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THE EFFECTS OF SURGICAL OPERATIONS ON THE PHYSIOLOGY OF RESPIRATION

A REVIEW OF PRESENT KNOWLEDGE

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From the very beginning major abdominal operations have been followed by respiratory-tract complications. The reasons for, and mechanisms of, these complications have been the subject of intense study. Some reflection of the changing views on their aetiology is given by the titles of papers on the subject.

The 'massive collapse' of Pasteur[1, 2] (1908 and 1913) and of Elliot and Dingley[3] (1914) gave way to the 'post-operative pneumonia' of Elwyn[4, 5] (1922 and 1924) and Featherstone[6] (1924), and later to the 'postoperative atelectasis' of such authors as Coryllos[7] (1932), Van Allen and Jung[8] (1932), DeTakats *et al.*[9] (1942), Stringer[10] (1947), and Lucas[11] (1950). More recently Brattström[12] (1954), Anscombe[13] (1957), and Piper[14] (1958), have been less ready to pinpoint the nature of the process, and have been content with titles such as 'postoperative pulmonary complication'.

The development of diagnostic radiology at the beginning of the century resulted in the demonstration of an incidence of postoperative pulmonary disease far greater than had been anticipated clinically, and through the years until the present time the high incidence thus demonstrated has remained remarkably stable.[13]

In 1958, at the Queen Elizabeth Hospital, Birmingham, Bevan[15] found a radiological incidence of lobar, or greater, atelectasis in 35% of a series of 82 consecutive abdominal-operation patients who had daily radiographs for the first 10 postoperative days. Recognizable segmental lesions were present in a further 17%, making the total radiological incidence of atelectasis 52%.

It is of interest to note that the apparent incidence of postoperative pulmonary complications increases whenever the physician is called in by the surgeon to review the situation.[16]

Similarly, as development of surgical and anaesthetic techniques allowed the surgeon to attack intrathoracic disease with a reasonable expectation of success, it became evident that respiratory complications were a major hazard in the management of patients who had undergone thoracic operations.[7] This was apparently particularly so in subjects whose pulmonary reserve was already diminished before operation.

The severe effects of the sudden reduction of respiratory reserve associated with the removal of lung tissue, of interference with thoracic cage or diaphragmatic movement, of the respiratory depressant action of powerful analgesics and anaesthetics, of pain, of infection, and often of circulatory disturbance, can bring the patient to the brink of disaster. At least half the early postoperative mortality from thoracic operations is due to acute cardiorespiratory failure,[17-23] and there are a number of reports in the literature dealing with the postoperative management of this condition,[7, 24-28] or with its anticipation.[29]

It is generally agreed, however, that the incidence of postoperative pulmonary complications in non-thoracic, non-abdominal operations is small—usually under 3%.[5, 13, 16, 30-32]

Numerous theories as to the mechanisms of the condition have been proposed and, according to Anscombe,[13] they can be divided into 6 overlapping groups as follows: (1) nervous reflex, (2) mucous plug, (3) deficiency of muscle power, (4) small pulmonary emboli, (5) pulmonary congestion, and (6) aspiration of foreign material. A similar and even more detailed review of the theories of causation is given by Brattström.[12] The main concern of this paper, however, is the physiological effects of the condition.

EARLY PHYSIOLOGICAL FINDINGS: AFTER ABDOMINAL OPERATIONS

1. Lung Volumes

(*a*) *Vital capacity.* The earliest studies came from Boston,[33, 34] and these were quickly followed by other papers from America and England.[35-38] The most recent and fullest studies are those of Anscombe[13] and Bevan,[15] and of Scheinin.[39] These authors without exception concluded that immediately after an abdominal operation the vital capacity is reduced, that this reduction is greater in upper abdominal than lower abdominal operations, that it is greater in males than in females, that it is independent of the type of anaesthesia (general, spinal or local), and that it is absent after limb and other peripheral operations. Anscombe[13] gives the postoperative reduction in vital capacity, as a percentage of the pre-operative capacity, as 65% after upper abdominal operations, 35% after lower abdominal operations, 25% after herniorrhaphy, and nil after peripheral operations. These are typical figures. Recovery to pre-operative values takes 1 - 2 weeks. The administration of opiates or other potent analgesics results in a slight increase in the postoperative values of vital capacity. Morphine[13] or intravenous procaine[32] improves the postoperative vital capacity on the average by one-eighth of the postoperative value. Powers[40] has shown that the routine administration of 5% carbon dioxide at the end of the operation, and for a few minutes 3 times daily thereafter for the first few days, reduces the postoperative fall in vital capacity by 20%, while a tight abdominal binder increases it by 7% of the pre-operative vital capacity. King[16] found a reduction in the clinical incidence of

complications after CO_2 therapy. In the presence of recognizable pulmonary complications the changes are more marked or recovery is slower.[41]

(*b*) *Residual volume.* Together with the fall in vital capacity there is a reduction in the functional residual capacity. Beecher,[38] using the original closed-circuit nitrogen-washout method of Christie,[42] found the residual volume in 35 patients to be decreased on the average by 13%, but Anscombe and co-workers[13, 43] found no change in this volume in 26 patients by the more accurate closed-circuit helium-dilution method of McMichael[44] as modified by Bates and Christie.[45] In an even more detailed study, by the same method, Scheinin[39] compared the pre-operative value for residual volume with the values measured on the 2nd, 5th and 10th postoperative day. He found no significant change or trend in 32 patients who had undergone laparotomy. All three authors agree with Brattström[12] in recording a fall in the functional residual capacity. Brattström, however, did not record the expiratory reserve volume, and so could give no figures for residual volume.

2. The Diaphragm

Pasteur,[1, 2, 46] on clinical grounds, noted that diaphragmatic movement was reduced or absent after laparotomy. He suggested that this was a basic aetiological factor in postoperative chest disease, drawing attention to the frequency of chest complications on the side of the lesion in diphtheritic paralysis of the diaphragm.

Shortly after the early measurements of vital capacity several groups of authors confirmed Pasteur's suspicions by radiological studies.[36, 37, 47-49] Allen[48] found a rise of the diaphragm on the day after operation in 61% of upper and 34% of lower abdominal operations; Muller *et al.*[36] give average values for the rise as 4·8 cm. on the right and 3·5 cm. on the left. Diaphragmatic movement was reduced in two-thirds of Allen's patients.

Various authors have studied the development and duration of postoperative pneumoperitoneum.[50-52, 15] It is commoner on the right, where the diaphragm rises higher,[36] and lasts from 3 to 10 days.

Bevan[15] found that the degree of pneumoperitoneum showed a marked positive correlation with the incidence of postoperative atelectasis. Pneumoperitoneum occurred in 77% of his patients and pulmonary complications in 52%. The closest association was in the group of upper abdominal operations.

3. Associated Changes in Respiratory Mechanics

Carlson[53] used pneumographic tracings to show that the respiratory rate was increased after operation, and again this was most noticeable in upper abdominal operations. He also showed that, in abdominal operations, abdominal respiratory excursion decreased while thoracic respiratory excursion increased, and that the reverse was true in thoracic operations.

Only Carlson,[53] Anscombe,[13] Bevan[15] and Scheinin[39] have attempted to assess any pulmonary mechanical factors other than resting lung volumes. Patey[37] took tracings of intra-abdominal pressure with a rectal balloon and showed that after abdominal operations the intra-abdominal pressure swing with respiration was reduced; and Carlson[53] extended this work by showing that the maximum sustained blowing pressure that patients could achieve was reduced to 40% of the pre-operative value in upper abdominal operations and 85% in thoracic operations. These findings, like the changes in vital capacity, and in diaphragmatic movements, took 1 - 2 weeks to regress.

Anscombe[13] measured the forced vital capacity and maximum expiratory rate (MER), defined as the forced vital capacity (FVC) divided by the time constant of the vital capacity trace (t), assuming the curve to be exponential, i.e. MER = FVC ÷ t, and found changes in the index which paralleled the changes in vital capacity. This index is essentially an index of flow rate. Flow rate is volume divided by time, and Anscombe's formula is equivalent (by simple algebraic transformation) to 'e' times the reciprocal of the mean flow rate. This parallel loss of flow rate and vital capacity has been shown to be a normal phenomenon by the elegant analysis of Fry and Hyatt,[54] who have shown that there is a limit to the maximum flow rate that can be obtained from a given lung volume, and that this rate diminishes with decreasing lung volumes.

Bevan[15] measured the one-second forced expiratory volume (FEV_1), but did not relate this to the total forced vital capacity, so that no measurements of airway resistance, even in an indirect form, are available in the literature. Scheinin[39] measured the maximum breathing capacity[55] before and after operation in 32 upper laparotomy patients. He found it reduced to 46% of the pre-operative value in the second postoperative day. He later found[41] that patients with postoperative pulmonary complications had greater loss of vital capacity and MBC than those without such complications, and also that the return to normal was delayed in this group.

4. Resting Respiratory Exchange

Beecher[38] calculated the resting minute volume as the average tidal volume multiplied by the measured frequency, and concluded that after operation it remained unchanged, the increase in frequency being compensated for by a decrease in tidal volume.

Although Overholt's paper[47] is entitled 'Postoperative pulmonary hypoventilation', yet he made no measurements of minute volume; he was using the term 'hypoventilation' in a sense different from that commonly understood today. What he meant was that the lower lobes above the raised diaphragm were being underventilated in relation to the rest of the lung, a concept that we now interpret in terms of altered ventilation:perfusion ($\dot{V}/Q$) ratios, and in thinking on these lines in 1930 he was far in advance of his generation. He also mentioned in that paper that he had measured arterial oxygen saturation and found it reduced in a proportion of cases; I have been unable to find any further reference to these data. Klotz and Straaten[56] assume hypoventilation to exist but give no proof of it in their paper.

In 1957 Hamilton and Devine[57] measured the end tidal carbon-dioxide tension, P_{ECO_2}, and minute ventilation, in 100 consecutive postoperative patients. They found that 25 of them had abnormally high values of P_{ECO_2}, indicating hypoventilation at the time of the study, which was 45 minutes after discontinuance of the anaesthetic. No data are given about the rate of recovery.

Troell,[58] and Carlsten, Norlander and Troell,[59] whose

study of postoperative cardiac output is discussed in the next section, make no reference to levels of minute ventilation although, since they used the direct Fick method for studying the cardiac output, presumably the data were available. This suggests that they noted no gross abnormalities.

Brattström[12] also found no evidence of hypoventilation when he calculated the minute ventilation during his nitrogen-washout studies. Scheinin[39] made direct measurements at 2, 5 and 10 days after upper laparotomy and found that minute ventilation was unchanged, but that this volume was the result of turning over a smaller tidal volume at a greater frequency, a pattern that had been noted by all authors since Beecher.[38] Scheinin found no statistical change in end tidal P_{CO_2} and concluded that postoperatively (second day) the alveolar ventilation was adequate. He also noted that end tidal P_{O_2} was unchanged.

5. Blood-gas Data and Haemodynamic Findings

As noted above, Overholt[47] was first in the field. The next references I have been able to obtain are the papers of Troell,[58] and Carlsten, Norlander and Troell,[59] who quote earlier work by Harild[60] which suggested that the commonest response to operation was a fall in cardiac output. These Scandinavian authors used the direct Fick method for measuring cardiac output, and found it to be increased the day after operation by 30 - 50% of the pre-operative values. Their figures for postoperative arterial oxygen saturation are the first published data that confirm the fall in arterial saturation after abdominal operations noted a quarter of a century previously by Overholt.[47] Troell[58] showed falls in arterial saturation of 6 - 11% at 8 hours after operation,[58] with a return to normal values with oxygen therapy. The second paper[59] showed a 5% fall in this value at 24 hours. These authors noted in addition that CO_2 values in arterial blood were usually not significantly changed by operation.

Only 2 other groups have reported data in cases of abdominal operation. In 1958 Mastio and Allbritten[61]

TABLE I. (BASED ON MASTIO AND ALLBRITTEN[61])

Blood-gas data in relation to operation Mean values in 16 patients

	Previously	*End*	*Recovery*	*24 hours after*
pH	7·36	7·28	7·25	7·31
Pa_{CO_2} mm. Hg. ..	42	50	51	45
Sa_{O_2}%	95	106	92	88
$\dot{V}_A$ 1/sq. m./min. ..	2·13		2·34	1·47

reported studies of blood-gas data and alveolar ventilation in a mixed group of 16 patients (8 thoracic and 8 abdominal) at the end of the operation, 30 minutes later in the recovery room, and on the day after surgery. Their main findings are recorded in Table I. They also studied the

TABLE II. (BASED ON MASTIO AND ALLBRITTEN[61])

	Before narcotic	*After narcotic*
Sa_{O_2}%	89	89
$\dot{V}_A$ 1/sq. m./min.	1·48	1·86

effect of a dose of 50 - 75 mg. of meperidine over one hour the day after surgery (Table II); the drug produced no obvious respiratory changes.

Also in 1958 Linderholm and Norlander[62] and Gordh, Linderholm and Norlander[63] published two significant papers. In the first they studied acid-base balance, and in the second oxygen tension in arterial blood when air or oxygen was breathed. The studies were made after premedication for anaesthesia and at various periods up to 24 hours after surgery, and attempts were made to relate the changes found to the type of anaesthetic employed. The older literature on this relationship is quoted, and it was confirmed that metabolic acidosis, or often respiratory acidosis, is found at the end of many operations; 24 hours later alveolar ventilation was normal as measured by arterial P_{CO_2} values, and the metabolic acidosis had disappeared.

In their oxygen-tension studies, they showed that premedicated patients had a lower arterial oxygen tension than they considered normal, and explained this by alterations in $\dot{V}/\dot{Q}$ ratios induced by bed rest and by postural changes, as found by Berggren.[64] The fact that on 100% oxygen breathing the alveolo-arterial oxygen-tension difference increased after operation suggested to them that there was increased venous admixture present as the result of atelectasis, although they only demonstrated radiological changes in 3 out of 47 patients, and only 1 of those 3 showed a postoperative increase in the alveolo-arterial oxygen-tension gradient. The significant figures in their data are contained in Table III.

TABLE III. (BASED ON GOURH, LINDERHOLM AND NORLANDER[63])

Anaesthetic	*Number of subjects*	*Air breathing*				*O_2 breathing* P_{AO_2} *minus* P_{aO_2}	
		Before operation		*After operation*		*Before operation*	*After operation*
		Pa_{CO_2}	Pa_{O_2}	Pa_{CO_2}	Pa_{O_2}		
Barbiturate N_2O	17	40	81	37	73*	154	318†
Ether	13	38	96	37	76†	111	166
Spinal	17	39	86	38	76	138	212†

*Significant at 5% level †Significant at 1% level

6. Abdominal Surgery: Conclusions

It is well established that after an abdominal operation, and particularly an operation in the upper part of the abdomen, there is considerable interference with the mechanical function of the lungs, with a reduction of the vital capacity and the maximal ventilatory capacity to around half the pre-operative values. Respiratory frequency is increased and tidal volume diminished, but total minute ventilation appears to be unaltered, and so is consumption of oxygen and output of carbon-dioxide.

Twenty-four hours after operation the acid-base balance of the body and carbon-dioxide homeostasis is reported as normal, but in most cases there is some oxygen desaturation of arterial blood as compared with the normal. This has been shown to persist in some patients even with the breathing of 100% oxygen. The desaturation has been thought by some to be due to atelectasis, by others to be due to hypoventilation. Atelectasis can be demonstrated radiologically in about 50% of postoperative patients.

PHYSIOLOGICAL STUDIES IN LATE POSTOPERATIVE PHASE: AFTER THORACIC OPERATIONS

In the past the pulmonary physiologist has worked much

more closely with the thoracic surgeon. In spite of this, few studies were made before 1956 of the effects of operations on pulmonary function in the immediate post-operative period. Up till that time most investigators had been concerned with the definition of the long-term effects of lung operations on pulmonary function.

Physiological studies in the late postoperative phase are legion. The series of papers from Dr. André Cournand's laboratory were, as usual, both early in the field and comprehensive in scope (thoracoplasty,[65] pneumonectomy,[66-68] collapse therapy,[69] thoracotomy,[70] ventilation-perfusion studies[71]).

Many other laboratories have also contributed to the problem, studying thoracoplasty and other collapse therapy,[72-75] extra-pleural pneumolysis[76, 77] decortication,[78] segmental pulmonary resections,[79-83] lobectomy,[84, 85] and pneumonectomy.[84, 86-91]

This list is by no means comprehensive. Most laboratories are in agreement, so that it will be sufficient to quote the findings of Cournand's laboratory, noting any disagreements and discrepancies in the findings in comparison with other studies, and taking a patient undergoing full pneumonectomy as the type case.[70, 71]

Pneumonectomy

In 7 patients the average findings 2 - 6 months after pneumonectomy were as follows (I quote from the 1950 paper;[71] items in brackets are my additions to their text, taken from their data):

'1. On deep inspiration the average size of the remaining lung was slightly greater than that of a normal homologous lung, but the average size on forced expiration was 150% of the predicted size of a normal lung and the ratio residual air/total capacity x 100 was elevated somewhat above normal.

'2. The maximum breathing capacity was reduced to 63% of the predicted value for a normal intact chest with both lungs.

'3. A small degree of hyperventilation was present during all periods of observation (4·6 l./sq.m./min.—cf. predicted 3·4 l./sq.m./min.). (The 1942 paper[67] shows that minute ventilation in 12 patients was well within the normal predicted range after pneumonectomy.)

'4. The index of intrapulmonary mixing was in the low range of normal.[92]

'5. The mean oxygen consumption at rest and during the standard exercise was normal.

'6. The arterial oxygen saturation was approximately normal at rest and very slightly reduced following exercise.

'7. The CO_2 tension in the arterial blood while breathing room air was normal at rest and during a steady state of bicycle exercise. The O_2 tension in the arterial blood during room-air breathing was slightly below the accepted normal value and the alveolo-arterial Po_2 gradient was slightly elevated. (In the only two patients studied before pneumonectomy these values were unchanged by the operation). Using these figures for the calculation of effective pulmonary blood flow and of effective alveolar ventilation expressed in per cent of total blood flow and total ventilation, it was found that both were slightly below normal standard values, being respectively 93·6 and 68%. (Minimum normal 94% and 70%.)

'8. The average alveolo-arterial Po_2 gradient while breathing a low oxygen mixture was within the normal range in all cases.

'9. The total pulmonary blood flow and pulmonary arterial pressures measured in two cases in the resting state were normal.'

In a group of patients studied on the average 77 months after pneumonectomy (range 13 - 131 months), there were only two differences of note from the results reported above, viz. (1) the size of the remaining lung on forced expiration was 175% of the predicted normal value for one lung, and (2) the mean pulmonary-artery pressure while normal at rest rose to an abnormal level (24 mm.Hg) on exercise in the three patients in whom data were available.

Other Thoracic Operations

1. Lung Volumes

Vital capacity. Thoracoplasty[18] and segmental resections for tuberculosis[82] or bronchiectasis[79] produce a nearly proportional loss of vital capacity according to the number of ribs cut or segments removed. Lobectomy produces a loss of vital capacity of 20 - 30%.[84]

Residual volume. After a thoracic operation the residual volume changes have a close positive association with vital capacity changes.[39]

2. Mechanics

The maximum voluntary ventilation, as reported above, is depressed after pneumonectomy. It is proportionately less depressed after less radical surgery, and after segmental resections of only one or two segments is hardly, if at all, affected.[82] In thoracoplasty the loss closely parallels the loss in vital capacity.[18] The $FEV_1/FVC\%$ is normal.

Segmental resection does, however, produce a fall in static pulmonary compliance proportional to the number of segments removed,[93] and pulmonary compliance is also lowered after pneumonectomy,[94, 95] in most cases to a level lower than might be predicted. The work of resting ventilation is therefore considerably above normal after pneumonectomy. This is interesting in view of the finding[96] that the compliance of one lung measured by static recoil at open thoracotomy is half the normal value for two lungs. This finding in itself contrasts with the work of Butler and Smith,[97, 116] who found decreased lung compliance in anaesthetized patients.

3. Resting Respiratory Exchange

All the authors already quoted who have made measurements of resting respiratory exchange after any thoracic procedure have reported that the values obtained were normal in all respects. In particular, values were unchanged from pre-operative values in the same patients. As might be expected, consumption of oxygen and output of carbon-dioxide are unchanged.

4. Diffusion

Several groups of workers have studied pulmonary diffusing capacity. Cournand *et al.*[71] found diffusion after pneumonectomy to be at the lower limit of the normal range by Riley's oxygen method. All other workers have measured diffusion of carbon monoxide. Bates,[94] using his own steady-state method,[98] found one-half of normal values for diffusing capacity about 1 year after pneumonectomy. Schramel *et al.*,[99] also using a steady-state method,[100] found a fall in resting diffusing capacity in the first week after cardiac bypass surgery, with a return to normal values within 1 - 2 weeks. They confirmed their findings in dogs subjected to simple thoracotomy. The fall in patients without pre-operative left-to-right cardiac shunts was to 40% of the pre-operative value. All other workers have performed their studies between 3 and 10 weeks after operation and have used the single-breath-holding carbon-monoxide method of Ogilvie *et al.*[101]

Larmi and his co-workers[102-104] found a postoperative

decrease in vital capacity of 41%, in total lung capacity of 21%, and in diffusing capacity of 14%, compared to pre-operative values, in patients undergoing mitral valvotomy. Following pneumonectomy there was a fall of 15% in diffusing capacity, compared with a 40% fall in total lung capacity. A similar fall in diffusing capacity accompanied lobectomy.

Burrows' patients,[105, 91] on the other hand, show proportionate decreases in lung volumes and diffusing capacity both early (4 weeks) and later (3 months) after all types of pulmonary resections, including pneumonectomy, providing the remaining lung is normal.

5. Blood-gas Data and Haemodynamic Findings

All authors agree with the results reported by Cournand *et al.*,[71] that at a late stage after a successful thoracic operation the arterial oxygen saturation is normal at rest (provided always that it was normal before operation). On exercise of moderate degree it is usually normal, or may be slightly below normal. Carbon-dioxide levels in the alveolus (P_{ACO_2}) and in the arterial blood (P_{ACO_2} C_{ACO_2}) are normal. Cardiac output at rest and on exercise is normal, but in some patients, especially in the older age groups, pulmonary arterial pressure is raised during exercise. Harrison *et al.*[106] found that the mean pressure in the pulmonary artery was raised when measured at rest 6 years after operation, even in patients who were clinically symptomless. They found an inverse correlation between the pulmonary-arterial mean pressure and the functional capacity of the patient, both as assessed clinically, and as assessed by tests of respiratory performance.

Findings such as these have led to the suggestion that unilateral pulmonary-artery occlusion developed by Hanson and co-workers,[107, 108] used either alone or in conjunction with bronchospirometry, is a useful pre-operative pulmonary function test. A rise in pulmonary-artery pressure on this manoeuvre augurs badly for the post-operative course.

PHYSIOLOGICAL STUDIES IN RELATION TO ANAESTHESIA

It is of considerable interest that the first report of respiratory insufficiency resulting from an open thorax is that of Vesalius (1543), who described the asphyxial effects of opening the thorax in living dogs and showed that, by cannulating the trachea with a reed and instituting positive-pressure respiration with his own expired air, he could revive the animal and keep it in good condition for as long as he wished while he demonstrated cardiac action to his students.

In more modern times considerable attention has been paid to the effects of anaesthesia in the production of anoxia and acid-base disturbance in thoracic and other surgical patients. Both the methods of anaesthesia used[109-113] and the type of anaesthetic[114, 62] have been studied. It has been found that oxygenation of the arterial blood can easily be maintained by breathing oxygen-enriched mixtures, but that unless respiration is aided by the anaesthetist or by some form of external pump, severe respiratory and metabolic acidosis can easily result, especially since lung-thorax compliance is reduced[95, 97, 115] and there is increased resistance to lung inflation.[115, 116]

The mechanism by which acidosis develops has been studied by a number of groups,[112, 113, 117-119] who have shown that, while there is some alteration in distribution of inspired gas as shown by changes in the ratio of physiological to anatomical dead space and in the end tidal arterial-blood CO_2-tension gradient,[119] these are of little clinical significance compared with faulty anaesthetic technique[118] and, in particular, failure of the anaesthetist to maintain *continuously* adequate minute ventilation.

The subject has recently been carefully reviewed by Gibbon and Nealon.[120]

In the First 24 Hours of the Postoperative Period

These studies can be divided into 'recovery room' studies and 'later' studies.

(a) Recovery-room Studies

When artificial respiration by the anaesthetist is discontinued the patient invariably underventilates,[117, 121-123] even if he has previously been hyperventilated for some time.[124] This hypoventilation may be so marked as to require the reinstitution of artificial respiration, particularly if the lungs are already diseased.[25] Tracheotomy and prolonged artificial ventilation may be required.[24] This phenomenon is by no means confined to thoracic patients, as has already been noted in the present article in the section 'Early physiological findings: after abdominal operations', paragraph (4) 'Resting respiratory exchange'.[57, 121, 61, 62]

(b) Later Studies

Provided that there is no untoward development, respiration usually recovers quite rapidly, so that a number of authors have reported that by 24 hours carbon-dioxide homeostasis has been restored at normal levels of alveolar and arterial P_{CO_2}.[125, 126, 62, 122, 123]

In addition arterial pH has usually been restored to normal values. Not all authors agree on this, however. Mastio and Allbritten[61] found in 16 patients that at 24 hours arterial pH, at 7·31, was low and arterial P_{CO_2}, at 45mm.Hg, was high (see Table I of present article). All authors, however, agree that arterial blood oxygenation is depressed during the whole of this period whether it is measured directly as saturation, or as arterial oxygen tension.

Beyond 24 Hours after Thoracic Operation

(a) Respiratory Exchange and Blood-gas Data

The very first observations of the effects of thoracic surgery on respiratory exchange are the experimental pneumonectomy studies in dogs of Heuer and Andrus[127] from the Johns Hopkins Hospital in 1922. These authors showed by the methods they then had available that after pneumonectomy alveolar oxygen fell, alveolar carbon dioxide rose, and venous blood oxygen saturation fell. The changes they noted took 2 - 3 weeks to regress.

The first studies on man are those in which Maier and Cournand[70] traced the level of arterial oxygen saturation after pneumonectomy and lobectomy. They found that thoracic operations, including simple thoracotomy, resulted in a fall of the oxygen saturation in arterial blood. Saturation returned to normal within 3 or 4 days after pneumonectomy in good-risk patients, but after lobectomy the fall in saturation was rather more severe, lasted rather longer,

and was frequently abnormal as long as a week from the date of operation. The average order of magnitude of the fall was 5%.

These authors noted that complications such as bronchopleural fistula and pulmonary collapse were associated with severer degrees of anoxia. Some of their cases were receiving adequate oxygen therapy when the blood was taken, and it is of interest that in none of the 7 patients in this group was the haemoglobin 100% saturated with oxygen. They performed similar studies in a group of abdominal surgical cases between the 1st and 4th postoperative days and found what they considered were normal values of arterial oxygen saturation in all. No preoperative values were available for this control series.

In the same year Adams *et al.*[109] reported postoperative reduction in arterial oxygen levels in 27% of 44 patients in whom samples were obtained between the 5th and 14th postoperative day.

The next study is that of Björk and Hilty.[126] These authors measured arterial blood-gas tensions by microtonometry (Riley) before, and at varying periods after, a selection of pulmonary resection operations and after thoracoplasty. They found that from 24 hours after operation onwards arterial P_{CO_2} levels were normal except when there had been an abnormally high value before operation. Arterial oxygen tension on the other hand was always reduced after operation; the value recovered progressively towards the patient's normal in the week following pneumonectomy, but was still abnormal after 2 weeks in 7 of 16 patients who had undergone lobar or segmental resections.

These authors also found that most patients were unable to hyperventilate for at least the first few days, and even those who could hyperventilate sufficiently to cause a fall

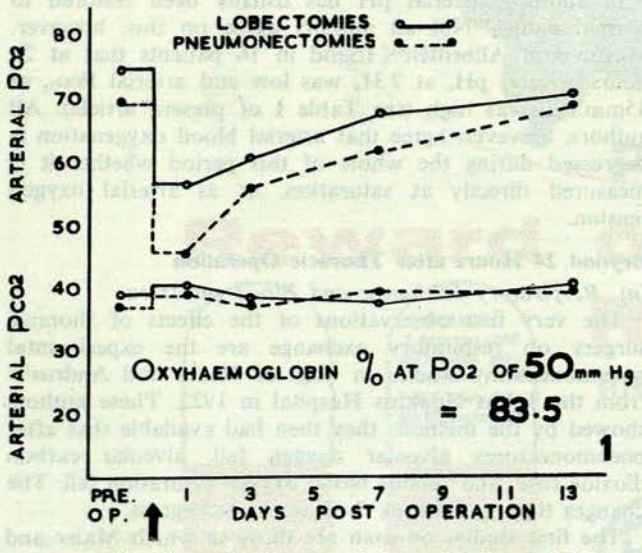

Fig. 1. This figure is constructed from the data of Björk and Hilty[126] on the effect of thoracic operations on arterial blood gas tensions, and gives average values for his patients before and after operation.

in arterial P_{CO_2} in the first week (2 out of 9 patients) were unable to raise the arterial P_{O_2} more than 5 mm.Hg by this manoeuvre. A graph made from these authors' data[126] is illustrated in Fig. 1. Resting values only are shown. Castellanos *et al.*,[128] using ear oximetry, were unable to produce 100% saturation in their patients even with an oxygen flow of 10 litres per minute by mask. Of their 50 patients 90% showed abnormally low values for oxygen saturation. As noted above, Linderholm and co-workers [62, 63] made similar studies in 1958 on abdominal operation patients.

A few other authors have made contributions. Siebecker *et al.*[129, 130] (1958 and 1960) found a fall in arterial oxygen saturation following thoracic operation, using a direct-reading ear oximeter, and their cases followed a similar course to those of Maier and Cournand[70] and those of Björk and Hilty.[126] They believe that the most likely cause of the fall in saturation is alveolar hypoventilation, since they claim that 3 or 4 deep breaths result in a significant rise in arterial saturation. They also discounted abnormalities in diffusion and in ventilation-perfusion ratios for various reasons.

Also in 1960, Clowes *et al.*[122] published a rather fuller study, which is one of the few studies* that give any actual measurements of ventilation, and in it the figures are only given as group data for pre- and postoperative tidal volume and respiratory frequency. No analysis of expired gas was made, so that alveolar ventilation could not be measured. Similarly, since arterial oxygen tension was not measured, no estimate of ventilation-perfusion abnormalities could be made. Like others before him, Clowes found that the respiratory acidosis of thoracic surgery had disappeared by 24 hours. Clowes and his co-workers[122] are, however, the only persons who have measured cardiac output immediately after thoracic operation. Using the dye-dilution technique and a Waters densitometer, they measured the cardiac output in 14 patients† before, during, and at 1, 2 and 7 days after operation (the injection site for the dye is not stated). From their data, the pre-operative value for cardiac output is 5·5±1·0 litres per minute. Postoperatively the values (in % of pre-operative value) are 119% at 1 day, 135% at 2 days and 119% at 1 week. Finally, in 1961[123] another paper appeared from Scandinavia in which 16 tuberculosis patients were investigated. This paper confirmed some of the findings discussed above.

(*b*) *Lung Volumes and Mechanics*

Smith *et al.*[131] took 50 patients undergoing thoracic operations and 25 undergoing non-thoracic non-abdominal operations, and studied them daily for the first week after operation by ear oximetry and spirometry. They found that in the thoracic group the vital capacity on the first postoperative day was only 50% of the pre-operative value, and that over the course of the first week it slowly rose to around 60% of that value. The one-second fraction ($FEV_1/FVC\%$) was unaffected, remaining at pre-operative levels, but the maximum expiratory flow rate[132] was re-

*The only other figures for postoperative ventilation are to be found in the paper on diffusing capacity by Schramel *et al.*,[9] where in the four patients without shunts the pre-operative figures and postoperative figures at 24 hours for P_{CO_2} and V are the same, and in the paper of Scheinin,[39] which is discussed later.

†14 patients are quoted in the text, but 15 appeared in the tables.

duced by 55%. The non-thoracotomy patients had a transient fall of 10% in vital capacity on the 1st post-operative day, but this had gone by the 2nd day. The other tests were unaffected by the surgical procedure. They interpret these findings, as they do their earlier studies, in terms of hypoventilation.

Gorlin *et al.*[81] made a study starting at the 7th post-operative day in patients undergoing minor pulmonary operations and found that the vital capacity had returned to normal in 13 out of 23 in 2 weeks and 22 out of 23 in 5 weeks. They found that residual volume was essentially unchanged by operation, so that total lung capacity followed vital capacity. It is difficult to interpret their figures for maximum breathing capacity (MBC), because the figures are quoted in terms of predicted normal values and, while the MBC fell postoperatively in all patients, in only 12 was it below the predicted normal values. Their figure 3, however, suggests that it remained depressed in some patients for longer than 6 weeks.

Scheinin,[39] whose work in lung volumes after abdominal operations is quoted above, has also made a similar extensive study in patients who have undergone thoracic operations. His results in 66 subjects show that the total lung capacity is almost equally reduced on the 2nd post-operative day after pneumonectomy (41% of pre-operative value), lung resection of lesser degree (45%), or simple thoracotomy (40%). Recovery of pre-operative values is of course incomplete; the value in thoracotomy had only reached 66% of the pre-operative value by the 20th post-operative day. This fall in total lung capacity (TLC) after thoracic operation is due to a fall in both vital capacity (30 - 34% of pre-operative value) and residual volume (54% of pre-operative value in pneumonectomy, 68% in lung resection, and 60% in thoracotomy). These results, by the closed-circuit helium-dilution technique, are in contrast to those in the smaller series of Gorlin *et al.*,[81] who used the open-circuit nitrogen-washout method.

Scheinin[39] also measured expired minute volumes and found that, in contrast to his results after abdominal operations, there was a statistically significant postoperative rise in ventilation after thoracic operations (109% of pre-operative value—$P<0.01$). This value was still significantly elevated at 10 days but not at 20 days. As in other series of either type of post-operation patients, respiratory frequency was raised (134% on the 2nd postoperative day) and tidal volume diminished (81%). The rise in frequency lasted 10 days but the fall in tidal volume was still present at the 20th day ($P<0.05$). The figures for postoperative maximum breathing capacity were very similar (41 - 43% of the pre-operative values) to those for total lung capacity.

He also found[39] that on the 2nd to 5th day the end tidal oxygen tension was slightly reduced, at 98% ($P<0.001$) of the pre-operative values, but end tidal carbon-dioxide tension was unchanged (103%). Oxygen consumption was raised for 10 days ($P<0.01$), while the ventilatory equivalent for oxygen was unchanged. Since the rise in postoperative oxygen consumption (109%) was exactly equivalent to the rise in ventilation (also 109%) at this time, he explained the rise in minute ventilation in terms of altered metabolism.

Scheinin's final contribution in this paper was a careful study of the effects of opiates and local analgesia on post-operative respiratory volumes. He was able to increase the postoperative figures for vital capacity by significant though unimportant amounts (100 - 200 ml.) and reduce the residual volume by 30 ml. ($P<0.01$). The MBC could be increased by 5 litres per minute, which was not significant. He has also shown[133] that postoperative pulmonary complications aggravate the disability and delay the patient's return to normal after thoracic operations just as after abdominal operations.[41]

CONCLUSIONS AND SUMMARY

It is well established that after *thoracic operations* there is considerable interference with the mechanical function of the lungs, with reduction of vital capacity to about half the pre-operative value, and also reduction amounting to about one-third of the pre-operative value in the residual volume. These findings are practically independent of the type of thoracic operation performed. The following are other postoperative developments after thoracic operations:

Respiratory frequency is increased and tidal volume diminished, but there is a significant increase in minute ventilation, which correlates well with a postoperative increase in oxygen consumption.

Carbon dioxide output is unchanged.

Twenty-four hours after operation the acid-base balance of the body is normal except where there has previously been compensated respiratory acidosis.

There is, however, some oxygen desaturation of arterial blood in most cases as compared with pre-operative values. This has been shown to persist even with 100% oxygen breathing, and has been variously attributed to impaired ventilation, atelectasis, and alveolar hypoventilation. Further work is necessary to define the cause of the arterial unsaturation.

Following *both abdominal and thoracic operations* there is a considerable immediate reduction in ventilatory mechanical ability and a fall in total lung capacity. These changes are minimally affected by the relief of post-operative pain. They are not the effect of anaesthesia.

Ventilation (as measured by alveolar or arterial carbon-dioxide levels and the acid-base balance) is restored to normal within 24 hours of surgery. In spite of this there is oxygen unsaturation of the arterial blood of the order of 5 - 10%. This is present from the end of anaesthesia, and regresses gradually over 1 - 2 weeks. The mechanism of this fall in saturation is not clear. It has been attributed to alveolar hypoventilation and to atelectasis.

After 2 - 3 weeks pulmonary function is normal in patients who have undergone abdominal operation, provided there has been no postoperative pulmonary disease. In those that have had a thoracic operation, pulmonary mechanical function gradually improves over a much longer period, but studies of resting respiratory exchange 3 - 4 weeks after the operation show that this has returned to normal by this time both at rest and on exercise.

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