surgical procedures performed, duration of anaesthesia, and preoperative spirometric measurements in the 14 patients studied*

Case	Age (yr)	Sex	Cigarettes per day	Surgical procedure	Duration of anaesthesia	Preoperative spirometry		
						FVC (l)	FEV_1 (l)	$FEV_1/FVC\%$
1	50	F	None	Hiatal hernia repair	2 h 30 min	2·42	1·95	81
2	56	F	20	Hiatal hernia repair	2 h 50 min	3·38	2·63	78
3	55	F	10	Hiatal hernia repair	2 h 5 min	2·50	1·85	74
4	64	F	15	Hiatal hernia repair	2 h 20 min	2·80	1·35	48
5	43	M	None	Hiatal hernia repair	3 h	3·95	3·20	81
6	49	F	15	Hiatal hernia repair and oesophagoplasty	2 h 30 min	3·75	2·70	72
7	63	F	None	Cardiomyotomy	1 h 55 min	2·55	1·80	71
8	62	F	None	Hiatal hernia repair	1 h 45 min	2·75	2·25	82
9	74	F	None	Hiatal hernia repair	2 h 15 min	1·75	1·20	69
10	36	F	15	Hiatal hernia repair	2 h 10 min	3·00	2·15	72
11	62	F	30	Hiatal hernia repair	2 h 30 min	3·00	2·25	75
12	67	F	None	Hiatal hernia repair	1 h 40 min	2·20	1·65	75
13	58	M	None	Hiatal hernia repair	3 h	4·50	3·10	69
14	64	F	None	Oesophagoplasty	2 h 5 min	2·60	1·80	69

was not reinflated until wound closure had been started. A record was made of the duration of anaesthesia and the volume of blood and intravenous clear fluid given.

After operation all patients were returned to the general surgical ward, and a routine policy of care followed. This involved the administration of eight litres per minute of oxygen via a Puritan nebuliser and face mask, so as to produce an oxygen concentration of 40%, for the first 24 hours. Chest physiotherapy was begun within a few hours of operation, and continued at regular intervals throughout the postoperative period.

Results

Details of the patients' ages, sex, smoking habits, spirometry, surgical procedures, and duration of anaesthesia are given in table 1. Table 2 shows the P_{O_2} values recorded for each patient, and the maximum percentage fall in P_{O_2} in relation to preoperative levels. The mean preoperative P_{O_2} was 11·8±0·29 kPa (fig 1), none of the patients having a value less than 10 kPa. On the first day after operation all patients had a P_{O_2} level less than before operation. In nine of the 11 patients studied on the first six days after operation, the lowest P_{O_2} for each individual was recorded on days one or two, and on day four in the remaining two. The pattern of postoperative hypoxaemia is shown in fig 1. Although this indicates an improvement in P_{O_2} values after the initial fall, no patient had regained the preoperative level by the sixth day. The maximum percentage reduction in P_{O_2} from preoperative levels in each individual ranged from 19·8% to 43·7% with a mean 31·0%. Figure 2 shows the mean percentage fall in P_{O_2} on each day after operation, the greatest fall being 29±0·26% on day two. The mean changes recorded in P_{CO_2} levels, shown in fig 1, were not significant.

Each patient was given one litre of clear intra-

Table 2 *Arterial P_{O_2} values for each patient before and after operation. Mean values on each day of the study are shown and the maximum percentage fall in P_{O_2} for each individual*

Case	Before operation	After operation						Maximum % fall in P_{O_2}
		Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	
1	11·3	8·1		9·4				
2	10·1	9·8		11·0				
3	10·3	9·2		10·8				
4	10·5	7·1	6·9	7·0	6·9	8·4	7·9	34·3
5	12·6	11·0	10·0	10·8	11·7	12·2	11·6	20·6
6	13·2	9·7	10·1	13·2	12·3	11·2	12·8	26·5
7	12·4	7·4	7·3	8·5	9·5	11·4	10·4	41·1
8	12·6	10·1	10·1	10·7	10·5	10·3	10·4	19·8
9	11·1	7·4	8·2	10·1	10·3	10·1	9·3	33·3
10	13·2	9·9	8·7	9·7	11·3	11·2	11·4	34·1
11	11·1	7·4	7·5	8·5	8·2	9·4	9·6	30·6
12	11·1	8·6	7·7	9·2	8·6	9·2	10·1	33·3
13	13·5	7·6	8·6	10·9	9·6	11·1	11·2	43·7
14	12·2	9·5	9·7	11·2	9·3	10·0	9·0	23·8
Mean	11·8	8·8	8·6	10·1	9·8	10·4	10·3	31·0

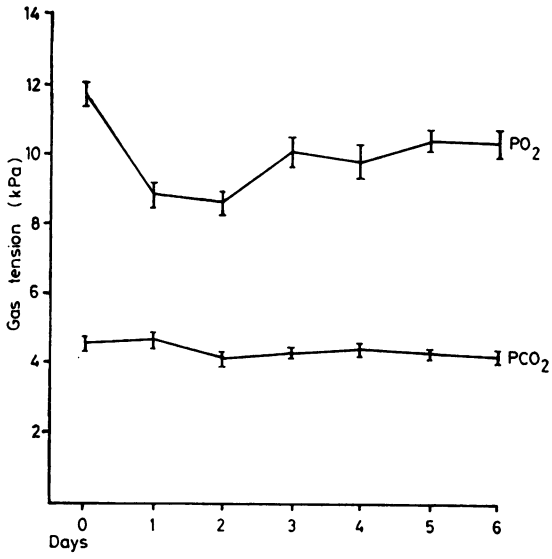

Fig 1 *Mean (± SE mean) arterial Po_2 and Pco_2 values before operation and on the first six days after thoracotomy.*

venous fluid during operation, and between five and six litres in the next 48 hours. Six patients also received blood either during operation or in the immediate postoperative period, but in no case were more than two units given. The mean haemoglobin concentration was 14±0·27 g/dl before operation, and 12·9±0·26 g/dl on day three.

All the patients made uncomplicated recoveries with the exception of case 9 who developed a wound infection. There were no hospital deaths, though one patient (case 10) died at home 20 days after operation, from pulmonary embolism.

Discussion

Maier and Courand[6] measured arterial oxygen saturation in patients undergoing pulmonary resection and found that it was frequently reduced for a week or longer after operation. This was confirmed by Björk and Hilty,[7] who attributed it to impaired ventilation caused by pain, atelectasis, and paradoxical chest wall movements. The concept that postoperative hypoxaemia is peculiar to thoracic surgery was disproved by Nunn and Payne,[8] who observed it after minor operations under general anaesthesia, and hypoxaemia after abdominal surgery is now well documented.[5 9 10]

In the present series in which lung function was not directly altered by resection, we have observed an overall pattern of hypoxaemia characterised by a fall in Po_2 maximal on the first two days after operation, followed by a gradual recovery towards preoperative levels. This is similar to the pattern that was noted by Siebecker *et al*,[1] who measured arterial oxygen saturation in patients undergoing pulmonary resection, and by Parfrey *et al*[2] in a study of arterial Po_2 levels after upper abdominal procedures. These findings raise the possibility that hypoxaemia after different types of operation has a common aetiology.

The physiological changes responsible for hypoxaemia after operation have been the source of considerable debate, particularly the extent to which true pulmonary venous shunting as opposed to a ventilation-perfusion mismatch is responsible. Spence and Alexander[11] emphasised the important role played by small airways closure which Alexander *et al*[12] concluded led to a reduction in functional residual capacity (FRC). In a study of 173 patients undergoing a variety of non-thoracic procedures they showed that a fall in Po_2 after operation was paralleled by a reduction in FRC. The latter was considered to be the result of factors such as abdominal distension and abdominal muscle spasm. While these may be of importance after laparotomy, we consider other

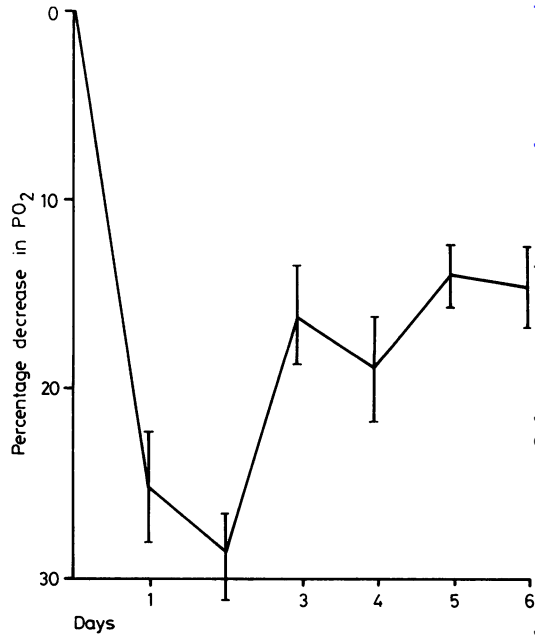

Fig 2 *Mean (± SE mean) percentage decrease in Po_2 from preoperative values during the first six days after thoracotomy.*

factors to be of probably greater significance in patients undergoing thoracotomy.

All our patients were operated on in the right lateral position. Craig *et al*[13] examined radiologically the dependent lung after operation in this position, and observed changes considered to represent areas of de-aeration caused by compression of the chest wall and a relative fixity of the parietes. These changes, which persisted for 48 hours or longer in some cases, might reasonably be expected to contribute to a reduction in FRC. Furthermore, for ease of access to the oesophagus, the left lung was collapsed and gently retracted in the present series. This too may be partly responsible for the observed hypoxaemia. Moreover, the inevitable trauma to the left lung caused by collapse and retraction may have made it abnormally sensitive to changes in the circulating blood volume. It is perhaps significant in this context that, though none of the patients showed overt signs of intravenous fluid overload, the period of maximum hypoxaemia coincided with that of intravenous fluid administration. Finally, pain from the incision, by inhibiting deep inspiration, would also be expected to impair respiratory function and reduce arterial P_{O_2}.

We were also interested in the severity of hypoxaemia after thoracotomy. The greatest reduction in P_{O_2} occurred on the second postoperative day, the mean percentage fall being $29 \pm 2{\cdot}2$. This is very similar to that noted by Parfrey *et al*[2] after upper abdominal procedures (28%). Other workers however, have previously reported less severe hypoxaemia after upper abdominal operations. Thus it is not clear whether simple thoracotomy—that is, without pulmonary resection—is associated with more severe hypoxaemia than is upper abdominal surgery. However, from the foregoing discussion regarding possible aetiological factors, it might be expected that a combined thoracoabdominal incision would produce more profound hypoxaemia than either alone.

In this respect, the work of Black *et al*[14] is of interest. In a group of 18 patients undergoing oesophageal resection via a left thoracoabdominal incision, they noted a mean fall in P_{O_2} from 11·7 kPa before operation to 8·0 kPa on the first postoperative day, followed by a rise to 9·0 kPa on the third day. This indicates more severe hypoxaemia than in the present series, possibly associated with division of the left hemidiaphragm and costal margin. However, in another group of 14 patients undergoing resection via abdominal and separate right thoracic incisions, an initial similar fall in P_{O_2} was followed by a recovery to 11·1 kPa on the third day. This suggests better recovery than in the present series, in which thoracotomy alone was performed. The explanation of this surprising finding is not known, but factors such as duration of anaesthesia and postoperative changes in haemoglobin concentration do not appear to account for it. It is perhaps merely a reflection of the rather small numbers of patients studied, though other factors which we are unable to compare such as, amounts of fluid given intravenously, may be relevant.

We conclude that left thoracotomy without lung resection is associated with a marked degree of postoperative hypoxaemia. Of the 14 patients studied, six had a P_{O_2} after operation of <8 kPa on at least one occasion. Despite this, all made uncomplicated recoveries. However, in patients with chronic respiratory disease, more profound hypoxaemia might be anticipated, and in such cases a knowledge of the expected changes should allow appropriate prophylactic measures to be taken.

References

1 Siebecker KL, Sadler PE, Mendenhall JT. Postoperative ear oximeter studies on patients who have undergone pulmonary resection. *J Thorac Surg* 1958; **36**:88–91.

2 Parfrey PS, Harte PJ, Quinlan JP, Brady MP. Postoperative hypoxaemia and oxygen therapy. *Br J Surg* 1977; **64**:390–3.

3 Editorial. Postoperative hypoxaemia. *Br J Anaesth* 1971; **43**:299.

4 Diament ML, Palmer KNV. Postoperative changes in gas tensions of arterial blood and in ventilatory function. *Lancet* 1966; **2**:180–2.

5 Parfrey PS, Harte PJ, Quinlan JP, Brady MP. Pulmonary function in the early postoperative period. *Br J Surg* 1977; **64**:384–9.

6 Maier HC, Cournand A. Studies of the arterial oxygen saturation in the postoperative period after pulmonary resection. *Surgery* 1943; **13**: 199–213.

7 Björk VO, Hilty HJ. The arterial oxygen and carbon dioxide tension during the postoperative period in cases of pulmonary resections and thoracoplasties. *J Thorac Surg* 1954; **27**:455–67.

8 Nunn JF, Payne JP. Hypoxaemia after general anaesthesia. *Lancet* 1962; **2**:631–2.

9 Palmer KNV, Gardiner AJS, McGregor MH. Hypoxaemia after partial gastrectomy. *Thorax* 1965; **20**:73–5.

10 Knudsen J. Duration of hypoxaemia after uncomplicated upper abdominal and thoracoabdominal operations. *Anaesthesia* 1970; **25**: 372–7.

11 Spence AA, Alexander JI. Mechanisms of post-

operative hypoxaemia. *Proc R Soc Med* 1972; **65**:12–14.

12 Alexander JI, Spence AA, Parikh RK, Stuart B. The role of airway closure in postoperative hypoxaemia. *Br J Anaesth* 1973; **45**:34–40.

13 Craig JOC, Bromley LL, Williams R. A study of the changes occurring in the dependent and contralateral lung during and after thoracotomy in lateral decubitus. *Thorax* 1962; **17**: 9–15.

14 Black J, Kalloor GJ, Leigh Collis J. The effect of the surgical approach on respiratory function after oesophageal resection. *Br J Surg* 1977; **64**:624–7.