IMMEDIATE POSTOPERATIVE CARE, WITH PARTICULAR REFERENCE TO BLOOD-GAS STUDIES

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WITH INCREASED STANDARDS of training, and improvements in anaesthetic techniques and drugs, anaesthetists now pride themselves that patients can usually be brought successfully through the most difficult of operative procedures. Where problems arise in greater incidence is in the immediate postoperative period. It is here that the anaesthetist must play an even more active role in the future than he is doing at present.

Recovery rooms, or postanesthetic areas, which allow postoperative patients to be cared for by specially trained nurses, were an outgrowth of the Second World War. Our responsibility as anaesthetists in this area was pointed out early by Dripps and Waters, who emphasized, in a connotation called the "Stir-up" regime, the importance of turning the patient frequently, having him cough at regular intervals, and urging him to breathe deeply.¹ Recognizing the difficulty of obtaining the co-operation of the patient to breathe deeply, they suggested the intermittent inhalation of carbon dioxide, an idea which in recent years has made every anaesthetist recoil in horror. They also recommended the continuous administration of oxygen, where indicated, by means of a catheter inserted through the nose into the oropharynx, a technique which has stood the test of time.

In the last 20 years, the role of adequate ventilation postoperatively has been stressed, with particular emphasis on carbon dioxide elimination. Mechanical ventilators have been developed, and the indications for their use have multiplied as the importance of preserving gas exchange to and from the lungs has been recognized. Hand in hand with this alertness has been the development of objective means of determining the efficacy of respiratory exchange. Reliable laboratory tests, which are not technically difficult to perform, have allowed us for several years to determine the pH and carbon dioxide tension, more or less at the bedside. With special equipment it has also been possible to determine the arterial oxygen saturation. But the saturation merely indicates how much oxygen is being carried in the blood as a percentage of that which the blood is capable of carrying under normal circumstances. A much more accurate measurement is the oxygen tension, which indicates the partial pressure of oxygen in the arterial blood, and therefore the availability or ease with which oxygen can be presented to the tissues. Within the last two to three years, as a result of the development of oxygen electrodes, it has for the first time become possible to measure under clinical circumstances the oxygen tension existing in blood. This breakthrough is of vital importance in the care of patients.

This paper is concerned primarily with the arterial oxygen tensions found in

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patients in the immediate postoperative period these determinations have been correlated with the ability of patients to eliminate carbon dioxide. It is important first to determine what the normal arterial oxygen tension is. The textbooks of physiology have told us that this figure is 100 mm Hg with the patient breathing room air. However, Lambertsen² indicates that the normal value for arterial oxygen tension is 95 mm Hg. More recently Raine and Bishop³ have shown that age and position are factors in the normal values for oxygen tension. In subjects less than 40 years of age, the mean arterial oxygen tension while breathing air was 97.6 mm Hg in the sitting position and 95.3 mm Hg in the supine position. In subjects over 40 years of age, the mean paO_2 was 90.0 mm Hg in the sitting posture and 88.2 mm Hg while supine. In our own institution, the arterial oxygen tension in 14 patients while lying supine in the ward awaiting elective surgical procedures averaged 80.4 mm Hg, with a range between 100 mm Hg in a 28-year-old patient to 57.5 mm Hg in a 68-year-old patient (Table I). The average age of these patients was 47.3 years, with a range between 28 and 68 years. It is apparent that the "normal" arterial oxygen tension is unknown, and indeed may vary from patient to patient.

TABLE I

	Age	Sex	pH	$paCO_2$ (mm Hg)	paO_2 (mm Hg)
1	28	F	7.40	43.2	93.1
2	28	F	7.42	30.7	100.0
3	38	F	7.41	39.6	87.4
4	39	M	7.43	36.4	63.2
5	45	F	7.44	35.6	70.1
6	46	F	7.46	30.4	74.7
7	46	F	7.39	39.2	88.5
8	46	F	7.44	37.3	93.1
9	48	F	7.46	43.4	74.7
10	51	M	7.44	37.3	75.9
11	55	M	7.39	41.3	71.3
12	60	M	7.42	44.8	80.5
13	64	F	7.41	37.2	96.6
14	68	M	7.40	37.2	57.5
Av	47.3		7.42	38.1	80.4

In our hospital random determinations of blood-gas values during and after operation in over 200 patients indicated that great variations could occur, depending on the physical status of the patient, the type of operation being performed, and the care with which the anaesthetic was being administered. It became obvious that adequate elimination of carbon dioxide from the lungs did not necessarily ensure a normal arterial oxygen tension, thus perhaps reaffirming the old physiological doctrine that it is 20 times easier to get carbon dioxide across the alveolar membrane than it is oxygen. For example, in a 77-year-old woman undergoing subtotal gastric resection (Fig. 1), it was not possible to obtain a normal arterial oxygen tension, even though ventilation was sufficient to produce a definite respiratory alkalosis. In another patient, a 23-year-old man having a closure of a transverse colostomy (Fig. 2), oxygen tensions were above normal during operation, but when he was allowed to breathe room air in the

Age 77 W F

Subtotal Gastric Resection

for Carcinoma

Thiopental 150 Mg N₂O, O₂, Ether

Succinylcholine 100 Mg

<u>Time</u>	<u>pH</u>	<u>pCO₂</u>	<u>PO₂</u>	
1 44	7 48	34 5	67	Beginning Surgery Spon Resp B P 160/90
2 20	7 64	20 0	50	Cont Resp B P 120/80
2 45	7 53	20 5	60	Cont Resp B P 120/80
3 08	7 65	18 5	56	Cont Resp B P 140/80
3 27	7 65	17 0	51	Cont Resp B P 160/90
3 50	7 62	17 5	67	Cont Resp B P 180/100

FIGURE 1 Arterial blood-gas values in a 77-year-old woman undergoing subtotal gastric resection. Note low arterial oxygen tensions in presence of induced respiratory alkalosis.

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Age 23 M C

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Gunshot Wound Abdomen

Closure Transverse Colostomy

Thiopental 250 Mg Gallamine 80 Mg N₂O, O₂ 2 5 1 5 Ether

<u>Time</u>	<u>pH</u>	<u>pCO₂</u>	<u>PO₂</u>	
10 05	7 39	40 5	118	During Surgery Asst. Resp
11 04	7 43	34 6	117	End of Surgery Breathing O ₂
Recovery Room				
11 15	7 37	40 1	63	Room Air
11 30	7 36	42 4	68	Same
11 45	7 36	42 4	67	Same
12 00	7 34	39 1	83	Same Restless

FIGURE 2 Arterial blood-gas values in a 23-year-old man having a closure of a transverse colostomy. Note reduction in arterial oxygen tensions in recovery room in spite of preservation of normal acid-base balance.

recovery room, the arterial oxygen tension was reduced to low levels, even though the pH and carbon dioxide tensions remained within normal limits.

For these reasons an objective study was undertaken to determine what happened to patients postoperatively.

Respiration By means of a Wright Respirometer and a Bennett face-mask, respiratory rates, tidal volumes, and minute volumes were determined in 20 patients the afternoon before operation (control), immediately on arrival in the recovery room, and at 10 and 30 minute intervals in the recovery room. All types of general anaesthesia were used, including ethyl ether, cyclopropane, halothane, and methoxyflurane, and the same physician performed all the measurements. Oxygen was not administered to this control group. The average minute volume preoperatively was 60 litres, and three readings postoperatively differed little (Fig 3). The average respiratory rate before operation was 17

FIGURE 3 Minute volumes (average and extremes) in 20 patients the day prior to operation, and in the immediate postoperative period

per minute, and the average tidal volume was 350 ml. These values were essentially unaltered in the postoperative period (Figs 4 and 5). Under the circumstances of this study, respiratory parameters were not grossly altered by anaesthesia and operation.

Blood gas values During anaesthesia an arterial needle was placed in the radial, ulnar, or brachial artery, and blood samples were withdrawn at various intervals in heparinized syringes under anaerobic conditions. Within 5 to 10 minutes of obtaining the sample, determinations of pH, carbon dioxide tension, and oxygen tension were made, always by the same technician, on an Instrumentation Laboratories apparatus. Corrections were made for body temperature and haematocrit.

FIGURE 4 Respiratory rates (average and extremes) in 20 patients the day before surgery and in the immediate postoperative period

FIGURE 5 Tidal volume (average and extremes) the day before surgery and in the immediate postoperative period

FIGURE 6 pH, paCO_2 , and paO_2 values in six patients who did not receive oxygen postoperatively. Note normal acid-base balance, but evidence of arterial hypoxaemia.

FIGURE 7 pH, paCO_2 , and paO_2 values in six patients who received oxygen in postoperative period. Note normal acid-base balance and satisfactory oxygen tension values.

In 6 patients followed postoperatively for 30 minutes, in whom a vigorous stir-up regime was maintained, but to whom no oxygen was given, the pH and paCO_2 were essentially within normal limits, but the paO_2 (average) was in the range of 80 mm Hg (Fig 6) A second similar group of patients to whom oxygen was administered, first by bag and mask and then by nasal catheter, showed marked increases in oxygen tension (Fig 7) The pH and paCO_2 values remained within normal limits

FIGURE 8 pH, paCO_2 , and paO_2 values obtained in six patients during anaesthesia and for 60 minutes in postoperative period patients did not receive oxygen postoperatively Note normal acid-base values, but arterial hypoxaemia postoperatively

In a third group of 6 patients (Fig 8), who were followed for 60 minutes postoperatively, an arterial blood sample was taken while under anaesthesia During surgery the average pH was 7.48, the paCO_2 was 30 mm Hg, and the paO_2 was 160 mm Hg It would appear that these patients were being adequately ventilated and oxygenated during anaesthesia However, in the postoperative period, although the pH and paCO_2 values were within normal limits, arterial hypoxaemia was again present Over a period of one hour there was a gradual increase in the paO_2 , and at 60 minutes it was approaching normal values

A fourth group of 8 patients were also followed for 60 minutes after operation, but oxygen by nasal catheter (6 to 7 litres per minute) was begun on arrival in the recovery room (Fig 9) These patients again were able to eliminate carbon dioxide satisfactorily, and the additional oxygen provided served to maintain a satisfactory oxygen tension in most instances

FIGURE 9 pH, paCO_2 , and paO_2 values obtained in eight patients during anaesthesia and for 60 minutes postoperatively. Oxygen given by nasal catheter. Note normal acid-base balance and satisfactory oxygen tensions.

The oxygen tension values obtained in the recovery room were broken down into age groups. In patients over 50 years of age who breathed only room air in the recovery room, the paO_2 was consistently just over 60 mm Hg (Fig 10). When patients of this age group were given oxygen by mask, more than adequate oxygen tensions were attained, however, with oxygen by nasal catheter, barely satisfactory tensions were found (Fig 11). In the age group under 50 years breathing room air, more adequate oxygen tensions were attained, but the average fell short of the "normal" (Fig 12). However, the administration of oxygen to this group resulted in high oxygen tensions, even when administration was by nasal catheter (Fig 13).

DISCUSSION

The conclusions to be derived from this study are few but significant (1) The "normal" arterial oxygen tension is unknown and may vary from one person to another, depending on his physical status and age (2) The administration of anaesthesia and the performance of surgery lead to a state of arterial hypoxaemia in the immediate postoperative period (3) The reduction in arterial oxygen tension exists in the presence of a minute volume gas exchange which usually is sufficient to maintain a normal pH and arterial carbon dioxide tension (4) The administration of oxygen postoperatively serves to correct the arterial hypoxaemia

satisfactorily in most patients. It is obvious, then, that *all* patients should receive oxygen following administration of general anaesthesia.

The natural question which arises is why patients become hypoxaemic following anaesthesia and operation. The first thoughts that come to mind are the apparent ones associated with respiratory depression following the use of anaesthetic drugs, postoperative pain limiting respiratory exchange, or unusual positions in the operating room leading to inadequate ventilation. These factors cannot be ruled out, and one or more of them may play a part in individual patients. However, in this group of patients, minute volume and tidal volume exchange did not differ significantly in the resting preoperative and postoperative periods. Also, parenthetically, there was great variation in the resting preoperative oxygen tension values.

The fact that arterial oxygen tensions were lower in the older age group leads to the possibility that diffusion defects across the alveolar membrane may be responsible for the hypoxaemia. Raine and Bishop³ doubt that this explanation is valid. However, it has yet to be ruled out.

FIGURE 10 Postoperative oxygen tensions in patients over 50 years of age who breathed room air in recovery room

FIGURE 11 Postoperative oxygen tensions in age group over 50 who breathed first oxygen by mask and then received oxygen by nasal catheter. Note relatively low oxygen tensions associated with nasal catheter administration.

FIGURE 12 Postoperative oxygen tensions in age group under 50 years who breathed room air in recovery room

FIGURE 13 Postoperative oxygen tensions in patients under 50 years of age who received oxygen. Note satisfactory values at all times

Bendixen and colleagues,⁴ who have noted consistent reductions in arterial oxygen tension during and after procedures in which patients have been ventilated with "normal" tidal volumes, believe that the hypoxaemia results from small, military areas of atelectasis that develop during the course of consistent tidal ventilation. They hypothesize that, unless deep sighing respirations are provided, as the awake person does involuntarily every few minutes, physiological shunts develop through which blood flows, but which cannot become oxygenated because of the surrounding atelectatic alveoli. A similar situation could occur in the recovery room, of course, where the patient cannot or will not take deep breaths. In this respect, the old therapy of carbon dioxide inhalations to promote deep breathing may have real merit. At the present time, analeptic drugs are being investigated as pharmacologic stimulants to promote deep breathing postoperatively.⁵

Perhaps the most widely accepted explanation for the arterial hypoxaemia is that an alteration occurs in the ventilation-perfusion ratios between gas and perfusing blood in the lungs.^{6,7} In older patients with chest disease, it is recognized that inequality of ventilation-perfusion ratios is the commonest cause of arterial oxygen

unsaturation It is more than likely that certain techniques of anaesthesia may also disturb the ventilation-perfusion ratio It is known that, during normal spontaneous ventilation, most of the inhaled gas goes to the lower lobes of the lungs through which most of the blood is flowing The diaphragm plays a vital part in this movement of gas During controlled respiration, with the diaphragm quiet, a greater proportion of the gas being pushed in goes to the upper parts of the lung, thus upsetting the ventilation-perfusion ratio

Arterial hypoxaemia may also result from increases in physiological dead space, which alter the ventilation-perfusion ratio Hypotension increases the physiological dead space, and the administration of ethyl ether and halothane, by some mechanism as yet unknown, also increases this parameter

Tamlin *et al*⁸ have recently implicated atropine as a cause of arterial hypoxaemia, although they offer no reasons for their belief A possible explanation would be on the basis of atropine increasing physiological dead space The findings reported in their paper are not too convincing, in that the oxygen tensions were derived indirectly, the ages of the patients were not taken into consideration, and the variations noted in the oxygen tensions of patients with and without administration of atropine were not marked

It is apparent that the cause of arterial hypoxaemia in the postoperative period is unknown it may be a summation of several factors in some patients Nor is the duration of this abnormality known Much information remains to be collected and interpreted one predicts that, with the newer laboratory techniques now available, great activity will be seen in this area in the next 10 years

SUMMARY

1 The present concept that the "normal" arterial oxygen tension is 100 mm Hg may have to be altered In patients being prepared for elective surgical procedures, the average oxygen tension was 80.4 mm Hg

2 In the immediate postoperative period, arterial hypoxaemia was present in all patients who were allowed to breathe room air At the same time, the pH and carbon dioxide tensions of the arterial blood were within normal limits

3 Patients in the age group over 50 showed greater degrees of arterial hypoxaemia than those under 50

4 The administration of oxygen by mask or nasal catheter postoperatively corrected the low arterial oxygen tensions All patients should receive oxygen in the postoperative period

5 The most likely explanation for the arterial hypoxaemia is a disturbance in the ventilation-perfusion ratio in the lungs

RÉSUMÉ

1 Il faut changer le concept actuel que la tension "normale" de l'oxygène artériel est de 100 mm de mercure Chez les malades préparés pour la chirurgie élective, la tension moyenne de l'oxygène était de 80.4 mm de mercure

2 Durant la période postopératoire immédiate, il y avait de l'hypoxémie

artérielle chez tous les malades qui respiraient l'air de la pièce. Au même moment, le pH et les tensions d'acide carbonique du sang artériel se tenaient dans les limites de la normale.

3 Les malades de plus de 50 ans ont présenté une hypoxémie artérielle plus importante que ceux de moins de 50 ans.

4 L'administration d'oxygène par masque ou cathéter nasal a corrigé les basses tensions d'oxygène artériel. Tous les malades devraient recevoir de l'oxygène durant la période postopératoire.

5 L'explication la plus vraisemblable de cette hypoxémie artérielle est qu'il se produit un trouble dans le rapport ventilation-perfusion dans les poumons.

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