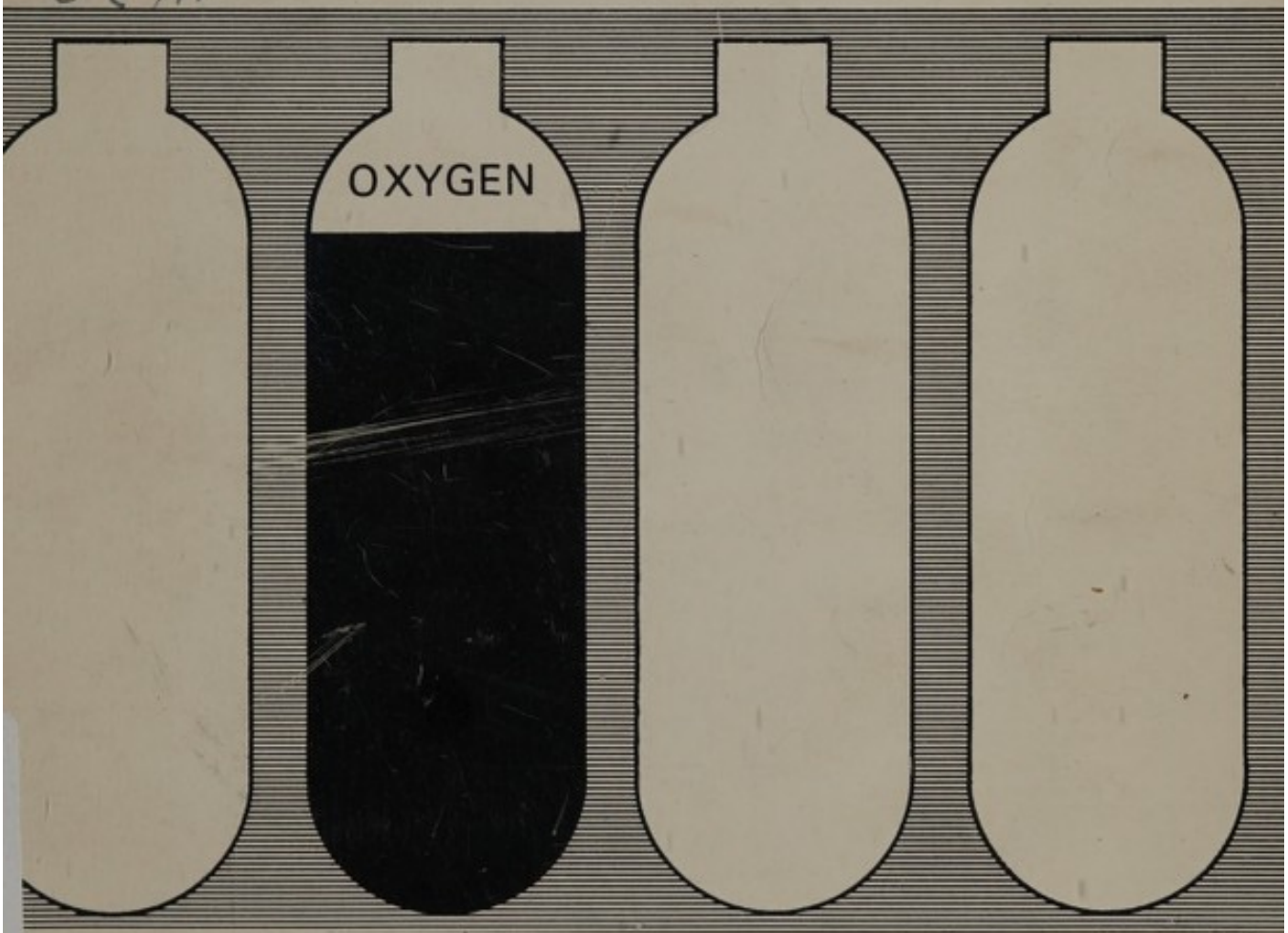
Uses and dangers of oxygen therapy : report of a sub-committee of the Standing Medical Advisory Committee.

SCOTTISH HOME AND HEALTH DEPARTMENT

SCOTTISH HEALTH SERVICES COUNCIL

Uses and Dangers of Oxygen Therapy



Report of a Sub-Committee of the Standing Medical Advisory Committee

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SCOTTISH HEALTH SERVICES COUNCIL

USES AND DANGERS OF OXYGEN THERAPY

Report of a Sub-Committee of the Standing Medical Advisory Committee

EDINBURGH
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* Dr Wilson died in September, 1966.

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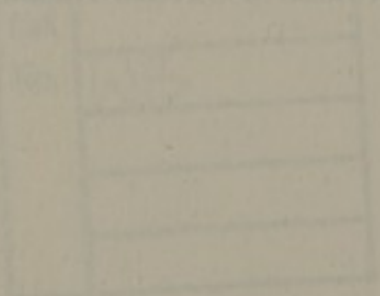

CHAPTER 1.

Introduction

1. We were appointed as a Sub-Committee of the Standing Medical Advisory Committee in August, 1965, "to examine and advise on the uses and dangers of oxygen therapy". Our first meeting was on 4th November, 1965, and in all we have held eleven meetings.

2. The breadth of our remit made it necessary to divide our studies of oxygen therapy into nine parts, namely: adult hospital practice, domiciliary practice, obstetric practice, paediatric practice, air travel, resuscitation, industrial practice, treatment of drowning and apparatus for oxygen administration.

3. There are, among our members, specialists in one or more of these fields whose interest, knowledge and experience have allowed us to examine each of these subjects in considerable depth. However, inevitably, wide consultation with acknowledged experts has also been necessary. It became apparent at an early stage in our deliberations that our report, if it were to deal adequately with our remit, would be longer and more detailed than the majority of reports of similar Sub-Committees. In view of the continuing debate and difficulty in obtaining and collating relevant material in a number of aspects of oxygen therapy, we have felt it necessary to make extensive reference to the work and publications of appropriate authorities.

4. We have endeavoured to make this report comprehensible, not only to medical practitioners, but also to workers in related professions and organisations who have responsibility and interest in situations where sick or injured people may require oxygen therapy. To assist in this direction we have included a chapter introducing the physiological basis of oxygen therapy.

5. We invited a large number of people to help us in our task and we have received both oral and written evidence from them. Their unstinting and enthusiastic support has been invaluable and we wish to record our gratitude to them. Their names are given in Appendix I. We are however, to be held entirely responsible for any conclusions drawn or recommendations made in the Report.

6. We would like particularly to mention the following on whom heavy demands were made: Dr J. E. Cotes, Professor K. W. Cross, Dr G. S. Dawes, Wing Commander J. Ernsting, Dr M. A. Heasman, Mr J. Kirkwood, Dr I. McA. Ledingham, Surgeon Rear Admiral S. Miles, Dr A. S. R. Peffers, Dr H. Simpson, Dr I. H. Stokoe, Dr C. H. M. Walker and Professor J. Walker.

7. The Chairman and other members of the Sub-Committee wish to place on record their particular appreciation of Dr D. C. Flenley's outstanding contribution to all aspects of the work involved in the preparation of this Report. He has shown throughout an unflagging enthusiasm and no task has been too heavy for him.

8. We deeply regret to record the death of our Medical Secretary, Dr A. L. Wilson, in September, 1966. His excellent work in the early stages of preparation of the Report was of great assistance to us.

9. We wish to express our gratitude to the two Secretaries — Dr D. M. Pendreigh, who took over the duties of Medical Secretary, and Mr I. G.

Dorward, Lay Secretary, for their loyal and skilled support. We also wish to record our gratitude to the Royal Infirmary of Edinburgh and Associated Hospitals Board of Management and the Board of Management for West Lothian (Bangour) Hospitals for allowing Mr Dorward to undertake the duties of Lay Secretary.

CHAPTER 2.

The Physiological Basis of Oxygen Therapy

10. Oxygen is used in man to enable oxidative metabolic processes in the individual body cells to produce the energy which is essential to maintain life. The aim of oxygen therapy, therefore, must be to ensure a sufficient supply of oxygen to the individual cells of the body. It follows that an appreciation of the normal mechanisms involved in the carriage of oxygen from the air to the cells is essential if oxygen therapy is to be employed in a rational manner.

11. The atmospheric air contains 20·94% oxygen, and even in city streets during a rush hour, this scarcely ever falls below 20%. The actual concentration of oxygen, in terms of the number of molecules of free oxygen in a given volume of a gas or a liquid, is measured by the partial pressure of oxygen, which is abbreviated to PO_2. The PO_2 in a gas is easily calculated by multiplying the percentage concentration of oxygen by the total pressure of the gas. In the case of moist air, this is 20·94% multiplied by the barometric pressure minus the vapour pressure of water. Thus, at sea level, the partial pressure of oxygen, or PO_2, of moist atmospheric air is usually about 150 mm Hg. At high altitude, although the air still has 20·94% of oxygen, the PO_2 falls, as the barometric pressure is less. For example, at 10,000 feet the PO_2 of moist atmospheric air is about 100 mm Hg. The concentration of carbon dioxide can also be expressed as a partial pressure (PCO_2) in mm Hg., by a similar calculation.

12. The function of the lungs can be briefly described as that of adding oxygen to the blood, and removing carbon dioxide (the major gaseous waste product of the body) from the blood. Air is drawn into the lungs by contractions of the intercostal muscles and diaphragm, and this inspired air, which has the PO_2 of moist atmospheric air, passes into the lung alveoli. These are provided with a rich blood supply, and it is in the alveoli that exchange of oxygen and carbon dioxide between gas and blood occurs. This gas exchange depends upon the solubility of the gas, and follows the laws of diffusion which state that gas molecules, both of CO_2 and O_2 move from a zone of high partial pressure to one of lower partial pressure. In the case of oxygen, gas moves from the alveolus to the blood in the pulmonary capillary, which is the blood vessel supplying the alveolus. Carbon dioxide, again directed by the same physical laws of diffusion, moves in a converse direction from blood to the alveolar gas. Carbon dioxide is 24 times more soluble in water than is oxygen, and transfer through aqueous media is therefore more rapid.

13. Once dissolved in the blood, oxygen undergoes a chemical combination with haemoglobin. The degree of chemical combination of oxygen and haemoglobin (to form bright red oxyhaemoglobin) depends upon the partial pressure of oxygen in the blood, so that at a PO_2 of 100 mm Hg., approximately 97% of the available haemoglobin will be converted to oxyhaemoglobin and the arterial blood will then contain about 18 ml. of oxygen/100 ml. blood, in normal man. On the other hand, at a PO_2 of 30 mm Hg., only about 60% of the haemoglobin will be chemically combined with oxygen. The relationship between the degree of saturation of the haemoglobin with oxygen and the blood PO_2 is very complex, being described graphically by "the oxyhaemoglobin dissociation curve" (Fig. 1 on page 4). The exact shape of this curve depends

upon such factors as the acidity of the blood and the body temperature, and it can be altered in those disease states associated with abnormal pigments in the blood.

14. It follows that the amount of oxygen carried in the blood will depend both upon the partial pressure of oxygen in the blood and upon the amount of haemoglobin available. Thus in anaemic patients with insufficient haemoglobin concentrations, the blood will carry less oxygen although the arterial PO_2 is nearly normal. In addition to the oxygen carried in chemical combination in blood (as oxyhaemoglobin), oxygen is also carried in physical solution. The amount of oxygen in the blood in this form is very small, but this oxygen in solution does increase in simple proportion to the partial pressure of oxygen in the blood. Thus if PO_2 is raised to very high levels, appreciable amounts of oxygen can be carried in this form; this is one of the principles underlying the use of hyperbaric oxygen therapy, where the patient breathes oxygen in a special chamber in which the pressure can be raised well above normal atmospheric pressure.

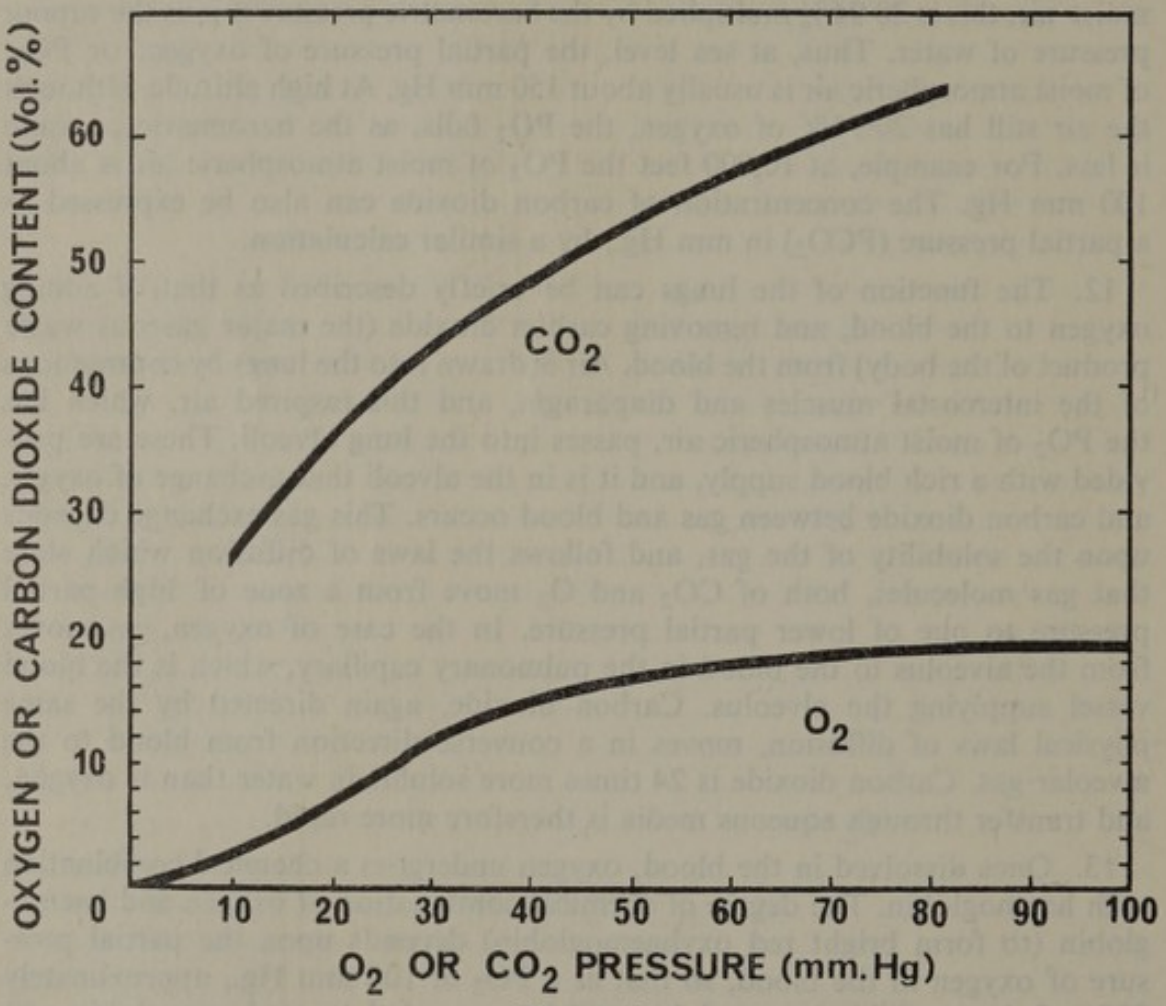

FIGURE 1

This diagram shows the amount, expressed as volumes per 100 ml., of oxygen and carbon dioxide absorbed and carried by the blood at different pressures (tensions) of these gases. Most of this gas is in loose chemical combination and only a little is in solution. Both these relationships are influenced by a number of factors, i.e. the amount of oxygen carried is affected by the acidity of the blood; the amount of carbon dioxide carried is affected by the degree of oxygenation of the blood. These variations are not depicted here for the sake of clarity.

Important points to note are:

Oxygen: The blood approaches near saturation (about 97% of maximum amount carried) at 90 mm Hg. This is approximately the normal pressure of oxygen in a healthy lung. Further rises of oxygen tension, which would occur with extreme hyperventilation or oxygen therapy only causes a very slight increase of the oxygen carried.

The blood carries well over 80% of its total capacity for oxygen at as low an oxygen tension as 50 mm Hg. which is nearly half the normal lung and arterial blood gas tension. At lower oxygen tensions, the amount carried falls rapidly. These properties give a considerable safety margin in the uptake of oxygen and facilitate its liberation to the tissues of the body.

In severe respiratory failure the oxygen tension in the blood leaving the lungs and entering the arteries is usually 50 mm Hg. or below. If it continues to fall, then dangerously low amounts of oxygen are carried in the blood. Controlled oxygen therapy, say 30%, raises the oxygen tension only moderately (55 to 80 mm Hg.) but this moderate increase causes a very marked rise in the oxygen content. Thus grave hypoxaemia (inadequate oxygen in the blood) is relieved, but undesirably high or abnormally high levels of oxygenation are avoided.

Carbon dioxide: It will be seen from the diagram that the blood has a far greater capacity for carbon dioxide than oxygen at any particular pressure (approximately three times greater). Further, the carbon dioxide content of the blood rises or falls in a manner approximately proportionate to the tension of carbon dioxide over the whole range here illustrated. Consequently the carbon dioxide content of the blood leaving the whole or part of the lungs falls or rises markedly with over or underventilation of the lung, responding faithfully to changes in lung carbon dioxide tension. This is in contrast to the oxygen content of the blood leaving the lung which, although falling markedly with underventilation of lung (low oxygen tension), is little affected by overventilation or oxygen therapy which cause a rise of oxygen tension from normal to supernormal levels.

15. The efficiency with which the lungs perform their function of adding oxygen to the blood can be assessed by measuring the partial pressure of oxygen in the blood leaving the lungs; this is the blood which enters the left heart and then flows in the systemic arteries. It can be sampled by arterial puncture, a simple and safe procedure in every day use in investigation of chest diseases in hospital patients. An arterial PO_2 below 70 mm Hg., when a patient is breathing air at sea-level, is abnormal. There are four mechanisms which may result in a fall in the arterial PO_2 below these normal levels, in a patient breathing air at sea level.

Ventilatory Failure

16. The patient may not be ventilating his lungs with a sufficient amount of fresh air. This results in a reduced elimination of carbon dioxide in addition to a deficiency of oxygenation, so that the arterial PCO_2 is increased above normal (35–45 mm Hg.) and the arterial PO_2 reduced below normal. This situation is known as ventilatory failure; it can arise suddenly from acute obstruction of the airway, in drowning, electrocution, or from overdosage with narcotic drugs, or from poisoning by agents producing muscular paralysis. Ventilatory failure can also develop acutely in the course of poliomyelitis, polyneuropathy, in acute diseases of muscle, myasthenia gravis, and as a complication of crushed chest injuries. The most common type of ventilatory failure in clinical practice in Britain arises as a complication of chronic bronchitis and emphysema. In these patients an exacerbation of a pre-existing chest infection can easily precipitate profound hypoxia and carbon dioxide retention. Indeed many of these patients with chronic bronchitis develop a state of chronic ventilatory failure, which although punctuated by exacerbations, may continue for many years, even though ultimately fatal. The recognition of ventilatory failure carries important implications for oxygen therapy and this is discussed in more detail in Chapter 3.

Ventilation Perfusion Imbalance

17. In practice, a low arterial PO_2, in a patient breathing air at sea level, results in most cases from improper mixing of gas and blood in the lungs; this is described as ventilation perfusion imbalance. If venous blood passes from the pulmonary artery to an alveolus (smallest unit of lung) which receives relatively little or no ventilation, the blood leaving that alveolus will contain more carbon dioxide and less oxygen than blood from an alveolus which is adequately ventilated. Conversely, if venous blood passes to an overventilated alveolus, blood leaving such an alveolus contains less carbon dioxide than normal but very little extra oxygen, for the haemoglobin is in effect fully saturated at any PO_2 above 100 mm Hg., and the higher alveolar PO_2 resulting from overventilation adds very little more oxygen to the blood. Thus overventilation in one area cannot compensate for underventilation in another as regards blood oxygenation (Figure 1). The overventilated alveolus contributes to the wasted ventilation, or dead space effect of the conducting airways, in which no gas exchange occurs. A further increase in dead space is caused by non-perfused but ventilated alveoli. The underventilated alveolus contributes to the venous admixture effect or the addition of relatively unoxygenated blood to blood leaving the lungs. The remaining alveoli with a proper balance of blood flow and ventilation constitute the area available for efficient transfer of respiratory gases. In normal lungs, when a man is upright, the apex receives more ventilation and less blood flow than the bases, where the converse applies. This division into three compartments, dead space, venous admixture, and ideal functioning alveoli, allows a convenient theoretical description of the behaviour of the lungs as organs of gas exchange. In reality there is a spectrum of ventilation to perfusion ratios in different alveoli which contributes considerably to the small difference in PO_2 between alveolar gas and arterial blood in a normal man. In lung disorders, such variations are greatly increased resulting in hypoxaemia (low arterial blood oxygen), but the arterial PCO_2 in these cases is normal, or even below normal unless there is a decrease in overall ventilation in addition to a disturbance of ventilation to perfusion ratios. Hypoxaemia due to underventilation of perfused alveoli is corrected by the administration of 30% oxygen which serves to distinguish this from hypoxaemia arising from the shunting of blood through abnormal anatomical channels. The hypoxaemia seen in pulmonary oedema, bronchial asthma, pneumonia, cardiogenic shock, and in chronic bronchitis and emphysema, is thought to result largely from ventilation perfusion imbalance, although other factors, such as overall hypoventilation may later complicate the picture.

Shunting of Blood

18. The third mechanism producing a low arterial PO_2 is the shunting of blood from the right heart to the left heart, without the blood being exposed to alveolar gas. This blood retains the low oxygen content, and high CO_2 content of mixed systemic venous blood, and therefore it lowers the arterial PO_2. However, due to very great capacity of the blood for carbon dioxide such shunts have little effect on the arterial PCO_2. Shunting can occur through abnormal anatomical channels outside the lung, as in the right to left shunts of cyanotic congenital heart disease, or within the lung as in the rare pulmonary arterio-venous malformations. Intra-pulmonary shunting has also recently been recognised as the cause of the low arterial PO_2 seen in some patients with

cirrhosis of the liver. The hypoxaemia arising from shunts is not fully corrected by inhalation of 30% oxygen.

Interference with Gas Transfer

19. Impairment of diffusion is another mechanism which may explain hypoxaemia, when breathing air at sea level, but it is not known to be relatively infrequent in practice. If the alveolar membrane is thickened, the path for diffusion of CO_2 and oxygen molecules between pulmonary capillary blood and the alveolus is increased. In the early 1950's it was thought that impairment of diffusion could account for the hypoxaemia seen in patients with damage to the alveolar walls from many different causes. It was called the "alveolar-capillary block syndrome" and although it cannot be denied that some hindrance to oxygen transfer does occur in these cases, more recent physiological studies have strongly supported the original observation (Donald, Renzetti, Riley & Cournand, 1952) that ventilation perfusion imbalance is very marked in these cases, and this is now considered to be more important than diffusional block in causing their hypoxaemia (Finley, Swenson & Comroe, 1962). Nonetheless the alveolar capillary block syndrome remains a useful concept and these patients do share a clinical and functional pattern of abnormality which include hypoxaemia aggravated by exercise, often a decreased level of arterial PCO_2 when at rest, and an abnormally low value for the gas transfer factor. A low value for the transfer factor, which is measured by the carbon monoxide method (Cotes, 1965), is not pathognomonic for the alveolar-capillary block syndrome, for low values are also seen in chronic bronchitis and emphysema, where the size of the effective blood gas interface is reduced. A low transfer factor and hypoxaemia will also occur when acute pulmonary disease, due to pneumonia, infarction, etc., is superadded to a patient whose pulmonary reserve is already compromised by a pre-existing chronic disorder. In this situation a further reduction in the area for effective transfer of gases between blood and alveolus can be critical.

20. Carbon dioxide is the most powerful stimulant to breathing that we know. In a normal person, an increase in the arterial PCO_2 produces enough increase in breathing to "blow off" the excess very quickly, and restore the normal values. To find a raised arterial PCO_2, therefore, at once indicates that the mechanisms which control breathing are not functioning normally. As mentioned in paragraph 16 this is seen most frequently in patients with long-standing chronic bronchitis and emphysema. In these patients it is commonly taught that the main stimulus to breathing arises from oxygen deficiency, as they no longer respond in a normal manner to their raised arterial PCO_2. More detailed studies have recently shown that some patients may have a reduced ventilatory drive from both hypoxia and from excess carbon dioxide, when compared with normal people (Flenley and Millar, 1967). Nonetheless it remains an important principle of oxygen therapy that a profound decrease in ventilation, with accumulation of dangerous levels of carbon dioxide, can frequently follow the administration of high concentrations of oxygen to these patients. This situation is known as "carbon dioxide narcosis".

21. Oxygen in the blood leaving the lungs has still to be delivered to the individual cells of the body. This blood circulates to all parts of the body and is distributed to the cells by successive branching of the arteries until tissue capillaries are reached. Blood in these capillaries gives up oxygen to the body

cells, the final stage in oxygen transport again being one of diffusion, with oxygen molecules coming out of chemical combination with haemoglobin, passing into solution in the blood plasma, then through the capillary walls and into the cells, where oxygen is consumed in the chemical processes of life. By the laws of diffusion, the cells which lie near to a capillary receive an abundant oxygen supply, whereas the supply to cells remote from the capillary is less secure.

22. It follows that those tissues of the body with a rich capillary network and plentiful blood flow are less at risk from dangers of oxygen deficiency if the partial pressure of oxygen in the arterial blood falls, than those tissues in which the blood flow is restricted. As delivery of oxygen from the capillary to the cell depends upon diffusion, an increase in the partial pressure of oxygen in the capillary blood increases the amount of oxygen which can be transferred. This concept provides another theoretical advantage for hyperbaric oxygen therapy. If the capillary PO_2 is raised to very high levels by hyperbaric techniques, it has been suggested that oxygen delivery to cells can be restored to normal, even in areas where the blood supply has been impaired by disease. Unfortunately, the practical application of this theory is hindered by the fact that very high levels of oxygen partial pressure appear to reduce the blood flow in many vascular beds and their capillaries. This fact, combined with the limitations imposed by the low solubility of oxygen in plasma, has made hyperbaric therapy in ischaemic diseases less successful than was at first hoped.

23. When oxygen is finally delivered to the cell, intact cellular enzymes are essential for its utilisation. A final barrier to oxygen use can arise if the cellular enzymes are poisoned, as with cyanide, and in a different way by dinitrophenol and even high levels of asprin (Smith, 1963). It will be obvious that oxygen therapy, which only increases the arterial oxygen partial pressure, can do little to relieve these situations.

24. In addition to the provision of oxygen for metabolic use by the cells, the inhalation of high concentrations of oxygen at one atmosphere can be useful in removing accumulations of other gases in body spaces, as in ileus, (a disease state with dilated, gas filled bowel) or in the tissues as in surgical emphysema (escape of air or gases into tissues). If an arterial PO_2 of the order of 600 mm Hg. is achieved by breathing about 95% oxygen little extra oxygen is carried by the blood. As this blood passes through the capillaries and oxygen is removed by metabolism, the PO_2 in the blood falls very markedly to about 60 to 80 mm Hg. in the venous blood. At the same time carbon dioxide is added in roughly equal amount to that of the oxygen removed. However, due to the difference in slope of the blood dissociation curves for these two gases, the increase in PCO_2 in venous blood over that of arterial amounts to only about 5–10 mm Hg. As a result the total pressure of gases in the venous blood (oxygen, carbon dioxide, nitrogen and water vapour) is considerably less than atmospheric. Thus gases other than oxygen pass into solution in the venous blood and are removed by the lungs.

Dangers of Oxygen Therapy

25. The dangers of oxygen therapy fall into two groups, the physical dangers and the physiological dangers. The physical dangers arise from the properties of oxygen in supporting combustion. The gas is not itself inflammable, but

any fire or spark may be highly dangerous in the presence of increased oxygen concentrations as there is inflammable material in almost all environments. Patients receiving oxygen therapy must not attempt to smoke, and no naked flame should be allowed near such patients. Oxygen under high pressure, as used in hyperbaric therapy, produces a great risk of fire, and strict precautions must be taken in a hyperbaric chamber. The exact conditions associated with such fires are not entirely clear, but recent work has emphasised the dangers of electrostatic sparks, and of clothing soiled by grease, sweat or petrol. These fires can be extremely difficult to extinguish and can produce fatal burns very rapidly. Extreme precautions must be taken in any chamber filled with pure oxygen at pressures higher than 0.4 atmosphere (Denison, Ernsting and Cresswell, 1966, Purser, 1966, Report, 1967), and it is far safer to administer hyperbaric therapy to the patient by a well-fitting oro-nasal mask, as opposed to filling the whole chamber with oxygen under pressure. The very infrequency of catastrophic accidents can be dangerous by causing insufficient attention to be paid to important and continued preventive measures. We commend for reading the detailed report produced in June, 1967 by a Working Party of the Western Regional Hospital Board on Fire Hazards in Hyperbaric Environments which examined this subject in depth. This report also recommended that the hazards involved in the use of oxygen tents and incubators should be investigated. The potential fire risk of oxygen therapy should never be forgotten.

26. The physiological dangers of oxygen therapy are four in number. The first and most common danger is the risk of producing ventilatory depression by high concentrations of oxygen. This risk arises only in patients with an elevated level of PCO_2, which is indicative of impairment of the normal control of ventilation. This occurs most often in cyanosed patients with a long history of chronic bronchitis. Such patients should receive only about 30% oxygen, at least until more sophisticated control in hospital is possible.

27. Two dangers are of more theoretical than practical importance in most medical practice. Prolonged inhalation of high concentrations of oxygen (over 60%) can produce a specific damage to the pulmonary epithelium (Lorrain-Smith, 1899) probably by inactivating the "surfactant" protein lining the alveoli which maintains the mechanical stability of the alveolar membrane. This is known as the Lorrain-Smith effect, after its discoverer. Retrosternal discomfort may occur after six hours of breathing 100% oxygen at sea level, but only after 36 hours with 60% oxygen. Lethal lung damage is invariably produced in warm-blooded animals subjected to high concentrations of oxygen for long periods. In most medical practice, this is not a major hazard of oxygen therapy as the continuous administration of such high concentrations presents technical difficulties (Chapter 11). However, patients whose treatment requires artificial mechanical ventilation may well be at risk from the Lorrain-Smith effect, for these machines often deliver oxygen enriched mixtures to patients continuously over long periods of time.

28. Another physiological danger arising from oxygen toxicity is acute poisoning of the central nervous system, causing convulsions. This is known as the Paul Bert effect, again after its discoverer. The risk occurs only when oxygen is breathed at pressures considerably greater than atmospheric. It is a very real danger in diving, and personnel engaged in hyperbaric oxygen

therapy are very aware of this risk. Although there is a wide variation in susceptibility, both from day to day and between individuals, it seems that fits from oxygen poisoning have never occurred when the partial pressure of inspired oxygen was below 1300 mm Hg. (Donald, 1947) (obtained by breathing 90% oxygen in a pressure of 2 atmospheres absolute). The effect appears to be related to the high partial pressure of oxygen on the enzymes of the brain cells.

29. In the early 1950's, retrolental fibroplasia was a leading cause of blindness in children. The disease is characterised by an obliteration of the developing vessels of the immature retina, and it particularly affects premature infants of 26 to 31 weeks gestation with birth weights between 800 and 1500 grams (Duke-Elder and Doloree, 1967). About one quarter of affected infants went on to develop permanent blindness. In 1951 Kate Campbell suggested that the high concentrations of oxygen that were routinely used in treating these babies may have been responsible for the retrolental fibroplasia. This suggestion was rapidly confirmed by clinical and experimental studies which led to the recommendation that oxygen concentrations over 40% should never be exceeded in the treatment of premature infants. The adoption of this principle was followed by a widespread decline in the incidence of the disease.

30. However, the problem of prevention of retrolental fibroplasia should be reconsidered. The respiratory distress syndrome of the new born (hyaline membrane disease) is seen particularly in premature infants. These infants may suffer from severe respiratory failure, with low arterial PO_2 and high arterial PCO_2. Concentrations of oxygen of the order of 70–90% may be needed to provide normal levels of arterial PO_2 (Warley and Gardner, 1962). There appears to be no danger of producing retrolental fibroplasia by such therapy provided that the arterial PO_2 is not allowed to rise to levels much above normal for several hours. There also appears to be no danger that high concentrations of oxygen in the air around the eye of the premature baby can cause this disease unless the arterial blood flowing to the retina contains blood with a high PO_2 (Ashton, 1964).

31. If premature babies are treated with high concentrations of oxygen it is imperative that these concentrations should be measured, and in ideal circumstances the concentration of oxygen should be adjusted so that the arterial PO_2 does not rise above normal. However, infants suffering from the respiratory distress syndrome may have a right to left shunt through a patent ductus arteriosus, which can result in a marked difference between the oxygen content in the blood delivered to the head and that delivered to the lower limbs and umbilical artery. It follows that accurate measurements of the PO_2 of arterial blood flowing to the head may be impossible. Dr G. S. Dawes has suggested to us that the only practical solution to this problem at present is to give an infant with severe respiratory distress sufficient oxygen to maintain the body temperature and oxygen consumption, and cautiously to try the effect of reducing the oxygen concentration at intervals of a few hours. Another useful measurement is the buffer base of the blood. If this is deficient it suggests a metabolic acidosis due to tissue hypoxia. If this is normal, it suggests adequate oxygenation. An alternative approach used in some centres is to ventilate the infant mechanically, and adjust the inspired oxygen concentration to maintain a normal PO_2 in blood samples from the temporal artery. The indiscriminate

use of high levels of oxygen without some form of monitoring in treatment of premature infants must continue to be recognised as carrying an unwarranted risk of producing permanent blindness.

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CHAPTER 3

Oxygen Therapy in Adult Hospital Practice

32. The foregoing description of the basic physiology of oxygen transport indicates how this can be disturbed in disease states. It concludes that the principle indication for oxygen therapy is the presence of a low arterial PO_2, which can be restored to normal by the inhalation of oxygen. It is important to realise that significant hypoxaemia is both difficult to define and difficult to recognise from clinical signs. However, the delivery of oxygen to the cells of the body also depends on the blood flow or cardiac output. In any acute clinical situation with a low cardiac output, it seems appropriate in the present state of knowledge to propose that an arterial PO_2 below 60 mm Hg. is undesirable. At the other extreme, if chronic hypoxia is associated with a cardiac output that is normal or even raised, an arterial PO_2 of at least 50 mm Hg. is a not unreasonable therapeutic aim. It must be admitted that this problem is far from solved and it is still the subject of active research (Hutchison, Flenley and Donald, 1964; Campbell, 1965; Freeman, 1966; Flenley, 1967).

33. The clinical recognition of hypoxaemia is difficult. Mental confusion, restlessness and amnesia which are corrected by oxygen therapy are often of little value as diagnostic guides in practice. An increased pulse rate which slows by 10 beats/minute on administration of oxygen would suggest the presence of significant hypoxia (Comroe, 1965), but again in clinical practice many other factors may modify this response.

34. Cyanosis, or bluish discolouration of the skin or mucous membranes, is caused by abnormal amounts of reduced haemoglobin in the small blood vessels. There are two important types of cyanosis. In *central cyanosis* the arterial blood being pumped to the body has a decreased oxygen content and contains abnormal amounts of reduced haemoglobin. In central cyanosis there is bluish discolouration of the tongue, conjunctivae on inside of the lips. Cyanosis of the skin may not be marked if the circulatory state is good and the skin is warm. This is the type of cyanosis encountered in respiratory failure and is of grave portent. The second type of cyanosis is *peripheral cyanosis* and is most common in people with heart disease where the total amount of blood pumped by the heart to the body is reduced. The blood flow to vital organs is sustained by reducing blood supply to other organs, notably the skin. The arterial blood is usually well oxygenated but the normal extractions of oxygen from the very reduced skin blood flow causes the blood in the small vessels of the skin to have a very low oxygen content and abnormally increased quantities of blue reduced haemoglobin. This condition may or may not be serious but it is little affected by oxygen therapy alone. Central cyanosis which is most reliably detected by examination of the tongue is serious, particularly in emergency situations, and can be relieved by oxygen therapy. However, the recognition of cyanosis is subject to a high observer error even in natural daylight. It is only when the arterial oxygen saturation falls to 75% (PO_2 from 36 to 45 mm Hg., depending on the arterial pH) that all observers recognise cyanosis (Medd, French, and Wylie, 1959); when the saturation is 81 to 85% (PO_2 41 to 56 mm Hg.) only half the observers record definite cyanosis (Comroe and Botelho, 1947). In the anaemic patient, hypoxaemia has to be even more

severe if cyanosis is to be detected. Abnormal blood pigments, which usually result from drug administration, can produce cyanosis without hypoxaemia. The detection of cyanosis also depends on the type of lighting; tungsten light, which is deficient in blue wavelengths, is particularly difficult (Medical Research Council, 1966). Kelman and Nunn (1966) showed that an arterial oxygen saturation below 90% (arterial PO_2 72 to 36 mm Hg. as arterial pH varies from 7·20 to 7·50) could be detected in 97·5% of cases, under suitable fluorescent lighting, if the cyanosis was observed in the lips and buccal mucosa.

35. In view of the problems in diagnosing hypoxaemia, a truly objective approach to oxygen therapy requires direct measurements of arterial blood PO_2. Although such methods are increasingly available in hospital practice, it is wrong to assume that oxygen should be given only when a low arterial PO_2 has been demonstrated in a particular patient. In this as in other branches of medical practice, rational therapy can be employed from a clinical diagnosis. This requires a knowledge of the level of arterial PO_2 which is to be expected in patients with this diagnosis. In order to extend this territory of rational oxygen therapy, more observations are needed of the incidence of low levels of arterial PO_2 in particular disease states. The recommendations for oxygen therapy given in this chapter are based so far as possible on the results of such studies reported in the medical literature.

36. If the inspired oxygen tension is below normal, the arterial PO_2 is also reduced. At sea level, the greatest dangers in this regard arises from faulty apparatus for inhalational therapy, but exposure to irrespirable atmospheres can occur in industry (Chapter 9). A notable example of the former is the Minnit "gas and air" apparatus, which has been used to provide analgesia in labour. Examples of this apparatus have been shown to deliver only 10% oxygen to the mother (Chapter 5) (Cole and Nainby-Luxmoore, 1962).

37. If the patient does not breathe enough to ventilate his alveoli adequately, the arterial blood contains too little oxygen and too much carbon dioxide. This situation is most commonly seen in patients with chronic bronchitis and emphysema, and the oxygen lack may become extreme if the patient suffers from an acute exacerbation of the underlying chest infection. In this situation, oxygen therapy carries particular dangers, for administration of high concentrations of oxygen to such patients may further reduce the breathing. As a result, carbon dioxide continues to accumulate although hypoxia is relieved, and the patient may die from respiratory acidosis. The increasing drowsiness of the patient is known as carbon dioxide narcosis (Donald, 1949; Westlake, Simpson and Kaye, 1955). The modern solution of this problem is to administer only enough oxygen to prevent death from hypoxia, but not enough to provoke serious underventilation and respiratory acidosis. This is known as controlled oxygen therapy (Donald, 1953; Campbell, 1960a). The modern technique is to administer approximately 30% oxygen to such patients, by a device such as the Edinburgh oxygen mask, the Ventimask, or by nasal catheter at a low flow rate of oxygen (Chapter 11). In severely ill patients, this therapy can be accurately controlled by repeated measurements of the arterial PO_2 and PCO_2 and the acidity of the arterial blood (Hutchison, Flenley and Donald, 1964). In less severe patients, measurements of the "mixed venous" carbon dioxide tension by a rebreathing technique is entirely adequate (Campbell and Howell, 1960). The exact details of such management are still the subject

of active research, but there can be no doubt that the widespread recognition of the dangers of uncontrolled oxygen therapy with high concentrations of oxygen (Chapter 11) has greatly reduced the incidence of carbon dioxide narcosis. Controlled oxygen therapy is but a part of the overall management of these patients, and efforts to relieve airway obstruction by assisted coughing, antispasmodics and even bronchoscopy, are vitally important. If conservative measures with controlled oxygen therapy are unsuccessful in steering a course between severe hypoxia and profound respiratory acidosis, other measures such as mechanically assisted ventilation are required (Flenley and Donald, 1967).

38. Inadequacy of ventilation can also arise from disorders of the neuromuscular mechanisms of breathing. Opiates and most hypnotics depress the central respiratory centre, and even small doses of such drugs may be very dangerous in patients with pre-existing respiratory failure due, for example, to chronic bronchitis. Serious respiratory depression, again with hypoxia and carbon dioxide retention, is not uncommon in severe barbiturate poisoning. If life is threatened from this cause, the treatment of choice is again mechanical ventilation and not with high concentrations of oxygen alone which may further depress ventilation although relieving hypoxia. There is little enthusiasm today for the so-called analeptic drugs in treating respiratory depression as a consequence of barbiturate poisoning (Matthew and Lawson, 1966).

39. Bulbar poliomyelitis, acute "infective" polyneuropathy and myasthenia gravis are probably the most common neurological disorders which may lead to serious hypoventilation with hypoxia and retention of carbon dioxide. Again, intermittent positive pressure respiration and not oxygen therapy is the treatment of choice in these situations. It is necessary to re-establish normal lung ventilation, and enrichment of the ventilating gas with oxygen is only needed if there is added lung damage. Trauma to the cervical spine, and acute myopathies involving the respiratory musculature also merit a similar approach. In chronic irreversible neurological conditions, such as motor neurone disease the physician will be reluctant to use such apparatus to prolong life in the hopeless case (Marshall, 1961).

40. In crush injuries of the chest, the outlook has been revolutionised in the past few years by the adoption of artificial ventilation with intermittent positive pressure (Griffiths, 1960). Recently, however, Reid and Baird (1965) have suggested that minor degrees of such injury could be managed by controlling oxygen therapy by frequent reference to blood gas analysis. However, in crush injury of the chest, positive pressure ventilation is not only an effective method of correcting blood gas abnormalities, but also serves the important function of splinting fractured ribs or sternum, and prevents further lung damage being caused by the respiratory muscles endeavouring to provide an adequate ventilation by their unaided and often frustrated efforts. All authorities join in the condemnation of routine administration of high concentrations of oxygen to such patients without either assisted ventilation or repetitive blood gas analysis. The development of a lethal degree of respiratory acidosis can be very insidious.

41. Lung disease is a frequent cause of arterial hypoxaemia. This is often associated with a low or normal level of arterial carbon dioxide tension for,

as has been stressed, the arterial PCO_2 is only increased in conditions on inadequate ventilation. In lobar pneumonia (Meakins and Davies, 1925), pulmonary oedema, pulmonary fibrosis, alveolar wall disease, thrombo-embolic pulmonary disease (Jones and Goodwin, 1965), atelectasis, and in many cases of bronchial asthma, although the arterial PO_2 is reduced to a greater or less degree, the arterial PCO_2 is normal or decreased. In these diseases the drive to ventilation may well be reflex as well as centrally mediated, and high oxygen tensions do not usually lead to significant falls in ventilation of the lungs. Oxygen therapy carries little, if any, danger of producing carbon dioxide narcosis in these conditions, and therapy with high concentrations of oxygen is advised.

42. Pulmonary oedema can complicate rheumatic, ischaemic or hypertensive heart disease, severe pulmonary infections, exposure to irritant gases, or arise as a consequence of head injury. When the syndrome results from heart, disease hyperventilation with a low arterial PCO_2 is often found (Fraser *et al* 1928). Although the arterial PO_2 is often reduced in pulmonary oedema from any cause (Cosby *et al*, 1957), there is little correlation between the clinical severity and the degree of arterial desaturation (Vitale, Dunlie, and Comroe, 1954). Unless the arterial blood gas tensions are known to be normal, it appears that high concentrations of oxygen should be given to all patients suffering from pulmonary oedema. But it must be pointed out that marked carbon dioxide retention has recently been described in some cases of severe pulmonary oedema secondary to heart disease. These patients who had been treated with high concentrations of oxygen and morphine were comatose (Anthonisen and Smith, 1965). Analysis of the arterial blood in a comatose patient with pulmonary oedema would appear to be wise. Morphine should be used in moderation. If the arterial PCO_2 is grossly elevated, treatment with intermittent positive pressure respiration is indicated. The striking success of controlled oxygen therapy with approximately 30% oxygen in treating acute exacerbations of chronic ventilatory failure has resulted in the rather indiscriminate use of these levels in treatment of pulmonary oedema as well. We do not recommend this practice, for in some cases of acute pulmonary oedema it results in failure to provide normal levels of arterial PO_2. Futhermore, in acute pulmonary oedema of cardiac origin, the cardiac output may be reduced resulting in severe impairment of oxygen transport if the arterial PO_2 is also below normal.

43. In chronic right ventricular heart failure complicating mitral stenosis the arterial PO_2 is usually nearly normal (Meakins, Dautebrande and Felter, 1923; Platts, 1953). These patients usually overbreathe, as shown by their low arterial PCO_2 (Curti *et al*, 1953). Oxygen therapy appears to have little place in the routine management of such patients. However, the relatively fixed low cardiac output which characterises this condition does mean that any reduction in arterial PO_2 seriously hampers the transport of oxygen to the body cells. If pulmonary emboli or acute pulmonary oedema complicate the picture, oxygen in high concentrations is strongly indicated. In right ventricular heart failure due to hypertensive or chronic ischaemic heart disease, the arterial PO_2 is again usually normal, but there have been relatively few observations reported in these patients (Novack *et al*, 1953). A certain number of cardiac patients particularly those with mitral stenosis, are more prone to chronic bronchitis, and if associated with CO_2 retention, controlled oxygen therapy with low concentrations is indicated.

44. Recent studies have shown that the arterial PO_2 may be below normal in patients suffering from acute myocardial infarction, even though they are not in a state of shock (MacKenzie *et al*, 1964; McNicol *et al*, 1965). The cardiac output in these patients is often at the lower limit of the normal range, which again carries the implication that cellular oxygenation must be on the verge of inadequacy (Flenley, 1967). There is therefore theoretical justification for the routine administration of oxygen in high concentrations to all patients with acute myocardial infarction in the first 24 to 48 hours. There is, as yet, no statistical proof that such therapy will effect mortality or morbidity in this condition, and so far as we can ascertain, this treatment has not been subjected to a controlled trial. It appears that the financial and logistic implications of such therapy, involving as it does routine treatment of the commonest medical emergency of our time, does merit the mounting of a trial to ascertain its value. If this treatment is to be used as a routine, we strongly advocate that only low concentrations of oxygen (approximately 30%) should be used in any patient with a history of chronic cough and sputum production.

45. It has recently been realised that many patients suffering from cardiogenic shock, (as shown by a low blood pressure, cold and clammy skin, and mental clouding), as a complication of myocardial infarction may have a considerable reduction in arterial PO_2. In a small proportion of these patients, the arterial PO_2 is not brought to normal levels even by high concentrations of inspired oxygen (Mackenzie *et al*, 1964; McNicol *et al*, 1965), although there is some rise in arterial PO_2 in all patients. Nonetheless, oxygen therapy with high concentrations of oxygen does seem to be strongly indicated in myocardial infarction with shock. Although there is no statistical proof yet available that oxygen therapy does influence the mortality of this highly lethal condition there are good grounds for recommending such therapy, based both on theory (Flenley, 1967) and on actual measurements of arterial PO_2 and cardiac output. It would appear desirable to give such therapy as early as possible, to give it continuously, and to give high concentrations of oxygen.

46. Many patients with bronchial asthma, during an asthmatic attack, have a low arterial PO_2 and low arterial PCO_2 because they overventilate despite the respiratory obstruction (Rees, 1966; Tai and Read, 1967; Waddell, Emerson and Gunstone, 1967). It has recently been recognised that some patients can develop carbon dioxide retention and respiratory acidosis in a prolonged attack of status asthmaticus as ventilatory exhaustion supervenes (Marchand and Van Hassett, 1966). It seems probable that such desperately ill patients will only be saved by intermittent positive pressure respiration, but active research on this problem is urgently needed. Again, the administration of sedatives to such patients may aggravate their respiratory depression, with very serious consequences.

47. In most varieties of the alveolar capillary block syndrome, the patients are overbreathing (Donald, Renzetti, Riley and Cournand, 1952) with a low arterial PCO_2, but a serious reduction in arterial PO_2 is uncommon except on exercise, until late in the course of the disease. Oxygen in high concentrations can be safely used in those patients with a normal or low arterial PCO_2.

48. In severe kyphoscoliosis or ankylosing spondylitis, the respiratory reserve can be very limited, and a mild respiratory infection, or even immobilisation in a plaster of paris jacket, may precipitate ventilatory failure with

hypoxaemia and carbon dioxide retention. Controlled oxygen therapy, and if necessary assisted ventilation, is then required as for the bronchitic patient.

49. The arterial blood is also deficient in oxygen in conditions where blood is shunted from the right to the left heart, without partaking in gas exchange in the lungs. This arises (most commonly) in cyanotic congenital heart disease, where there is an abnormal anatomical communication, but it can also occur in cases of pulmonary arterio-venous anomaly. In these conditions, even very high concentrations of oxygen in the inspired gas do not usually produce normal values of arterial PO_2. Oxygen therapy, therefore, is unlikely to be of much value to such patients except during a superadded respiratory infection. But there is no danger of producing carbon dioxide narcosis by oxygen therapy in these patients.

50. The position of oxygen therapy in haemorrhagic and surgical shock is less certain. The classical studies of Cournand *et al* (1943) showed that "the arterial blood was normally saturated with oxygen, except in a few instances with known pulmonary disease". It would therefore seem that oxygen therapy is not required in shock, and indeed in 1957 the Medical Research Council Committee on the Treatment of Wound Shock recommended that "oxygen should not be used as a routine treatment for shock". We feel that the position should be reconsidered. All workers agree that the blood lactic acid concentration is raised in surgical and haemorrhagic shock, and the higher the level of lactic acid, the worse is the outlook for survival (Peretz *et al*, 1964). This rise in lactic acid is generally held to indicate that many cells of the body are using anaerobic metabolic pathways. Thus hypoxia at a cellular level is an invariable feature of clinical shock. This cellular oxygen deficiency results from the low cardiac output which is characteristic of shock. It follows that any reduction in arterial oxygen tension, when combined with a low blood flow may result in a fatal degree of cellular hypoxia. It has recently been suggested by McKay (1965) and Hardaway (1966) that disseminated intravascular coagulation is a prominent feature in irreversible haemorrhagic and surgical shock. These authors have described microthrombi in the lungs of such patients at post mortem, and Hardaway (1967) has attributed the low arterial PO_2 he found in 19 patients with post-operative and septic shock to these microthrombi. Confirmation of this work is awaited.

51. While recognising that the primary treatment of haemorrhagic shock remains the restoration of blood volume, we recommend that high concentrations of oxygen should also be used. The danger of failing to diagnose respiratory obstruction or crush injury of the chest may, in practice, be slightly increased by such routine use of oxygen. But, we suggest that correct instruction of first aid workers is the true defence against this problem. Pulmonary oxygen toxicity is only to be found if high concentrations of oxygen are given for a prolonged period by a mechanical ventilator. In septic or bacteremic shock, there is little evidence of arterial hypoxia and in this type of shock the cardiac output is not always low (Waisbren, 1967).

52. The need for oxygen therapy in the post-operative period following general anaesthesia, depends upon the presence of a low arterial PO_2. The difficulties of recognition of this situation from clinical examination have already been discussed (paras. 33–34). Arterial hypoxaemia has long been

known to occur after open chest operations and oxygen therapy is widely employed in the immediate post-operative care of such patients. The occurrence of hypoxaemia after other operative procedures under general anaesthesia has recently been recognised, but the extent of this hypoxaemia is still debated (Lancet, 1965; Nunn, 1965). It appears that some fall in arterial PO_2 to levels below those to be expected in patients of the same age is not at all uncommon at this time. In some instances arterial PO_2 levels below 50 mm Hg. have been recorded. The greatest hypoxaemia is found after upper abdominal surgery and may persist until the first post-operative day (Diament and Palmer, 1966). Although the mechanisms underlying this hypoxaemia are still under discussion, it appears that "the inhalation of 30 to 40% oxygen is clearly sufficient to ensure normal levels of arterial oxygen in most patients" (Nunn, 1965).

53. In view of these recent observations, we suggest that oxygen in concentrations of around 30%, by an Edinburgh mask, Ventimask or nasal catheter (Chapter 11) be given for 24–48 hours following general anaesthesia for upper abdominal surgery, if there is any history of pre-existing bronchitis, or any degree of post-operative chest complication. It is important to realise that indiscriminate use of high concentrations of oxygen, as can be obtained by the Polymask or Pneumask (Chapter 11), are potentially hazardous in these patients, unless close observation and facilities for blood gas analysis are available. Some patients tend to breathe less during this period and respiration may be further depressed by analgesic drugs, so that there is a risk of causing carbon dioxide narcosis if high concentrations of oxygen are used routinely. Further experience is necessary to determine the incidence and duration of serious hypoxaemia after general anaesthesia, in order that this treatment may be placed on a sound basis.

54. In patients suffering from carbon monoxide (coal gas) poisoning, the oxygen carrying pigment of the blood (haemoglobin) combines firmly with carbon monoxide, and is not available for oxygen transport (Chapter 9). Hyperbaric oxygen therapy can provide enough oxygen in solution in the blood plasma to overcome the difficulties of oxygen transport when haemoglobin is not available. The rate of clearance of carboxyhaemoglobin from the blood is increased by hyperbaric oxygen therapy. In this technique, high concentrations of oxygen are administered to the patient, who is placed with his attendants, in a pressure chamber. This treatment is only available at a few centres (Glasgow and Aberdeen in Scotland, with chambers operating at 2 atmospheres absolute). These chambers are very expensive and use complex equipment needing constant skilled maintenance. Over 100 cases of coal gas poisoning have been treated with the hyperbaric technique in Glasgow, and the clinical results appear to be superior to those obtained with conventional therapy; but a controlled clinical trial comparing this treatment with therapy by 100% oxygen given at atmospheric pressure has not been undertaken (Norman and Ledingham, 1967).

55. Other conditions in which hyperbaric oxygen therapy has been used include myocardial infarction where no definite conclusive benefit has so far been reported, but studies continue (Cameron *et al*, 1966). In embolism and thrombosis in other regions of the body, it appears that patients with long-standing chronic occlusive vascular disease may be helped by hyperbaric

oxygen therapy during an acute deterioration in their condition, but the long-term results are not particularly encouraging (Ledingham, 1967). In traumatic ischaemia more promising results are obtained. In gas gangrene, oxygen at 3 atmospheres pressure has been claimed to be the best available form of treatment (Brisnanelkamp, 1965) but no results from controlled trial are available. Modern therapy with antibiotics has produced very striking results in cases of gas gangrene, and careful comparison with the results of hyperbaric oxygen treatment in this condition are needed (Ledingham, 1967). Open heart surgery, in infants with cyanotic congenital heart disease, has been rendered more safe by oxygen at 3–4 atmospheres absolute in the opinion of American surgeons who have used the technique (Bernhard, Davis and Cross, 1965). In all these conditions the treatment must be regarded as experimental, and we understand that there are no plans to extend the use of hyperbaric chambers in Britain at present, until a more definite role for such treatment is established. These chambers are very expensive in capital outlay and personnel, and the use of compressed air (and oxygen) carries risks of decompression sickness, including bone necrosis (Davidson, 1964), explosions and barotrauma which can only be avoided by careful supervision. The use of small "one man" hyperbaric chambers for administering high partial pressures of oxygen as an adjunct to radiotherapy appears to have a more established place (Churchill-Davidson *et al*, 1966), but this topic is not discussed in this report. Hyperbaric oxygen therapy also carries the risks of oxygen poisoning (Paul Bert Effect) and pulmonary oxygen toxicity (Lorrain-Smith Effect) for the patient who is breathing very high partial pressures of oxygen (para. 27), but the attendants are exposed only to air at these increased pressures.

56. Recent work has emphasised the importance of the Lorrain-Smith Effect in man (Northway *et al*, 1967; Nash *et al*, 1967). Pure oxygen at atmospheric pressure produces a rapidly progressive fall in vital capacity in normal men exposed to the gas for more than sixty hours, and this is paralleled by a rise in the alveolar to arterial oxygen tension gradient (Editorial: New Eng. J. Med. 1967). These effects are rapidly corrected in the early stages by a return to breathing air. For this reason, coupled with the very considerable technical difficulty of giving high concentrations of oxygen continuously, the dangers of the Lorrain-Smith Effect in man have not been important in clinical practice. However the increasing use of mechanical ventilators, which may be driven by oxygen under pressure, has led to the recognition of the dangers of pulmonary oxygen toxicity when high concentrations of oxygen are given to these patients over a prolonged period. In reviewing recent case reports involving such oxygen toxicity, the New England Journal of Medicine (Editorial, 1967) suggests that oxygen dosage should be regulated to provide sufficient oxygen transfer to the tissues. This requires an increase in facilities for measurement of arterial oxygen tension in patients receiving prolonged mechanical ventilation with oxygen. We suggest that attention be drawn to the dangers of pulmonary oxygen toxicity in these patients, and that the need for facilities for frequent blood gas analysis in assisted ventilation units be recognised, in order to ensure that arterial PO_2 is maintained at a normal level and is not dangerously elevated.

57. High concentrations of inhaled oxygen to remove accumulations of gas in body cavities or in the tissues are not widely employed in medical practice. The theoretical basis for this treatment was described in Chapter 2, and was first confirmed in animal experiments by Fine, Frehling and Starr (1935).

It has been used in treatment of gaseous abdominal distension in surgery (Congdon and Burgess, 1939), in surgical emphysema, in air embolism (Kelly, Gilson and Meakins, 1947) in spontaneous pneumothorax (Press, 1947), and to prevent headache following air encephalography. From these reports it appears that inspired concentrations of above 95% oxygen must be maintained for several hours. Although the dangers of pulmonary oxygen toxicity (Lorrain-Smith Effect) are probably not encountered until about 10 hours of such therapy (Welch *et al*, 1963), the practical difficulties of administering these high concentrations with conventional oxygen masks (Chapter 11) may have prevented wider knowledge and application of this treatment.

58. Other conditions associated with oxygen deficiency at the tissue level are unlikely to benefit from conventional oxygen therapy. These include local embolism and thrombosis. In anaemia, from whatever cause, although the oxygen content of the blood is decreased, it has long been taught that the arterial blood is normally saturated with oxygen (Meakins and Davies, 1923). It is now known that the anaemic patient is often over-breathing, with a low arterial PCO_2, and that his arterial PO_2 is slightly below normal; but due to the shape of the oxyhaemoglobin dissociation curve his arterial saturation is well maintained (Ryan and Hickam, 1952). The efficiency of oxygen delivery to the body cells is almost normal due to the increased cardiac output (Bishop, Donald and Wade, 1955). Nevertheless if the arterial PO_2 is further decreased, as in exposure to high altitude, or following anaesthesia or pulmonary infections, it would appear that oxygen therapy, with low concentrations, may well be advisable. Poisoning of intracellular cytochrome enzymes by cyanide is discussed in Chapter 9.

59. In summary, oxygen therapy is particularly indicated in conditions where the arterial PO_2 is below normal. In most of these conditions an increase in inspired oxygen concentration will result in improvement in the arterial PO_2 sufficient to materially affect cellular oxygenation. Treatment with high concentrations of oxygen, at atmospheric pressure, carries a serious risk of producing only carbon dioxide narcosis and respiratory acidosis in patients with pre-existing carbon dioxide retention, most commonly due to chronic bronchitis and emphysema. The indications for oxygen therapy are summarised in Appendix II for the benefit of clinical staff. There is probably also a case for short courses on oxygen therapy for other relevant personnel particularly pharmacists and nurses. Simple hospital leaflets as produced for diets, anticoagulants etc. would be useful in highlighting the main principles in the therapy and its administration.

Piped Medical Gas Supplies in Hospitals

60. In 1966, two serious incidents involving piped medical gas supplies occurred in Scottish hospitals. We feel that the problems involved in the proper care and maintenance of new and existing systems of piped oxygen in hospitals do not fall within our remit. We do not include physicists or engineers amongst our members, and we are not therefore competent to advise on the engineering aspect of these matters, except from the medical point of view.

61. It is obvious that it is essential that the nature of any gas administered to a patient should be accurately known, and oxygen is no different from any other drug in this respect. It is a colourless, odourless gas and in this respect

does not differ from compressed air, or nitrous oxide, which are also piped in many hospitals. Nitrogen is also not readily distinguishable from oxygen. If oxygen were the only piped gas, no danger of confusion with nitrous oxide or nitrogen would arise. Such a proposal was universally condemned by a large number of the leading Scottish anaesthetists whose opinion we sought. The inconvenience of a return to gas supplies in cylinders was not considered to be tolerable in the long and complex operations of modern surgery. Furthermore, as recent events have shown, the removal of piped supplies of nitrous oxide would not prevent all accidents. We do not therefore recommend that oxygen should be the only piped gas in hospitals.

62. The Scottish Home and Health Department has recently advised Regional Hospital Boards and Boards of Management in Scotland on procedures to be followed on the acceptance of new piped medical gas installations, and on the safety precautions required during and after the carrying out of modifications, alterations or extensions to such systems (Scottish Hospital Memorandum No. 67/1966). Included in the guidance given in this memorandum is a recommendation that a "Responsible Officer" be designated by the hospital board, whose duty shall be to satisfy himself that the entire installation of work has been completed satisfactorily in all respects before declaring the installation to be ready for use. We feel that the duties and responsibilities of the Responsible Officer should be more clearly defined.

63. In light of this memorandum from the Scottish Home and Health Department, the Western Regional Hospital Board (Scotland) established a Working Party in November, 1966, to consider problems associated with piped medical gas and suction systems, and to advise the Regional Board on procedures to be followed. This Working Party, which included anaesthetists, medical administrators, engineers, a physicist, a biochemist and representatives of the British Oxygen Company reported in February, 1967. We are indebted to the Western Regional Hospital Board for the opportunity to study their report. This report proposes a Code of Practice, now adopted by the Western Regional Hospital Board, which appears to us to be eminently satisfactory, and we recommend that it be adopted by other hospital authorities.

64. It is common practice in many large hospitals in the United States to appoint an "inhalational therapist", with special experience and training in this field (Sakland, 1962). We suggest that the problems of inhalation therapy in hospital are such as to justify the establishment of the post of "gas technician". The duties involved in this post would be the care and maintenance of all piped gas systems in the hospital, and of all apparatus for inhalational therapy including oxygen masks, nebulisers, tents, incubators, etc. Such a post would involve close liaison with the persons responsible for care and maintenance of anaesthetic apparatus and artificial ventilators and in small hospitals the duties of these two posts could possibly be combined. This gas technician would be responsible for the operation of the oxygen analyser and Draeger Polytests proposed in the Code of Practice of the Western Regional Hospital Board. He would require to be trained in the use of these instruments and in the operation and maintenance of the other apparatus for which he is responsible. Hospital authorities should ensure that they carefully define such a technician's responsibilities and also to whom he is responsible. The line of demarcation between the duties of such a technician and these of the hospital

engineer should be clearly defined to avoid confusion in the future; unless this is done there may be some critical area of neglect. We suggest that such appointments should be made in one or two hospitals only, on a trial basis in the first place, to assess how best such personnel might be deployed.

65. In addition, we recommend that each Regional Hospital Board should appoint a technical advisor on inhalational therapy with duties including the supervision, on a regional basis, of facilities for inhalation therapy in the region. This advisor, who would probably be a Medical Physicist, would be expected to have knowledge and experience of engineering practice in this field, and to be able to advise hospital authorities on the use and selection of equipment for inhalational therapy. It would be desirable for him to work in close collaboration with anaesthetists and other clinicians who are interested in inhalation therapy.

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CHAPTER 4

Oxygen Therapy in Domiciliary Practice

66. In domiciliary practice, oxygen therapy may be indicated in a number of acute clinical situations similar to those already discussed in hospital practice (Chapter 3). The long term usage of oxygen therapy is particularly involved in treatment in the patient's home. It is the purpose of this chapter to consider the practical problems in the use of oxygen in the home, and during transport of patients to hospital.

67. Although oxygen therapy in general practice is an established form of treatment, and there was a considerable increase in the cost of such therapy in Scotland between 1960 and 1965, there does not seem to have been much enquiry into the use of oxygen therapy in general practice. However, in January and February of 1965, McNeill and Watson found that 22 patients in Dundee (population 185,000) were receiving oxygen therapy, and nearly all of these were disabled by chronic respiratory disease.

Survey of Oxygen Therapy in General Practice

68. We carried out a survey on the use of oxygen therapy in general practice in the South East Region of Scotland in the winter of 1966. A questionnaire was sent to 686 general practitioners in this area in March, 1966. Sixty per cent completed the questionnaire and these doctors were responsible for the medical care of 822,926 people. The practices were classified as urban, rural or mixed. On this basis, 71% of the population concerned were in urban practices, 9% in rural practices, and 17% in mixed practices.

69. Oxygen therapy had been used in the previous three months in 284 cases, giving an incidence of use (in that period) of 0·346 cases per 1,000 persons on general practitioners' lists in the urban practices, and 0·299 cases per 1,000 persons on the lists in rural practices. These rates were not significantly different. These figures lead to the conclusion that oxygen therapy was used for approximately one patient in every 3,000 in this three month period in both town and country practice.

70. The ages of the patients who received oxygen therapy in the three month period are shown in the histogram in Appendix III. This shows that many more males than females were given oxygen (208 males to 64 females) and that the male patients between 50 to 75 years of age greatly predominated. The details of the clinical diagnosis in the patients receiving oxygen are shown in Table 1 in Appendix III. Patients with chronic bronchitis and cor pulmonale accounted for 59% of the total cases receiving oxygen in the three winter months under consideration. Coronary thrombosis requiring oxygen therapy accounted for 5% of the total cases, pulmonary oedema for 4%, and congestive heart failure (other than cor pulmonale) for 3%. These figures support the findings of the Dundee survey, the conclusion being that chronic respiratory disease was by far the commonest clinical indication for oxygen therapy in the home, during the winter months of 1966 in the South Eastern Region of Scotland.

71. It is further to be noted that chronic respiratory disease (cor pulmonale, chronic bronchitis, respiratory failure) is also the reason for the use of oxygen

therapy over a prolonged period, particularly in the older patients. Of the 52 patients in whom oxygen was used for over one year, 39 (75%) suffered from cor pulmonale, chronic bronchitis, or respiratory failure.

72. The questionnaire also enquired into the use of oxygen during transport of emergency cases to hospital. In the six months prior to the survey, oxygen was used during transport in 216 cases, of whom 42% suffered from cor pulmonale or chronic bronchitis, 20% from coronary thrombosis, and 12% from pneumonia.

73. Information was sought on the type of apparatus used to administer oxygen. The Polymask was used for 121 patients with cor pulmonale or chronic bronchitis, and the Venti mask, or Edinburgh mask, in 33 patients. The Polymask was almost always used for patients with coronary thrombosis, congestive heart failure, or pulmonary oedema. Nasal catheters, oxygen spectacles, or oxygen tents were not used by those doctors during the period under consideration. The results are somewhat alarming, for the Polymask can provide high concentrations of oxygen (Chapter 11), which are potentially dangerous in the treatment of patients with chronic bronchitis or cor pulmonale (Chapter 3). McNeill and Watson (1966) described one case where the use of the Polymask in domiciliary practice was followed by serious respiratory acidosis, which probably contributed to the patient's death. We are left with the unproven suspicion that this danger does not occur with the expected frequency because the unsupervised patient at home rarely uses the oxygen mask continuously, or even very efficiently. If this suspicion is correct, then much long term oxygen therapy in patients with chronic respiratory disease avoids danger merely by being wasteful.

74. We were also concerned with the problems encountered in the supply of oxygen for use in general practice. Of 255 doctors in urban practices, 35% had used oxygen in treatment of emergencies during the previous six months. The 39 doctors in rural practices had used oxygen more frequently for treating emergencies in this six month period; 41% recalled using such therapy. Half of the rural doctors carried oxygen themselves, but only 21% of the urban practitioners did so. This difference is statistically significant and probably accounts for the different rate of use in emergencies. It was far more usual for the doctor practising in the town who required oxygen in an emergency to ask for it to be brought by ambulance (67%), compared with his colleague in a rural practice (13%), again a significant difference.

75. The doctors were asked to give their preferred source of supply for oxygen for emergency use. Fifty per cent of doctors in urban practice preferred the ambulance service to supply such oxygen, yet only 21% of the country doctors preferred this, the difference being highly significant. But there was no significant difference in the number of doctors in town and country who would prefer to carry their own equipment (41% of the urban practitioners and 51% of the rural doctors)[1].

76. Problems in the supply of oxygen were encountered by 257 doctors. It was not surprising to find that the difficulties of country doctors in obtaining supplies by day were encountered more often than in the case of urban doctors.

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[1] The percentages do not total 100% as several doctors made more than one choice of source of supply.

It appeared that the problems in obtaining oxygen at night in urban and rural practices were equally great. Other problems arose in the servicing of oxygen equipment, in patients not understanding the use of oxygen, and a small number of doctors noted dangers arising from use of oxygen (open fires, smoking, etc.), but no explosions of fires were recorded.

77. Finally, it must be emphasised that this survey was based entirely upon a retrospective questionnaire. The conclusions drawn are therefore only as reliable as the memories and records of busy professional men. But we feel that the survey does provide valuable insight into the usage of oxygen in general practice, and we wish to record our gratitude to all those doctors who took part in the survey.

The Requirements for Domiciliary Oxygen Therapy

78. The requirements for oxygen therapy in general practice appear to fall into three categories:

(a) The provision of oxygen in emergency situations, including the transport of patients to hospital.

(b) The use of long term oxygen therapy in chronic hypoxic states.

(c) The provision of patient carried oxygen apparatus.

Oxygen Therapy in Domiciliary Emergencies

79. Our survey shows that myocardial infarction, pulmonary oedema and exacerbations of chronic bronchitis were the commonest conditions in which oxygen was used in an emergency in the patient's home. Acute bronchial asthma and lobar pneumonia were also indications for oxygen therapy. There is obviously no difference in the clinical indications for oxygen between domiciliary and hospital practice (Chapter 3), although oxygen is more difficult to provide in the patient's home. In some of these cases, notably in pulmonary oedema, acute myocardial infarction, severe exacerbation of chronic bronchitis, or bronchial asthma, hospital admission may be required. We must emphasise that low concentrations of oxygen, of the order of 30%, as given by an Edinburgh mask at 2 litres/minute ("Med" setting on the fixed flow regulator, Chapter 11), are indicated in treatment of acute exacerbations of chronic bronchitis, and in most cases of bronchial asthma in the home. Pulmonary oedema, myocardial infarction with shock, and lobar pneumonia require high concentrations, given as 6 litres of oxygen/minute ("High" setting of the regulator, Chapter 11), with a Polymask or M.C. mask.

80. During the transport of these patients to hospital, oxygen therapy should be continued. The same principles as outlined in the previous paragraph apply, and it follows that ambulances should carry an oxygen cylinder, fitted with a fixed flow regulator, and both an Edinburgh mask (or Ventimask) and Polymask (or M.C. mask). The ambulance man should give oxygen as prescribed by the doctor, but if he receives no specific instructions we recommend that all cases needing oxygen be given low concentrations only, i.e. 2 litres of oxygen/minute by Edinburgh mask or 4 litres/minute by Ventimask (Chapter 11). The details of oxygen therapy equipment in ambulances are discussed in Chapter 11, and a modified form of instructions to ambulance staff, to give effect to this recommendation, is given in Appendix IV.

81. The general practitioner will sometimes be involved in resuscitation, most frequently at second hand, fulfilling the role of the "professional rescuer" as discussed in Chapter 8. Resuscitation may be needed in cardiac arrest, domestic accidents, electrocution, road accidents, domestic gas poisoning, near-drowning, etc. Resuscitation is fully discussed in Chapter 8, but we feel that some points are worthy of repetition. Artificial ventilation with expired air, after ensuring that the airway is clear is now fully established as by far the most efficient first aid method. We are of the opinion that a professional rescuer, (which includes the general practitioner), should carry some simple aid to resuscitation, such as a bag and mask resuscitator and oropharyngeal airway. This apparatus is further discussed in Chapters 8 and 11. The ambulance man should also be similarly equipped, and we recommend that such equipment should include a lightweight, self-contained supply of oxygen. This modification is not at present available, but we urge that manufacturers should be acquainted with the need.

Long Term Oxygen Therapy

82. The major use of oxygen in domiciliary practice lies in the long term management of chronic respiratory diseases. However, it is very difficult to be certain that such therapy really does benefit the patient. There is little doubt that the development of right-sided heart failure (cor pulmonale) reduces the life expectancy in patients with chronic lung disease. Patients suffering from cor pulmonale have a lower arterial PO_2 when breathing air than do patients with chronic chest disease who have never been in heart failure (Platts, Hammond and Stewart-Harris, 1960). Is it therefore possible that correction of such hypoxia by long term oxygen therapy, will decrease the mortality from cor pulmonale? Recently Cole (1968) has reported a fall in pulmonary artery pressure and pulmonary vascular resistance in six severe chronic bronchitics who were given oxygen continuously by nasal catheter for four to six weeks. This is interesting and encouraging, and in view of the important implications further research to answer this problem does seem to be urgently required, despite the great difficulties of mounting such an investigation. In Scotland, in 1957, chronic bronchitis was certified as the cause of incapacity lasting more than one year in 10 cases per 100,000 of population. Our survey showed an incidence of use of oxygen therapy lasting more than one year, for chronic respiratory disease, of 4·7 per 100,000 of population in south-east Scotland in three months of 1966. If we postulate that any patient with chronic respiratory disease who is incapacitated for more than one year needs long term oxygen therapy, then use of such therapy, on the long term basis, would increase by a factor of 2 or 3 times. It is apparent that there are many assumptions in this rough calculation, but it does give some indication of the size of the problem. It would appear to be advisable to try to determine if such an increase in use of oxygen would be worthwhile.

83. It is important to point out again that most of these patients with chronic respiratory disease require only about 30% oxygen, and that higher concentrations may be dangerous. As these patients constitute the greatest number of those who need domiciliary oxygen, it is desirable that the standard oxygen giving set, supplied for domiciliary therapy, should include a "low concentration" device (Edinburgh mask or Ventimask) and not, as at present, the Polymask. The Polymask or M.C. mask would remain as the alternative

device which would need to be prescribed precisely, when the doctor felt that high concentrations of oxygen were needed (as in pulmonary oedema, etc.).

Patient-carried oxygen sets

84. The administration of oxygen to the ambulant patient has been carefully investigated by Dr Cotes, of the Pneumoconiosis Research Unit, Medical Research Council, to whom we are indebted for advice. It would appear that oxygen therapy during exertion is only of value if there is objective evidence that the patient's breathlessness is thereby relieved. To ensure that this is so, it is necessary to compare the patient's response to exercise when he is breathing either air or oxygen, administered in a manner so that he is unaware which is being given. The intensity of the exercise is first adjusted by trial and error so that the patient is obliged to stop on account of breathlessness after two minutes breathing air. The effects of air and oxygen are then compared. Experience would suggest that a patient is only likely to benefit from using a patient-carried oxygen set if his exercise period is doubled as a result of a reduction in pulmonary ventilation when breathing oxygen (Cotes and Gilson, 1956; Cotes, 1960).

85. Facilities for carrying out such tests will obviously be limited. In addition there is support for the view that a patient-carried oxygen set will be of benefit if the patient's breathlessness is relieved by oxygen irrespective of whether the pulmonary ventilation is reduced. Many patients with chronic respiratory disease find great subjective benefit from breathing oxygen after a period of exercise. It is possible that the relief is a placebo effect, but more research on this subject appears to be needed.

86. On the strict criteria of the "doubling of exercise time", a patient-carried oxygen set is found suitable for only a few patients. Many patients are either too breathless to exercise sufficiently to benefit from oxygen or do not fulfill these criteria, (Cotes, 1960). Patients whose exercise ability is limited by cough, fatigue or dizziness are not likely to benefit from oxygen therapy on exercise. The use made of a patient-carried oxygen set depends very much on the patient's motivation. Many patients will not wear an oxygen mask in the street, but a "palm breathing device" (Chapter 11) could help to overcome this problem. In addition, portable oxygen equipment is sometimes not used for its intended purpose; it can be regarded as a "status symbol", or used to provide oxygen after exercise, in the same manner as a fixed oxygen cylinder.

87. Patient-carried oxygen sets do need fairly regular maintenance. In addition, the patient or his relatives must understand how to refill the small lightweight oxygen cylinder from a large fixed cylinder; otherwise the running costs of the treatment are very high.

88. In summary, we conclude that the problems of portable oxygen therapy are far from simple. It is difficult to select those patients who are likely to benefit, and difficult to be sure that the equipment is properly used and maintained. In addition, it is not certain that life or comfort of many of these patients is necessarily improved by use of such apparatus. Furthermore, the apparatus is expensive and we do not recommend that such equipment should at present be included in the Drug Tariff. This means that such equipment can only be provided under the National Health Service on the advice of a Consultant.

At the same time, it is desirable to investigate the value of such patient-carried oxygen sets in larger numbers of patients.

89. Many patients who would not benefit from a patient-carried set can be given oxygen on exertion in the home by means of a 20 ft. length of 3 mm. bore polythene tubing, with adapters to fit a standard oxygen cylinder and an oxygen mask. It is desirable to make this tubing and adapters available on the Drug Tariff, so that they may be prescibed with an oxygen giving set on Form E.C. 10.

Practical Difficulties

90. The questionnaire to General Practitioners has pinpointed the fact that practical difficulties exist in the provision of oxygen in domiciliary practice. These are:

(i) The supply of oxygen and oxygen therapy equipment.
(ii) The servicing of oxygen cylinders and other equipment.
(iii) The lack of understanding by patients of the operation of oxygen therapy equipment.
(iv) Dangers in the use of the equipment.

In considering these difficulties, account should be taken of the fact that in domiciliary practice, oxygen therapy is required in the different situations of emergency and long term use.

91. The existing arrangements for the provision of oxygen therapy in domiciliary practice are described in Memorandum E.C.S.(P) 9B/1965 to General Practitioners. Under these arrangements, each Executive Council is required to supply general practitioners in its area with a list of chemists who have satisfied the Executive Council that they regularly stock oxygen equipment, are prepared to deliver the oxygen giving set and cylinders to the patient's home and are prepared to set up and explain the operation of the oxygen set to the patient. The list includes details of chemists who are prepared, in an emergency, to provide oxygen therapy after normal business hours. The intention is that these arrangements will make it unnecessary, except in the most urgent circumstances, for doctors to apply to the ambulance, hospital and other services for assistance in supplying oxygen for use in the patient's home.

92. In spite of these arrangements which were intended to improve the situation which existed before 1965, it is apparent from our enquiries in 1966 that general practitioners are still not fully satisfied. Oxygen therapy in domiciliary practice can be provided from four sources: (a) the chemist, (b) the general practitioner carrying his own, (c) the ambulance service and (d) the hospital service. Of these sources, preferences for emergency supplies in reply to our questionnaire were:

(a)	Contracting chemist	— 19% of urban doctors 27% of rural doctors
(b)	Doctors carrying their own	— 41% of urban doctors 52% of rural doctors
(c)	Ambulance service	— 50% of urban doctors 22% of rural doctors
(d)	Hospital service	— 10% of urban doctors 22% of rural doctors

(These figures do not total 100% on account of the fact that some doctors stated more than one preference).

93. We suggest that the arrangements for the chemist to supply oxygen therapy equipment are reasonably satisfactory in long-term cases. However, it seems unreasonable, particularly in rural districts, to expect the chemist to provide a 24-hour emergency service.

94. Where oxygen is required in an emergency, it should be readily available. In emergency in domiciliary practice, oxygen therapy may be necessary for only a short time, often until the patient can be transferred to hospital. Difficulties encountered by doctors carrying their own equipment arise mainly from the size and weight of oxygen cylinders, from servicing difficulties, and from the cost of purchasing equipment and replenishing the stocks of oxygen.

95. The principal function of the ambulance service is the transport of patients, and the transport of equipment only adds to the enormous burden already put upon the service. We regard the transport of oxygen therapy equipment to the patient at home as a misuse of the ambulance service. In rural districts, there might be unavoidable delays in the delivery of equipment for emergency use and ambulances might be better used for the transport of patients to hospital, provided oxygen equipment were available for use during the journey (Chapter 11).

96. A minority of doctors preferred oxygen equipment to be supplied by the hospital service. Hospitals are not geared to provide such a service, and to do so would involve considerable expenditure in the provision of personnel, equipment and transport.

97. In cases of emergency, for the sake of convenience and speed, we recommend that all general practitioners should carry their own supplies of oxygen and oxygen therapy equipment.

98. This equipment should be readily maintained and easily handled and portable. For this purpose, lightweight cylinders of the smallest possible dimensions, capable of providing a flow of 6 litres of oxygen/minute for one hour should be available. We have found that none of the "portable" oxygen sets described in Chapter 11 meet this requirement. In view of the distances which general practitioners may have to walk, the weight of the cylinders is of great importance. The weight and size of the reducing valves, flowmeters, masks and other equipment which go to make up the portable oxygen set for use by general practitioners also requires careful consideration. It is likely that a suitable portable oxygen set will be expensive, but it is important that no general practitioner should be reluctant to carry one because of the cost involved. For this reason, it is suggested that the equipment should be provided to each general practitioner free of charge.

99. With regard to the replenishing of cylinders, there appear to be two alternatives. Either the lightweight portable cylinders could be recharged by the general practitioner himself from a large cylinder kept at his surgery premises, or the cylinders could be provided by the chemist on an exchange basis. The chemists themselves could either recharge the portable cylinders, or carry a stock of them. By relieving chemists of the need to provide a 24-hour service for the supply of oxygen therapy equipment, it should be possible for chemists to service oxygen therapy equipment supplied by them to the patient's home, and to check and replenish the supply at regular intervals. It should also be possible for these chemists to assist general practitioners to show patients

how to use the equipment and how to overcome the dangers in use. The role of Health Centres in the supply of oxygen therapy equipment for domiciliary purposes will no doubt have to be explored at a later date.

100. We strongly recommend that every encouragement should be given to general practitioners to carry their own oxygen therapy equipment for use in emergency situations. In view of the technical problems invloved in the provision of lightweight oxygen equipment and the associated problems of the supply and maintenance of this equipment, we recommend that a Working Group be established to advise in this matter, and on the problem of the provision of oxygen by means of bag and mask resuscitators (para. 143, Chapter 8; para. 154, Chapter 9; para. 212, Chapter 11). Such a working group could also look into the technical problems of sterilisation of oxygen equipment and the use of disposable masks and catheters.

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CHAPTER 5

Oxygen Therapy in Obstetrical Practice

101. "Direct measurements on human foetuses in utero are as yet insufficient to give a clear picture of the normal oxygen tensions. It is only during the last few years that information has become available from animal experiments with long-term implantation of catheters. These have shown that the partial pressure of arterial oxygen (PO_2) of the foetus is very stable during the last half of gestation. Although it is low by adult standards it does not fall, nor does the O_2 carrying capacity of the blood rise, until shortly before or during delivery. Contrary to previous opinion there is no direct evidence to support the view that in a normal pregnancy the foetus outgrows the capacity of the placenta to supply it with its basic needs. It does not appear to suffer from O_2 lack, probably because of an adequate O_2 saturation and high umbilical and foetal systemic blood flow. The maternal arterial PCO_2 is reduced during pregnancy and the foetal arterial PCO_2 is normally at approximately the same level." (W.H.O. Report, 1965)

102. In situations associated with a low arterial PO_2 in the mother there is little doubt that the transfer of oxygen to the foetus will be impaired. Foetal hypoxia arising as a result of such maternal hypoxia will be corrected by administering oxygen to the mother, provided that the maternal-placental-foetal circulation is intact and functioning efficiently. It is therefore desirable to administer oxygen to any pregnant woman in or out of labour, who suffers from any condition causing a lowered arterial PO_2 (Chapter 3). In obstetrical practice, mitral stenosis with pulmonary oedema and pneumonia are among the most frequent medical indications for oxygen therapy.

103. Elevation of the arterial PO_2 above normal values has been found to have very little effect on the foetal arterial PO_2. This appears to result from the high oxygen consumption of the placenta itself (Campbell *et al*, 1966). It follows that the administration of oxygen to a mother, who has a normal arterial PO_2 when breathing air, can make little difference to foetal oxygenation. There is indeed some evidence that high oxygen tensions constrict the foetal vessels in the placenta (Nyberg and Westin, 1957). The work of Saling in Germany on acid base values in foetal scalp blood suggests that this effect of oxygen may increase foetal acidosis.

104. It has recently been reported that the arterial oxygen saturation of 61 women in late pregnancy varied from 58·5% to 99·7% with a mean value of 85·2%, (Stenger *et al*, 1966). Although these American patients were studied just prior to Caesarean section, they were described as "normal". Animal studies suggest that such a reduction in maternal arterial PO_2 would be accompanied by a fall in foetal arterial PO_2 (Born, Dawes and Mott, 1956). One might conclude from these studies that oxygen should be administered routinely to all pregnant women at term, for some may have undetected arterial hypoxia. However, we do not take this view. When these results of Stenger *et el*, (1966) are compared to those of Vasicka *et al*, (1960), Rooth and Sjostedt (1960) and Wulf (1964), (Appendix V) the concensus of opinion is seen to be that the maternal arterial PO_2 is normal in late pregnancy in normal women. We

do not therefore recommend that oxygen should be administered as a routine to all women in late pregnancy and labour.

105. In the last few years evidence has grown that the "Minnit" gas/air apparatus, as used for analgesia in obstetrical practice, can deliver oxygen concentrations as low as 10% to the mother in labour (Cole and Nainby-Luxmoore, 1962). This apparatus should not, in our opinion, be used, for in this situation the dangers of severe foetal hypoxia are very grave. Any anaesthetic regime that allows the administration of oxygen concentrations to fall below 21% is also to be condemned. A Committee of the Medical Research Council under the chairmanship of Sir Dugald Baird has recommended that a 50/50 mixture of nitrous oxide and oxygen in a single cylinder will provide safe analgesia in labour. This mixture is commercially available as "Entonox" (British Oxygen Company). At temperatures below–8° C there is a possibility that the mixture could separate out into its component gases, but this is unlikely to be a hazard in practice (Gale, Tunstall and Wilton-Davies, 1964).

106. Oxygen therapy may be required in some of the rare catastrophies of labour. In amniotic fluid embolism, the clinical picture of shock is accompanied by severe hypoxia, which presumably results from the multiple thrombi in the pulmonary arterioles (McKay, 1965). The pulmonary aspiration syndrome (Mendelson, 1949) can produce severe hypoxia which only responds to clearing of the airway and intermittent positive pressure ventilation with high concentrations of oxygen (McCormick, 1966). In shock resulting from other obstetrical causes, such as haemorrhage or rupture of the uterus, oxygen therapy should, in our opinion, be included in the management (Chapter 3), although it will be of secondary importance to the restoration of blood volume and appropriate surgical measures.

107. Foetal tissues are obviously exposed to lower tensions of oxygen than those of the adult. In normal circumstances, the foetal PO_2 is stable in the later part of pregnancy, but this is critically dependent upon the placental circulation. If maternal blood flow to the placenta is reduced, as in the maternal hypertensive syndromes, or as a result of a strong uterine contraction in a prolonged labour, or maternal hypotension due to blood loss, drugs or anaesthesia, the administration of oxygen to the mother may be of some help to the foetus, and is unlikely to harm the mother. There is need for further research on this situation.

108. The most uncertain problem is the value of oxygen administration to the mother when there is foetal distress. It is difficult to define foetal distress and there is a multiplicity of causes. Futhermore there is a marked reduction in the exchange of oxygen and carbon dioxide between the mother and baby during the normal process of delivery (James, 1960) and there is a rapid recovery from the low PO_2 levels and acidosis *after* birth. In *abnormal* situations with foetal distress, the most important therapeutic procedure is immediate delivery and the sooner the baby is born the better. The interference with the maternal-placental-foetal circulation which is usual in such circumstances makes the value of oxygen administration doubtful, and it is probable that there is little benefit for the foetus from any increase in oxygen saturation of blood in the intervillous space. The obstetric opinion which we have sought has varied from the view that oxygen therapy in foetal distress in the absence of maternal hypoxia is of doubtful value, and may even be harmful, to the view that it

may occasionally help and cannot do harm. In the present state of our knowledge, we do not think that an authoritative opinion can be expressed. Indeed, we have been impressed by the comparative lack of physiological data concerning gas exchange between mother and foetus, and until such knowledge is forthcoming, the use of oxygen during pregnancy and labour cannot be placed upon a sound physiological basis.

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CHAPTER 6

Oxygen Administration in Paediatric Practice

109. Oxygen therapy is a common requirement in paediatric practice. During the neonatal period severe respiratory distress commonly creates an emergency situation and can arise from a variety of causes. The accessibility of the umbilical arteries makes periodic measurements of the PO_2 a practicable proposition in the newborn infant, and the PCO_2 pH and acid base state can also be determined on arterial or arterialized capillary blood. The importance of measurements of the oxygen concentration when any additional oxygen is given to the newborn infant is now well appreciated. Although carbon dioxide retention is common in these babies, the administration of oxygen, even in high concentrations, does not result in the respiratory depression so often encountered in the adult suffering from respiratory failure. Furthermore, large right-to-left shunts may occur in neonates with severe respiratory distress (a return to the foetal circulation) and this may make it impossible to raise the arterial PO_2 to satisfactory levels even when high concentrations of oxygen are given. In older infants and young children on the other hand, the technical difficulties of obtaining arterial blood have so far prevented the detailed measurements which have helped to rationalise the use of oxygen in the newborn and in the adult. There is evidence that in acute infections of the lower respiratory tract during infancy and childhood, there is no danger of producing carbon dioxide narcosis during oxygen therapy (Simpson and Flenley, 1967). Nevertheless, further research is required to clarify the use and effects of oxygen therapy in this age group.

Indications for Oxygen Therapy

110. (a) *Neonatal period*

(i) **Asphyxia neonatorum** (failure to establish respiration after birth)

(ii) **Respiratory distress** due to:
Idiopathic respiratory distress syndrome (Hyaline membrane disease).
Intrauterine (congenital) pneumonia.
Meconium aspiration syndrome.
Spontaneous pneumothorax.
Congestive cardiac failure, etc.

(b) *Infancy and Childhood*

(i) Acute viral bronchiolitis.

(ii) Bacterial (and other) bronchopneumonia.

(iii) Acute laryngo-tracheo-bronchitis (including epiglottitis) — tracheostomy often necessary.

(iv) Acute asthma.

(v) Cystic fibrosis of the pancreas (mucoviscidosis).

(vi) Congestive cardiac failure.

(vii) Severe dehydration.

(viii) Oligaemic shock, e.g. burns, severe haemorrhage.

Methods of Administration

(a) *Neonatal period*

111. In **asphyxia neonatorum** oxygen may be given with advantage to the gasping infant from plastic filter funnel or small face mask. It is regrettable that district midwives and general practitioners conducting home confinements frequently lack this simple apparatus which need amount only to a lightweight oxygen cylinder, tubing and funnel or mask.

112. In severe cases with Apgar ratings of 3 or less (Appendix VI) tracheal intubation and intermittent positive pressure inflation of the lungs with oxygen (or air) is often essential. This is only practicable in hospitals where trained staff and suitable apparatus (e.g., the Oxygenaire Resuscitator) are immediately available. In an emergency outside hospital, mouth-to-mouth breathing, properly carried out, may be life-saving.

113. Hyperbaric oxygen has also been used in the resuscitation of the severely asphyxiated newborn (Hutchison *et al*, 1966). It has been shown conclusively that the use of hyperbaric oxygen as specified by these workers gives results equalling those obtained by intubation and intermittent positive pressure inflation by highly qualified practitioners (consultants and selected registrars). (Hutchison, Kerr, Inall and Shanks, 1966). We felt that whereas the hyperbaric procedure used by Hutchison and his colleagues could be carried out with only a minimum of special training, there was considerable doubt that the high standards of intubation and ventilation in the controlled study pertained generally. During a three year trial by Hutchison and his colleagues, 450 infants received hyperbaric treatment and 403 survived. None of the survivors have shown any evidence of retrolental fibroplasia or of lung damage caused by oxygen. There have been no episodes of convulsions while the infants were at pressure. The risks of explosion are negligible if the procedure is carried out in the manner described.

114. After reviewing the relevant literature and published debates concerning this therapy we support the continuance of the hyperbaric technique in the Glasgow Medical School. We would also encourage other appropriate centres to appraise this technique against that of intubation and assisted ventilation.[1]

115. In **respiratory distress** oxygen is best given in an incubator where high humidity (75-95 per cent) and accurate temperature control 85-95° F are also obtainable. The most recent incubators (e.g., the new Oxygenaire and Isolette) incorporate an oxygen flow control to limit the oxygen concentration, under normal working conditions, to 35%. This requirement is specified by the British Standards Institution (British Standard 3061: 1965) but many incubators still do not comply with this specification and it is strongly recommended that the oxygen concentrations in incubators should be monitored

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1 Professor J. H. Hutchison, who is a member of the Sub-Committee, asked not to be involved in our deliberations concerning hyperbaric oxygen therapy in asphyxia neonatorum. These comments are therefore made by the Chairman and members of the Sub-Committee excluding Professor Hutchison.

We would like to express our gratitude to the Chairman of the M.R.C. Committee on Hyperbaric Oxygen Therapy who convened an invaluable special meeting at our request with a number of invited expert participants. The above recommendations are made by the present Sub-Committee and not by the M.R.C. Committee on Hyperbaric Oxygen Therapy which reviewed and discussed present knowledge and opinion without making recommendations.

with a suitable paramagnetic oxygen meter. Oxygen concentrations above 35% are generally forbidden to avoid the risk of retrolental fibroplasia in premature babies. There is, of course, no such risk when the arterial PO_2 is below normal, and in cases of severe respiratory distress it is frequently necessary to raise the oxygen concentration in the incubator to much higher levels. The British Standard specification requires that the oxygen flow control shall be easily adaptable to provide a higher concentration, but the fact that higher concentrations are being given must be clearly indicated on the incubator. These requirements are met in the new Oxygenaire and Isolette incubators where an oxygen concentration exceeding 60% is readily obtainable. Even higher concentrations, approaching 100%, can be obtained by placing a suitable plastic hood over the infant's head inside the incubator and running an oxygen lead into it. *High oxygen concentrations must only be used under expert paediatric supervision and the levels should be carefully controlled by periodic measurements with an oxygen meter.* Some paediatricians also monitor the infant's arterial PO_2, pH and PCO_2 levels at frequent intervals to ensure more accurate and safe administration of oxygen. This practice is increasing and we would greatly encourage this in clinical situations where oxygen concentrations above 35% are being administered owing to severe lung damage or respiratory failure.

116. A portable battery/mains operated incubator is available (Oxygenaire Mark III) for transporting infants from home to hospital. This avoids the dangers of hypoxia and hypothermia during the ambulance journey and it is recommended that any ill newborn infant should be transported in this way.

117. Oxygen may be administered to the newborn for short periods in a plastic cot covered with a hinged plastic lid. Vickers Ltd. (Oxygenaire) supply an oxygen inlet to these lids on which is stated the oxygen concentration to be predicted at different flow-rates.

(b) *Infancy and Childhood*

118. Masks are only practicable in older children. The paediatrician, therefore, uses a variety of oxygen tents but this raises the problem of obtaining arterial blood for gas analysis which is necessary for the accurate control of oxygen therapy. In addition to oxygen a common requirement e.g., in bronchiolitis and laryngo-tracheo-bronchitis, is high humidity, and sometimes coolness for highly fevered children. Some apparatus can regulate all three factors, oxygen, humidity and coolness e.g., the Humidaire (Oxygenaire) and the Croupettes — large and small (Air-Shields (UK) Ltd.). For older children, the Humidaire and Croupette may be too confined, and suitable tents are available e.g. Oxygenaire Universal Model, and the Oxygenaire Mark V Refrigeration Model which provides high humidity and some degree of temperature control. In both tents a CO_2 wash out device is fitted which produce a fall in CO_2 concentration to 0·5% or less, but as this is based upon the principle of an entrainment its use results in a marked fall in the concentrations of oxygen. However, CO_2 narcosis does not seem to be a danger in infancy whereas an adequate oxygen concentration within the tent is extremely important. It should be noted that while Reynolds (1963) suggested that an inspired oxygen concentration of 40% could raise the arterial PO_2 above 100 mm Hg. in babies with bronchiolitis, Simpson and Flenley (1967) found that the administration of 40% oxygen failed to raise the PO_2 over 80 mm Hg. in

4 out of 32 cases (12%). In routine practice an oxygen concentration of 40% is frequently not achieved in oxygen tents. This is partly because the zip fastening on the tent must be opened for feeding, toilet and nursing procedures and because the canopy of the tent is sometimes not properly tucked in around the cot-side. In the case of some tents, the oxygen concentrations achieved under standard conditions fail to match the concentrations claimed by the manufacturers (Simpson and Russell, 1967). It is now established practice to measure the oxygen concentration in incubators for the newborn, and similar measurements should be made more frequently in the case of oxygen tents for older children. This would ensure both greater accuracy in the administration of oxygen by the paediatrician and greater awareness of the need for meticulous handling of the tent by nursing staff. The object should be to raise and maintain the oxygen concentration in the tent to at least 40%.

Risks of Oxygen Administration in Infancy and Childhood

119. The danger of retrolental fibroplasia in the premature infant is now well recognised. The concentration of oxygen in incubators should be permitted to rise above 35% only in cases of severe respiratory distress when cyanosis is not abolished, and preferably under biochemical control. Oxygen should not be given for any period longer than is necessary.

120. In older infants nursed in oxygen tents where it is difficult to maintain oxygen concentrations much above 40% for long periods the risks are minimal. The extreme importance of avoiding open fires or naked lights where oxygen tents or incubators are in use cannot be over-emphasised but the dangers should be self-evident.

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CHAPTER 7

Oxygen and Travel by Air

121. As altitude increases the pressures of the constituent gases of the atmosphere decrease uniformly. At first the PO_2 in the alveoli also decreases uniformly but the PCO_2 is little altered. At an altitude of 10,000 ft., however, the atmospheric pressure is approximately 525 mm Hg. and the arterial PO_2 falls to 60 mm Hg. at which level the hypoxic stimulus to breathing begins to operate, so that thereafter the arterial PO_2 does not fall so rapidly with decreasing atmospheric pressure. This ventilatory response to hypoxia also washes more carbon dioxide out of the alveoli so that the PCO_2 in the alveoli and arterial blood becomes less as altitude increases. The effect of hypoxia of moderate degree is insidious and the subject may not be aware of a slight impairment of judgement and night vision or, if he is inactive, of an increased liability to fatigue on effort.

122. Modern aircraft fly at high speed and a great altitude. It would be ideal to maintain a cabin pressure of one atmosphere, equivalent to the pressure at sea level. To do so would make unrealistic demands on the power from the aircraft's engines and on robustness of the structure of the aircraft. At the present time a cabin pressure equivalent to atmospheric pressure at an altitude of 8,000 ft. is the normal limit of acceptance, but in practice unless the aircraft is flying at its ceiling, it is more common for the cabin altitude of passenger aircraft to be about 5,000 ft. to 6,000 ft. At an altitude of 5,000 ft. the arterial PO_2 is about 80 mm Hg. with arterial oxygen saturation of 95%. The effect upon the subject is equivalent to breathing 17% oxygen at sea level. In normal subjects this is negligible, but in some patients a slight reduction in arterial oxygen tension may cause symptoms and it is important that practitioners should be able to anticipate these. Advice will often be sought by patients about the advisability of air travel and about any precautions that may be necessary and, although the practitioner can refer these questions to an experienced member of the medical staff of the air line, it is desirable that he should have a working knowledge of the problems involved.

123. The patients who are particularly vulnerable to a lowered oxygen tension in inspired air are those who have difficulty in oxygenating their blood, even at sea level, because of pulmonary or cardiac disease and those with impaired circulation of a vital organ of the body such as the brain or heart.

124. Patients with central cyanosis at sea level have an arterial PO_2 that is on or close to the steep part of the oxyhaemoglobin dissociation curve (Chapter 2). A further slight reduction in PO_2, due to altitude, would result in a large reduction in oxygen saturation and tend to cause tissue hypoxia. As has already been mentioned in Chapter 3, patients with chronic pulmonary disease may have arterial desaturation associated with either a normal or raised carbon-dioxide tension and this influences the correct choice of oxygen therapy. For any patient who is reasonably comfortable breathing air at sea level, all that should be necessary to relieve dyspnoea and restore his "normal" arterial oxygen tension during flight is for him to breathe 25% oxygen in air when the cabin altitude is 5,000 ft. or 30% oxygen in air when it is 10,000 ft. These concentrations can be achieved by supplying oxygen through an Edinburgh

mask at a flow rate of 1 and 2 litres/minute respectively. For the severe chronic bronchitic who has hypercapnia as well as arterial desaturation, it is potentially dangerous to give oxygen much in excess of these concentrations because of the risk of hypoventilation and carbon dioxide narcosis. Since it is considered unwise to give more than 30% oxygen to these patients at sea level without careful supervision, it would be equally unwise to give them more than 35% oxygen at 5,000 ft. or 40% at 10,000 ft. Any patient suffering from an acute exacerbation of chronic bronchitis is unsuitable for air travel because oxygen therapy at that stage of the illness is sufficiently difficult without the added complication of altitude.

125. Central cyanosis due to heart disease results either from the secondary effects of heart disease on the ventilation and perfusion of the lungs or from direct anatomical shunting of blood from the right to the left side of the heart. In either case carbon dioxide retention is very unlikely and consequently higher concentrations of oxygen can be given without fear of carbon dioxide narcosis. It is obvious that patients in heart failure, from whatever cause, should not travel by air until maximal improvement in their condition has been obtained by treatment. Even then, the additional stress and excitement of travel together with the reduced arterial oxygen tension due to altitude, may be enough to precipitate failure. It is advisable, therefore, that a special request be made for a provision of oxygen during the flight.

126. In patients with an anatomical shunt, additional oxygen will not correct the cyanosis, but it should be sufficient to prevent an important fall in the saturation of arterial blood at altitude, if the oxygen tension is on the steep part of the oxyhaemoglobin dissociation curve. This applies particularly to the transport of patients, often young children, with cyanotic congenital heart disease from one country to another where investigation and surgical repair of the cardiac lesion is contemplated. Special arrangements will have to be made for very young children and since oxygen tents and oxycots are considered to be dangerous in long-distance, scheduled aircraft because of the fire hazard, small sized oxygen masks must be provided. For older patients, oxygen can be given through a correctly fitted Polymask at a flow rate of 6 litres/minute which provides an inspired concentration of about 60% oxygen (Chapter 11). If this uses up oxygen too rapidly and necessitates an excessive weight of cylinders, the use of a mask with a reservoir bag and a valve to prevent re-breathing would help to conserve oxygen that is otherwise wasted during the expiratory phase of respiration. An alternative would be to supply oxygen through a demand valve on inspiration only, and to ajust the regulator to allow dilution of the oxygen with cabin air according to the altitude and the patient's requirements.

127. If the blood supply to any organ is impaired, the amount of oxygen available for metabolism is not necessarily less when the arterial oxygen tension falls. This is because, under these circumstances, the extraction of oxygen from blood flowing through the organ may become more complete. However, the penetration of oxygen into tissue that is very poorly perfused depends on the oxygen tension gradient and this is inevitably reduced as the arterial PO_2 falls. Furthermore, in the case of heart muscle, oxygen extraction from the coronary blood is already fairly complete, so that the coronary venous blood is normally only about 20% to 30% saturated with oxygen. This does

not allow for a much greater extraction of oxygen by the myocardium if the work of the heart increases or if the arterial oxygen tension falls and, therefore, coronary blood flow must increase to meet the oxygen requirements. In patients with diffuse coronary artery disease this can only be done to limited extent and thus it may be necessary to increase the inspired oxygen concentration. It must be admitted that many patients suffering from angina pectoris travel by air without any symptoms at all but whenever a patient's exercise tolerance is severely limited at sea level it is a wise precaution to administer oxygen at the first intimation of anginal pain during flight. Patients who have recently suffered from coronary thrombosis are generally advised not to travel within 8 to 12 weeks from the start of their illness but each case should be assessed separately. Thus it might be justifiable for a patient who has suffered a mild attack to travel earlier but one whose illness has been complicated by cardiac failure might have to wait longer. The exertion and excitement of air travel are additional factors which must be taken into account and if required the airlines are prepared to help in reducing these to a minimum by special boarding facilities, etc.

128. Patients with impaired cerebral circulation might also be expected to present a special case for additional oxygen during flight but their need appears to be less than one might expect. Mild confusion in the elderly may be helped by oxygen but, otherwise, the degree of hypoxia encountered is unlikely to influence an established neurological lesion.

129. Apart from these two broad groups in which oxygenation of blood is impaired and the circulation of an organ is defective, there are two further conditions in which oxygen therapy must be considered. In anaemia, from any cause, although the arterial PO_2 is nearly normal, when breathing air at sea level, the content of oxygen in the arterial blood is below normal. At altitude the decreased PO_2 results in a further fall in arterial oxygen content. Although correction of the anaemia itself is the most satisfactory treatment, the provision of some oxygen at altitude can prevent this further fall in PO_2, and render the patient's oxygen transport at least as efficient as that which he enjoyed at sea level. We therefore recommend that 30% oxygen should be given to patient's with a haemoglobin concentration below 7 Grams/100 ml. of blood, if they must travel by air, but it is preferable that they delay air travel until treatment has raised the haemoglobin to higher levels (not less than 8 Grams/100 ml. or more). The other special condition is sickle cell anaemia in which the red blood cells are particularly vulnerable to a low oxygen tension which causes them to distort, clump and haemolyse. This condition is particularly common among persons of African descent. Occasional cases of splenic infarction have occurred in such cases at cabin altitudes of 4,000 to 6,000 ft. and, therefore, whenever anyone known to have sickle cell anaemia travels by air, oxygen should be given throughout the journey and the cabin altitude kept as low as possible. Others may be unaware that they carry the sickle cell trait but sudden pain in the left upper abdomen, particularly in a negro, suggests the possibility of splenic infarction and oxygen should be administered at once.

130. Many other problems arise in the transport of invalids by air and, although they are very important, they are not specifically concerned with oxygen therapy. Thus, volume changes of gas within an enclosed space such as the thorax, abdomen or sinuses, occurs as a result of changing atmospheric

pressure and may give rise to symptoms; embarrassment may be caused by patients with an offensive discharge such as a colostomy; respirators may be required for the ventilation of patients with respiratory paralysis etc., and a host of other problems may occur which are not covered by this chapter.

131. There is, however, one remaining problem related directly to oxygen which affects all travellers by air whether they are healthy passengers, patients or air crew and that is a sudden failure to maintain cabin pressure when the aircraft is flying at high altitudes. This is a very rare emergency but it is a very important one since at the altitudes flown by modern jet aircraft, the time of useful consciousness after sudden complete failure of cabin pressure may be measured in seconds. Sudden complete failure is fortunately very unlikely, but it is vitally important for the flight crew of the aircraft to have immediate access to oxygen to enable them to bring the aircraft down to an altitude of 10,000 ft., at which level the degree of hypoxia while breathing air is acceptable in an emergency. At altitude greater than 33,000 ft., breathing even 100% oxygen fails to prevent hypoxia and a positive pressure mask has to be supplied for the air crew to enable them to bring the aircraft down to safe levels. The routine practised during flight in civilian aircraft varies with the altitude but at 30,000 ft. or higher, the flight crew have 100% oxygen with a pressure mask immediately available in case there is any pressure failure. For all passengers, there is a special installation operated by an aneroid valve which provides oxygen immediately the cabin altitude exceeds 14,000 ft. Oxygen stowages open automatically and masks are presented to each individual for immediate use and this should serve to cover the emergency until the pilot can bring the aircraft down to a safer level. In addition to this, aircraft usually carry emergency oxygen supplies for approximately 10% of passengers, to cover unexpected illness, but where there is foreknowledge of any special requirement for a patient, additional equipment is readily available. Nowadays very few invalids have to be rejected for air travel on grounds of the adverse effects of altitude.

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CHAPTER 8

Oxygen in Resuscitation [1]

132. "Resuscitation may be defined as the reversal of the process of death" (Swann, 1953). This may be achieved if oxygen is supplied to the vital centres of the brain before oxygen lack results in irreversible damage. Total anoxia of the cerebral cortex in man probably causes death within 3 to 6 minutes (Greene, 1962), but it is more important to realise that a delay of a few seconds in starting resuscitation can change certain success into certain failure, (Swann and Brucer, 1951). Thus any method of resuscitation must be applied with great urgency. In this respect, therefore, undue reliance on apparatus may do considerable harm. Immediate artificial ventilation with expired air has saved lives that would have been lost if the time had been wasted in obtaining resuscitation apparatus and oxygen.

133. Respiratory resuscitation should be attempted in emergency situations where there is upper airway obstruction, absence of breathing, cyanosis or irregular breathing in the unconscious patient. It is far better to start artificial ventilation when it is not needed than to miss a chance to save life. There is now no doubt that expired-air ventilation is the method of choice in first aid, (Whittenberger, 1962). Descriptions of the technique are given in First Aid (1965), the manual of the St. John Ambulance Association, the St. Andrew's Ambulance Association and the British Red Cross Society. We wish only to emphasise two points. The two-handed tilt, consisting of hyperextension of the neck and an upward pull on the jaw, are essential to prevent obstruction to the airway. If the rescuer doubles his depth of breathing he exhales about 12 litres/minute of gas containing 18·5% oxygen. Studies on anaesthetised and paralysed volunteers have shown that normal arterial oxygenation can be obtained by this method (Elam *et al*, 1958). The world-wide success of expired-air resuscitation in actual practice is incontrovertible.

134. If expired air is sufficient for resuscitation, particularly if the rescuer doubles his tidal volume, what place has oxygen? If the rescuer is tempted to await oxygen supplies and apparatus, and not to start expired-air ventilation immediately, the answer must be emphatically none. However, provided this danger is avoided, oxygen does have a place, in our view. It is inevitable that the "professional rescuer", be he ambulance man, industrial first aid worker, nurse, or doctor, will be involved most frequently at second hand in attempts at resuscitation. When he arrives, it is quite probable that expired-air ventilation will already be underway. If the professional rescuer carries apparatus, its use can only be justified if it is more effective than resusctiation by expired air. Oxygen is no panacea in saving life. The need in respiratory resuscitation is for ventilation of the lungs, both to provide oxygen and to remove carbon dioxide. Simple administration of oxygen, without adequate ventilation, is

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[1] The term 'resuscitation' can cause confusion as it is used in everyday speech and can even be applied to the simple use of brandy or smelling salts. It now has a definite connotation in emergency and first aid work and always means the urgent commencement of some method of assisted ventilation. In recent years external cardiac massage is also carried out in certain cases. The giving of oxygen alone without assisted ventilation is not considered to be resuscitation in this context.

dangerous and we recommend that this practice has no place in the treatment of respiratory failure.

135. The requirements for effective respiratory resuscitation are two fold, firstly to ensure a patent airway, and secondly to inflate the lungs. The two-handed tilt is an effective method of achieving a patent airway (Greene, 1962), but it can be tiring to maintain over long periods. Artificial aids may be necessary and vary from the insertion of a simple oropharyngeal airway, or nasopharyngeal airway to endotracheal intubation by an expert, or even tracheostomy. The Guedel oropharyngeal airway is usually sufficient, but problems can arise in the semi-conscious patient, from laryngospasm, gagging and vomiting. Incorrect insertion can push the tongue into the pharynx, and for efficient use the correct size of airway for that patient should be used. The Brook airway (Brook and Brook, 1960) or Resusitube (Safar, 1958) both have a shortened oropharyngeal airway. A nasopharyngeal airway is simple to insert, and is tolerated well by the semi-conscious patient. Nasal bleeding during insertion can be embarrassing to the doctor but is very rarely serious.

136. Once a patent airway is provided, by one of these devices, or by positioning of the head, an apparatus for inflating the lung can be used. In the event of this apparatus failing, the attendant must realise that his own expired air can be life-saving. The bag and mask resuscitator, introduced by Ruben and Ruben (1956), is in wide use, and is very effective. The models of this apparatus used by the St. Andrew's Scottish Ambulance Service are discussed in Chapter 11. These resuscitators have the great advantage that the operator is near the patient's head, where he can maintain neck extension to keep a patent airway and correct any leaks around the mask. Furthermore, the sensation during compression of the bag helps to detect airway obstruction or changes in lung stiffness, and allow the operator to change the volume and rate of inflation. The Ambu bag (Ambu International) can develop a pressure of 40 cm. water as a maximum, (Spence, 1962) and this avoids damage to adult lungs, although it can be dangerous to an infant's lungs. By addition of a reservoir tube of 500 mls. capacity to the air entrance port of the Ambu bag, an inspired oxygen concentration of about 76% can be achieved with an oxygen flow of 10 litres/minute, or about 50% with an oxygen flow of 4 litres/minute (Saklad and Gulati, 1963). When 15 litres of oxygen/minute were given by manual ventilation with an Ambu bag modified in this way, an arterial PO_2 of over 400 mm Hg. was achieved in curarised surgical patients, and at 8 litres of oxygen/minute the arterial PO_2 was over 200 mm Hg. (Heller, Watson and Imredy, 1968). These figures compare favourably with those obtained when ventilation was carried out with pure oxygen using a conventional anaesthesia gas machine (Heller and Watson, 1961). It is apparent that this simple modification to a bag and mask apparatus can produce very high levels of arterial oxygen tension, if these are required. Such equipment is not yet commercially available, but we recommend that manufacturers should consider these modifications. Hand bellows resuscitators (Lucas *et al*, 1959) appear to us to have no advantage over the bag and mask apparatus, and they do not allow the operator to detect changes in lung stiffness or airways resistance so readily. We do not recommend the use of hand bellows resuscitators.

137. Automatic cycling ventilators are frequently employed in resuscitation. All machines suffer from one great disadvantage, in comparison with the bag

and mask, in that they cannot compensate for changes in the airways resistance or lung stiffness, or give immediate evidence of leaks. An attendant is therefore always necessary, and if the attendant is there, he can use in preference a bag and mask resuscitator. This is not unduly tiring to use, as the daily practice of anaesthetists testifies. Of the available automatic ventilators, time or volume cycled machines are less effected by changes in both lung stiffness and airways resistance than the pressure cycled machines (Radford and Whittenberger, 1962). The two machines in common use in the United Kingdom, the Stephenson Minuteman Resuscitator (British Oxygen Co.) and the Automan Resuscitator (Siebe Gorman Ltd.) are described in Chapter 11.

138. Measurements of the arterial PO_2 in an emergency prior to resuscitation are unknown to us, and we cannot therefore present evidence to support our belief that oxygen enrichment of the resuscitating gas is desirable. However, some experimental work is relevant. Failure to take a deep breath over several minutes is known to lead to alveolar collapse, at least in animals, and the hypoxaemia following general anaesthesia in man has been thought to result from this mechanism (Chapter 3). Again the inhalation of small volumes of blood, alcohol, saliva or water causes marked arterial hypoxaemia in anaesthetised sheep (Halmagyi, Colebatch and Starzecki, 1962). Crush injuries of the chest may cause ventilatory failure with both hypoxaemia and carbon dioxide retention (Chapter 3). It is also quite possible that the victim may have pre-existing respiratory disease.

139. Burns of the respiratory tract carry a high mortality. They arise particularly when the victim is exposed to fire in an enclosed space, and the only clinical sign may be facial burn. If pulmonary damage has occurred it may be several hours before it causes arterial hypoxaemia. Oxygen in concentrations of 40%, with high humidity may be life saving in the later stage of treatment. Positive pressure ventilation will be needed if pulmonary oedema develops, but it should be remembered that these patients are extremely susceptible to fulminating respiratory infections (Editorial, Brit. Med. J., 1967), and great precautions to maintain sterility are required.

140. The main hazard from smoke in a fire is that it renders escape difficult by reducing visibility, but in addition air in burning buildings may contain toxic products of combustion. Deaths from exposure to gases from a fire have been largely attributed to carbon monoxide, and this was thought to be a major cause of death in up to 40% of victims. Nitrogen oxides, hydrogen cyanide, formic acid, sulphur dioxide, ammonia, benzene, phosgene and many others are all known to be potential toxic products that may be obtained from burning materials used in buildings and furnishings, but very little is known about these hazards in practice. Carbon monoxide is known to be produced in quantities when a fire occurs in an unventilated space. It has been calculated that the contents of a linen cupboard burning with its door ajar could fill a house with a dangerous concentration of carbon monoxide. In view of this hazard, we suggest that fire service personnel be instructed in the use of a bag and mask resuscitator, with supplementary oxygen, as described under the treatment of carbon monoxide poisoning in Chapter 9.

141. Circulatory arrest, resulting from either ventricular fibrillation or cardiac asystole, is now recognised as a common cause of death in acute myocardial infarction. Circulatory arrest can also rarely occur, of course, in numerous

other situations including anaesthesia, surgery, anaphylaxis, following the intravenous injection of various drugs, angiography and electrocution, etc. (Stephenson, 1964). Circulatory arrest is diagnosed by the absence of two or more arterial pulses (radial and carotid, or carotid and femoral) in the unconscious patient, or by failure to correct deep cyanosis by 6 inflations of the lung by expired air, in a patient who is not breathing. Closed chest cardiac massage, (Kouwhenhoven, Jude and Knickerbocker, 1960) and expired air ventilation are now standard first aid practice in the treatment of circulatory arrest (First Aid, 1965). The main achievement of the modern coronary care unit in hospitals is the rapid recognition and treatment of circulatory arrest, particularly the correction of ventricular fibrillation by D.C. countershock (Lawrie *et al*, 1967). In less favourable circumstances, however, closed chest cardiac massage may be needed for some time, until definitive diagnosis and treatment of the cardiac arrhythmia is available. Closed chest massage only produces a very low blood flow (Mackenzie *et al*, 1964), and preservation of vital brain centres is critically dependent upon adequate oxygenation of the blood. During chest compression, a large proportion of the blood that flows through the lungs does not partake in gas exchange (Gilston, 1965). Oxygen enrichment of the resuscitating gas is highly desirable in these circumstances, for even with 100% oxygen the arterial PO_2 may be less than 100 mm Hg.

142. In summary, therefore, we are of the opinion that oxygen enrichment of the resuscitating gas is an added advantage in most patients requiring respiratory resuscitation. Nonetheless there will be no benefit from such oxygen therapy if expired-air ventilation is withheld in an emergency pending the arrival of suitable apparatus.

143. We suggest that the "professional rescuer" should carry oxygen. However, we are not persuaded that automatic cycling ventilators, driven by compressed oxygen, are the best means of providing oxygen in these circumstances. These machines are heavy, rather complicated to use, and expensive. In our view the simple bag and mask resuscitator, with either an oropharyngeal or nasopharyngeal airway and an additional supply of oxygen constitute the best equipment. This equipment can be easily carried, along with a simple foot operated aspirating pump for clearing fluid from the airway. Oxygen can be supplied from a separate cylinder, for example, in the ambulance during a journey, but, in addition we feel that a small lightweight cylinder of oxygen, holding not less than 40 litres, should be included as an integral part in the bag and mask equipment. This matter is further discussed in Chapter 11, where the matter is suggested to fall within the remit of the proposed Working Party on oxygen equipment for the General Practitioner (Chapter 4).

144. The following procedure is recommended for respiratory resuscitation:

(a) Recognise the need for resuscitation.

(b) Open the victims mouth and scoop out any obstruction, such as vomit, false teeth, etc.

(c) Hyperextend his neck, and lift his jaw forward.

(d) Start mouth to mouth or mouth to nose expired-air ventilation, the rescuer breathing twice as deeply as usual.

(e) If the arterial pulse is absent at two or more points or if deep cyanosis is not relieved by six satisfactory inflations of the lung, start closed chest cardiac massage, with six chest compressions to one lung inflation.

(f) When a "professional rescuer" arrives, he will insert a nasopharyngeal or oropharyngeal airway, but continue to keep the victims neck extended, and his jaw forward.

(g) He will then start to inflate the lungs with air, using bag and mask. Oxygen will be added to the bag, if it is available.

(h) Artificial ventilation and closed chest cardiac massage should continue, if necessary, until the patient reaches hospital.

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CHAPTER 9

Oxygen Therapy in Industry

145. There is today a wide range of industrial concerns from large organisations with excellent occupational health services at one end to small factory units often with little knowledge of the health implications of their work processes at the other. There are many factories in the latter category and in the United Kingdom most men and women employed in industry probably work in firms with a labour force of less than 500.

146. In industry any emergency may be dealt with initially and often, indeed, until the patient is despatched to hospital by a first-aid worker whose standard of training may vary very widely. Resuscitation apparatus is infrequently required and therefore the standard of maintenance may sometimes be low. One must also bear in mind the continuous sales pressure, particularly on the larger industries to purchase such apparatus. These background factors are on the whole more likely to determine the policy with regard to resuscitation in a particular industrial situation than medical and scientific arguments.

Statutory requirements relating to the provision of Oxygen Therapy in Industry

147. It is obviously of interest to note the references to the administration of oxygen in the Factories Act and legislation made under it:

(i) Section 30 of the Factories Act 1961 refers to work inside any confined space in which dangerous fumes are liable to be present and lists the various precautions necessary. These include the wearing of a suitable breathing apparatus. This may consist of a simple tube with the further end in the fresh air so that ordinary air is breathed, or else it may be a breathing apparatus with cylinders of compressed air or possibly oxygen. This breathing apparatus is clearly only a safety measure and is not a means of treatment. Clause 6 of the same Section 30 states that "there shall be provided and kept readily available a sufficient supply of breathing apparatus of a type approved by the Chief Inspector, of belts and ropes, and of suitable reviving apparatus and oxygen, and the apparatus, belts and ropes shall be maintained and shall be thoroughly examined at least once a month . . . by a competent person". As well as dealing with procedures for entering confined spaces in which dangerous fumes may be present this same section also covers the situation in which atmospheric oxygen may be deficient. Clause 6 states *inter alia* that "suitable reviving apparatus and oxygen" shall be readily available. Under Section 30 (7) it is also a statutory obligation to have a number of persons trained in the use of reviving apparatus where men are entering vessels which may have contained dangerous fumes.

(ii) In the Chemical Works Regulations (S.R. & O. 1922, No. 731) Regulation 6 states that there shall be provided in every works where dangerous gas or fumes is liable to escape a sufficient supply of (a) breathing apparatus, (b) oxygen and suitable means for its administration, (c) lifebelts.

(iii) Also under the Shipbuilding Regulations there must be provided in every shipyard ". . . . a sufficient supply of suitable reviving apparatus and oxygen" (Regulation 79).

148. The Chemical Works and Shipbuilding Regulations are obviously concerned with apparatus for resuscitation, but make no attempt to lay down specifications for the type of equipment, or for its maintenance. In addition to use in resuscitation, oxygen will occasionally be required for treatment of medical emergencies occurring at work, such as myocardial infarction with shock, pneumonia, pulmonary oedema, etc. (Chapter 3). However, the specific problems of oxygen therapy in industry are concerned with resuscitation, which may be required in:

(a) Carbon Monoxide Poisoning.

(b) Gassing by other Noxious agents.

(c) Poisoning by miscellaneous chemical agents.

(d) Electrocution, drowning, haemorrhagic shock, etc.

Carbon Monoxide Poisoning

149. Carbon monoxide is formed by the incomplete combustion of carbonaceous materials. Dangerous levels can arise in coke, oven plant, foundaries, gas-works, steel-works, and wherever blast furnaces, gas producers, and water gas plant are operated. Improper use of water heaters (geysers), boilers or braziers, or overheating of compressors can also lead to carbon monoxide formation. Dangerous concentrations may result from leakages of gaseous fuels. Carbon monoxide may occur in mines as the result of explosion of over-rich atmospheres of fire-damp and air and coal dust mixtures, from spontaneous heating and shot-firing. In addition to these industrial hazards, carbon monoxide is the toxic substance in the exhaust from petrol engines, and in domestic gas. It is also the best recognised danger in the gases produced by a fire in a poorly ventilated building (Chapter 8). The Scottish Gas Board inform us that carbon monoxide concentrations in the town gas in Central Scotland has fallen from 24% by volume in 1960, to 7% by volume in 1967. This results from the introduction of carbon monoxide shift reduction units on production plants, and it is the Board's policy to continue the installation of these units, so that the concentration is expected to fall below 5%, or even 2%, in future years. From 1970 onwards, natural gas from the North Sea will be incorporated into Scottish domestic gas, and the Board anticipate that during the following ten years the carbon monoxide concentration in domestic gas in Scotland will fall towards zero.

150. The factory Inspectorate recorded 4 deaths in the United Kingdom in 68 cases of carbon monoxide gassing in industry in 1965, whereas in 1945 there were 18 deaths in 218 cases. In comparison the Inspectorate were notified of 79 gassings by chlorine in 1965, of 10 gassings with nitrous fumes and 15 with trichlorethylene, but no deaths from any of these gasses. In comparison, gassing accidents from carbon monoxide are much more frequent in the home. The total number of domestic accidental deaths from coal gas poisoning fell from 195 in 1955 to 86 in 1966 in Scotland. Gassing by carbon monoxide has not notably participated in the great increase of "Self poisoning" in recent years (Matthew, 1966), suicidal deaths from coal gas poisoning falling from

182 deaths in 1955 to 118 in 1966, in Scotland. It is important to point out, however, that although domestic gas will continue to become less lethal as the carbon monoxide concentration falls in future years, there is no immediate prospect that exhaust gas from petrol engines will become any less toxic. When the carburettor of a petrol engine is set for maximal power, the exhaust gas can contain up to 10% carbon monoxide.

151. Carbon monoxide kills by interfering with oxygen transport in the blood. It combines with haemoglobin, as does oxygen, but the affinity of haemoglobin for carbon monoxide is some 200–300 times that for oxygen. In addition the carboxhaemoglobin so formed interferes with the function of the remaining haemoglobin. A gassed workman with only 40% of his haemoglobin uncombined with carbon monoxide is in grave danger of death, unlike a patient with chronic anaemia and only 40% of the normal haemoglobin concentration (Haldane and Priestley, 1935). The dangers of carbon monoxide are related to both the duration of exposure and the concentration in the atmosphere. An exposure to 0·1% for 90 minutes can endanger life (Henderson and Haggard, 1927).

152. The combination with haemoglobin is reversible and carbon monoxide can rapidly be cleared from the blood if a high arterial PO_2 can be obtained. In addition oxygen is urgently needed to restore aerobic metabolic pathways in the body cells. These physiological facts underlie the treatment of carbon monoxide poisoning. The first imperative necessity is to remove the victim to fresh air as soon as possible, and then to start expired-air mouth to mouth ventilation as soon as any airway obstruction is removed. There is no risk of poisoning to the rescuer by using mouth to mouth ventilation.

153. The subsequent treatment has been controversial, particularly in regard to the role of carbogen (5% CO_2 and 95% oxygen, or 7% CO_2 and 93% oxygen) as opposed to 100% oxygen, as a resuscitating gas, (Donald and Paton, 1955; Marriott, 1955; Medical Research Council, 1958; Marriott, 1958). All authorities agree that carbon monoxide removal is principally dependent upon lung ventilation, and the arguments have centred on the value of added CO_2 in stimulating ventilation. Animal studies have shown that 5% CO_2 and 95% oxygen can remove carbon monoxide from the blood more rapidly than by 100% oxygen (Killick and Marchant, 1959). However, 7% CO_2 and 93% oxygen has no advantage over 5% CO_2 and 95% oxygen, in this regard (Douglas *et al*, 1961). It must be stressed, however, that CO_2 is mainly of value by reason of the increase in ventilation which it produces. An additional theoretical advantage would lie in the increased cerebral blood flow following CO_2 inhalation.

154. On consideration of this problem, particularly of the practical difficulties of providing carbogen for use only in this specific poisoning, and the difficulties of training first aid workers to recognise carbon monoxide poisoning, we recommend that carbogen should be provided for the first aid treatment of gassing by carbon monoxide only in situations where carbon monoxide gassing is the sole hazard. This would apply in coke oven plant, foundries, gas-works, steel-works and where blast furnaces, gas producers and water gas plant are operated. In other situations, including most coal-mines, the specific risk of gassing by carbon monoxide does not appear to be very much greater than that from other noxious gases. We would propose that as the

main aim of carbogen is to increase ventilation, this can be achieved with almost equal efficiency by hand ventilation of the gassed victim with a bag and mask resuscitator to which an oxygen supply can be attached. With a reservoir tube on the inlet 76% oxygen can be given by this apparatus with oxygen flow of 10 litres/minute (Chapters 8 and 11). We recommend that such apparatus should be available in every ambulance, and in industrial first aid centres when it becomes commercially available. The proposed Working Party on oxygen equipment for the General Practitioner could include this within its remit. Furthermore the treatment of poisoning by other noxious gases (paras. 156-161) would then be identical at the first aid level with poisoning due to carbon monoxide poisoning. The bag and mask resuscitator (with oxygen) could be used to hyperventilate the patient, and thereby promote the increased excretion of carbon monoxide which is the main merit of treatment with carbogen.

155. Some rare cases of carbon monoxide poisoning have been reported where prolonged coma was associated with a metabolic acidosis and a state of spontaneous hyperventilation (Leathart, 1962). The administration of carbogen to such patients would undoubtedly increase their acidosis. The first-aid treatment of carbon monoxide gassing by means of artificial hyperventilation with oxygen, from a bag and mask resuscitator fitted with a reservoir tube on the inlet, will only be easy to apply in unconscious patients. However, it is particularly these patients, with a carboxy-haemoglobin concentration in the blood of 40% or more (Ministry of Labour, 1965) that are in the greatest danger of death.

Other Noxious Gases

156. The other noxious gases encountered in industry may be considered in two main groups in so far as their toxic effects on man are concerned. The gases in the first group produce rapid unconsciousness and respiratory failure, if the victim is exposed to high concentrations. In these cases respiratory resuscitation is required by expired-air artificial ventilation, as discussed in Chapter 8. In the second group the principal effect is on the mucosa lining the airways and the lung alveoli, with the production of pulmonary oedema which may be delayed in onset after the exposure.

157. The first group includes the simple asphyxiants (Hunter, 1961) nitrogen (black damp) and methane (fire damp), which produce hypoxia by dilution of the available oxygen in air. These gases may be encountered in mines, enclosed spaces such as ships' holds, near blast furnaces and in agricultural silos. The asphyxia can be very insidious, and rescuers without breathing apparatus are also exposed to great risk, particularly as hypoxic symptoms are accelerated by muscular exertion. Carbon dioxide is a toxic agent in its own right, and an atmosphere of 20% carbon dioxide and 80% oxygen will cause unconsciousness.

158. The chemical asphyxiant, hydrogen sulphide, can produce rapid respiratory paralysis if the victim is exposed to high concentrations. This gas (stink damp) is produced by the decay of organic matter, and it is found in mines, sewers, vats and tanneries, as well as in the chemical manufacturing industry. Narcotic agents which produce general anaesthesia and respiratory depression include the vapours of many organic solvents, notably trichlorethylene and carbon tetrachloride. Chloroform, cyclopropane, butylene, methyl

chloride, nitrous oxide, petroleum fume, carbon disulphide, benzene and zylene, all share a narcotic action in high concentrations. Prolonged exposure to low concentrations of many of these agents may result in other effects, such as damage to the liver, kidneys or bone marrow, and these may well be of greater importance than the respiratory effects.

159. It must be emphasised that if the rescuer himself inhales these toxic atmospheres he too may become a victim. The first need in resuscitation is to remove the victim to fresh air. If this is not possible, or entails a long delay, artificial ventilation with respirable air is needed in the toxic atmosphere. This can be provided by the rescuer's expired air, if he has some form of breathing apparatus which is modified for this purpose, or alternatively a bag and mask resuscitator can be used for a short period if it is fitted with a filter on the air intake which is effective for that particular gas. However, it is in these circumstances, such as mining accidents, with a long inevitable delay in evacuation, that an automatic cycling respirator, driven by oxygen, is particularly valuable.

160. The second group of gases includes the chemical irritants (Hunter, 1961). Sulphur dioxide, ammonia, chlorine, nickel carbonyl, nitrogen dioxide (nitrous fume) and phosgene are all well known to cause pulmonary oedema. The most dangerous are those least soluble in water, and a delay of 6-12 hours after exposure in the onset of pulmonary oedema is characteristic of phosgene and nitrous fume gassing. Methyl bromide, used as a refrigerant, fumigant and fire-extinguisher, is very poisonous and causes pulmonary oedema in addition to central nervous system involvement.

161. Pulmonary oedema from chemical irritation of the lung will require oxygen in high concentrations, as in any other type of pulmonary oedema (Chapter 3). These patients should, of course, be treated in hospital. An interesting account of the therapeutic and administrative problems of treating many simultaneous cases of acute chemical injury to the airway and lungs is given by Conner, Du Bois and Comroe, (1962). A quiescent period of six hours was followed by oedema of the airway mucosa followed by atelectasis and bronchopneumonia, resulting in hypoxaemia. Mechanical respiration and intensive nursing care resulted in 50% survival in six cases with severe lung damage. This incident arose when a tank of Dow 421, an organic commercial solvent, burst in a narrow courtyard. It is a sobering thought that the widespread use of road tankers in the bulk transport of chemicals could possibly result in large numbers of the general public being victims of a similar disaster, although the risk would be less if the gas were released in the open-air.

Miscellaneous Chemical Agents (Hunter, 1961)

162. In addition to use in resuscitation from gassing accidents, oxygen is also recommended in the treatment of poisoning by numerous chemical agents encountered in industry. Aniline, nitro benzene, dinitrobenzene and trinitrotoluene are all absorbed to some extent through the skin, and all produce conversion of haemoglobin to methaemoglobin. This compound does not combine reversibly with oxygen, and oxygen transport by the blood stream is thereby impaired. Methaemoglobin causes the intense cyanosis of patients seriously poisoned by these substances. In these cases the immediate first aid measure must be to remove clothing and wash the skin, to prevent further

absorption. Respiratory resuscitation may be needed for some of these substances produce respiratory depression after an initial period of central nervous system stimulation.

163. The insecticide parathion and other organo-phosphorous compounds are powerful cholinesterase inhibitors, but poisoning can result in pulmonary oedema, in addition to neuro-muscular paralysis. Intramuscular atropine is the first aid measure of choice in this situation. Advice on the details of treatment for poisoning by these agents is available from the Chemical Defence Experimental Establishment, Porton Down, Salisbury. Oxygen is advised at the first sign of pulmonary oedema. The complex salts of platinum can produce "platinum asthma" in susceptible workers, and oxygen and adrenaline may be necessary in treatment. Poisoning by hydrogen cyanide is treated by the emergency inhalation of amyl nitrite, or by the intravenous injection of 0·3 grams of sodium nitrite over 10 minutes followed immediately by 25 grams of sodium thiosulphate injected at the same slow rate. Oxygen therapy has no substantiated place in this therapeutic regime (Beards, Knowles and Bain, 1964).

164. Substances encountered in industry are constantly changing, new toxic hazards appear and well-known dangers are removed by substitution of new materials. The indications for oxygen therapy discussed above will certainly become outdated. The Ministry of Labour, H.M. Factory Inspectorate, publicise information on industrial hazards, and how to avoid and treat them in numerous ways. They publish booklets on Safety, Health and Welfare, and Methods for the Detection of Toxic Substances in Air. The Poisons Information Bureaux (Scottish Bureau at the Royal Infirmary, Edinburgh — Telephone 031-229 2477) provide information on specific dangers of poisoning and recommended first aid measures and definitive medical treatment in emergency situations.

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CHAPTER 10

Oxygen Therapy in Treatment of Drowning

165. In the United Kingdom approximately 2,000 people die each year from drowning. In the Royal Life Saving Society's analysis of 1,327 fatal drowning accidents in 1961, most of the cases were under 25 years of age. Just over 75% of these fatal drowning accidents (as distinct from suicides) occurred in fresh water. As a cause of death in young persons, therefore, drowning is second in importance only to road accidents. These figures reveal a problem of considerable national importance. The disturbances in function which occur in drowning are not widely known, and the place of oxygen therapy in the treatment of this common emergency merits discussion.

The Pathological Physiology of Drowning

166. Much of the knowledge of the physioligical disturbances in drowning has been derived from animal studies and there must be some caution in applying these results to man. Drowning is a far more complex affair than mere asphyxia, although gas exchange in the lungs does cease rapidly, so that progressive hypoxia, hypercapnia and acidosis ensue. Water is kept from entering the lungs at first by repeated swallowing and by spasm of the glottis. Large quantities of water enter the stomach and often cause vomiting. Glottic spasm and swallowing are reflex in nature, and consciousness can be lost without any water entering the lungs. This is known as dry drowning, and possibly occurs in 20-40% of cases.

167. Eventually, water does enter the lungs, as the reflex activity diminishes with loss of consciousness. In the case of sea water drowning, the fluid is strongly hypertonic to blood and is drawn from the blood into the alveoli, and haemoconcentration results. Damage to the lungs causes severe pulmonary oedema. The casualty from sea water drowning will often have a fine froth at the lips, revealing his pulmonary oedema. In fresh water drowning the fluid is hypotonic to the blood, and the water is drawn into the blood stream. In animals, this is shown to result in profound haemodilution and haemolysis, with a rise in serum potassium and death from ventricular fibrillation. Practical experience in fresh water drowning in man, however, has not shown such severe haemodilution. It is possible that the pulmonary surfactant protein which lines the alveoli acts as a barrier to fluid exchange in man when fresh water fills the alveoli although we know of no experimental work on this topic. In the rare instances of near drowning in chlorinated water (as in swimming baths), the problem has been the late development of pulmonary oedema, possibly due to the irritant action of chlorine.

168. The clinical picture of human near drowning differs little between fresh water and salt water drowning. In most cases cyanosis, breathlessness and basal crepitations are present and some neurological features occur, such as restlessness, confusion, unconsciousness or fits. Pulmonary oedema is common both in salt water and fresh water near-drowning in man. In some cases apparent recovery following resuscitation is followed by the rapid development of respiratory distress and death from pulmonary oedema. This situation is known as "secondary drowning". It can be anticipated by the appearance

of diffuse mottling on a chest X-ray taken during the phase of apparent recovery. An important feature in the convalescent phase is the risk of serious pulmonary infection or chemical pneumonitis from microbial or chemical contamination of the water. Although the neurological deficits in patients who have been resuscitated may initially appear to be very grave, case reports have shown that marked improvement in such signs can occur. The danger of preserving function of the heart and lungs by resuscitation, only to be left with severe permanent brain damage, would appear to be very remote in practice.

Treatment of Near Drowning

169. All workers now agree on the superiority of expired-air mouth to mouth, or mouth to nose artificial ventilation over all other manual methods. Mouth to mouth ventilation must be the first, urgently applied treatment, preceded only by removal of gross airways obstruction, for example, by inhaled false teeth, weed, or solid vomitus. Time should not be lost in attempting to drain fluid from the lung, as nearly all the fluid in fact comes from the stomach (Donald, 1955). Of course a patent airway must be maintained, as discussed in Chapter 8. All first-aid workers should be instructed in the use of expired air resuscitation (the "kiss of life"), and the need for urgency in starting this treatment must be emphasised. Many cases are known where untrained laymen have succeeded in saving life by this means and education of the general public in this method is to be strongly encouraged.

170. The place of closed chest cardiac massage in the resuscitation of the apparently drowned is not entirely clear, as the incidence of remediable circulatory standstill is not known. Undoubtedly most patients who are saved at present do not require this procedure. Furthermore there is a considerable risk of fracturing ribs or damaging the internal organs, particularly if the technique is applied by untrained people (Klassen, 1963; Smith, 1965). It seems wise to use closed chest massage only when cardiac arrest has been positively diagnosed, by the absence of two or more arterial pulses or by failure to correct deep cyanosis by five or six inflations of the lungs by expired air ventilation. We recommend that closed chest cardiac massage should only be used by professional rescuers, that is doctors, nurses, or first-aid workers who are trained in its use, and then only after they have made a diagnosis of cardiac arrest. Although a published series of cases is not yet available it has been reported by several workers that in some cases of cardiac arrest a proper action of the heart may be restored by a blow on the precordium, using the ulnar border of a clenched fist (Scherf and Borneman, 1960; Semple *et al*, 1963). This harmless and simple procedure could well be more widely adopted at the start of mouth to mouth respiration or during resuscitation.

171. Only when these emergency measures are instituted, should any time be spent on bringing apparatus to assist in artificial ventilation. We feel that there is a case for the addition of oxygen to the ventilating gas at this stage, and this could be provided by a bag and mask resuscitator, with additional oxygen either from an external source, or as an internal part of the bag and mask resuscitator (Chapters 8 and 11). Treatment which involves apparatus must always be regarded as secondary in importance to emergency measures of mouth to mouth ventilation and closed chest cardiac massage since the issue of life or death may be decided in seconds rather than minutes.

172. When more facilities become available following hospital admission, tracheal intubation, with suction or bronchoscopy, will precede automatic ventilation by intermittent positive pressure. Ventricular fibrillation can now be treated very effectively by D.C. countershock, but this is most likely to be effective in the well oxygenated patient, in whom any acidosis has been treated with intravenous sodium bicarbonate. Correction of electrolyte imbalance (hypernatremia and haemoconcentration) particularly in sea water drowning, would follow treatment of ventricular fibrillation. Gastric aspiration may be needed to remove large quantities of swallowed fluid. In the unusual event of marked haemolysis developing in fresh water drowning, fresh blood transfusion or even exchange transfusion may be needed. Antibiotics to prevent or treat bacterial pneumonia should also be given.

173. The use of oxygen at this stage will be to obtain adequate oxygenation during assisted ventilation and later when spontaneous respiration restarts, to correct hypoxia resulting from pulmonary damage or oedema. In animal work it has been shown that continuation of blood flow in areas of non-ventilated lung is the physiological mechanism of hypoxia. This occurs as a result of intense constriction of the small terminal airways, which has been shown to be a reflex response to the presence of fluid in the lungs (Colebatch and Halmagyi, 1961, 1962). Reflex bronchiolar constriction can be overcome by isoprenaline, (Halmagyi *et al*, 1964) and there is experimental support for adding an isoprenaline spray to treatment with intermittent positive pressure respiration and 100% oxygen, in the severely hypoxic victim of near drowning. The use of 100% oxygen continuously for more than 12 hours does run a risk of producing pulmonary oxygen toxicity (the Lorrain-Smith effect). It is unlikely that continuous artificial ventilation with high concentrations of oxygen would be needed for longer periods.

174. It may well be asked if all this effort is either necessary or effective. This cannot be answered by reference to the results of treatment in any series of cases, but some graphic accounts in the literature would strongly suggest that such treatment can indeed save a life which would otherwise be lost. Kvittingen and Naess (1963) describe the successful resuscitation, without residual neurological damage, of a 5-year-old boy who was totally submerged in a partially frozen Norwegian river for 22 minutes. The child was treated with expired air ventilation, external cardiac massage, blood transfusion, exchange transfusion, intermittent positive pressure ventilation, and digitalis for pulmonary oedema. Despite prolonged unconsciousness and convulsions, followed by a period of temporary decerebration and apparent blindness, six months later he was physically normal, and apart from diminished peripheral vision, his nervous system and mental function were almost normal. Such a dramatic story of restoration of life to the apparently dead does, indeed, show that the effect can be worthwhile.

175. In conclusion, oxygen therapy, with or without intermittent positive pressure plays only a small part in the complex, modern treatment of near drowning. Although the full treatment will be only needed for the very ill patient, it is important to realise that hospital in-patient observation for at least 24 hours is desirable for all cases of apparent drowning because pulmonary oedema or infection can be serious late sequelae.

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CHAPTER 11

Apparatus for Oxygen Administration [1]

176. An increase in the inspired oxygen concentration can be achieved by use of oxygen masks, nasal catheters and oxygen spectacles, or by placing the patient in an oxygen tent, or in an incubator in the case of infants. It is also possible to fill the whole room with the desired concentration of oxygen, but this is very expensive with methods available at present and not without danger. The efficacy of all these methods must be judged on three grounds, the level of inspired oxygen achieved, the accuracy of control of this concentration where this is important and the comfort of the patient when receiving the therapy.

Oxygen Cylinders

177. Oxygen is supplied either by a pipeline from a central source, or from compressed gas in cylinders at the bedside. All oxygen for medical use in Scotland is supplied by the British Oxygen Co. who publish a helpful booklet entitled "Oxygen Therapy" (1960). Oxygen cylinders are painted black with a white top, and the 48 cu. ft. and 120 cu. ft. capacity are the cylinders most commonly used. These are filled to a pressure of 1,980 lbs. per sq. in., and have a bull nosed valve fitting. Each cylinder is hydraulically tested by the firm which fills it at 5 year intervals under the Gas Cylinder (Conveyance) Regulations, SRO 1931, No. 679. We feel that if manufacturers indicated on oxygen cylinders their capacity in Metric as well as Imperial units this would prove very helpful to clinicians. If the pressure gauge indicates the pressure in atmospheres in the cylinder then the clinician could easily work out the amount of gas in the cylinder (pressure in atmospheres multiplied by capacity in litres).

178. The charge for filling a cylinder with oxygen varies with the frequency with which refills are required. From 1st January, 1968, the charge made by the British Oxygen Co. for filling a 48 cu. ft. cylinder varied from 15/- to 8/-, the lower cost applying to large consumers of oxygen, such as large hospitals, and from 18/4d to 11/3d for a 120 cu. ft. cylinder. The charge for hiring a cylinder from the British Oxygen Co. is 2/6d per month at the highest rate, or on their "commuted rental" system £1 for one year, £1 10/- for 2 years and £2 for 3 years, these charges being the same for either size of cylinder. It would appear that at present on price alone the 120 cu. ft. cylinders are more economical. However, these large cylinders are very heavy (130 lbs. when full) and cannot be easily carried upstairs by one man. They are therefore generally unsuited for domiciliary use, unless the chemist who undertakes delivery has sufficient staff to carry them. Large cylinders (240 cu. ft. at 25/9d to 18/4d) appear to be particularly suitable for hospitals where bulk supplies of liquid oxygen are not available. We understand that the British Oxygen Co. would prefer to supply 48 cu. ft. cylinders for domiciliary practice. The company cannot guarantee that the hire or refill charges will remain unchanged.

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1 The names and addresses of all commercial firms mentioned in this Chapter are contained in Appendix VII.

179. Oxygen is also supplied for industrial use in cylinders and it is possible that "industrial oxygen" may occasionally be used for medical purposes. The British Oxygen Co. supplies oxygen for both medical and industrial purposes which contains 99·5% or more pure oxygen. The British Pharmacopoeia standard for medical oxygen is 99·0%V/V, with the residue consisting of argon, and traces of nitrogen or of hydrogen. Industrial oxygen cylinders are not evacuated entirely before refilling; they are not painted as frequently as medical cylinders and the valve fittings are in brass and not chromium plated. These cylinders are filled to 2,500 lbs. per sq. in. pressure, and could conceivably damage medical equipment intended for use with medical gas cylinders which are filled to 1,980 lbs. per sq. in. pressure. We would not recommend that "industrial oxygen" should be used for medical purposes as a routine, but there should be no hesitation in using this gas in an emergency if medical oxygen is not available.

Valves and Flow Regulators

180. In addition to the on/off control valve of the cylinder, a pressure reducing valve is required, and this is conveniently combined with a flow regulating device. The automatic pressure reducing valve supplied by the British Oxygen Co. reduces the pressure from 1,980 lbs. per sq. in. in the full cylinder, to an outlet pressure of about 7 lbs. per sq. in. and a fine adjustment control then regulates the flow in the range 0 to 10 litres/minute. This automatic valve maintains this flow rate until the cylinder is nearly empty. On the other hand, a fine adjustment valve alone will allow the flow rate to fall steadily as the cylinder pressure falls, and consequently requires continual re-adjustment to maintain a constant flow. The fine adjustment valve alone merely constricts the outflow of oxygen from the cylinder, and if the tubing "downstream" from this valve becomes blocked pressure will build up to that of the cylinder contents. This could cause any glassware in the circuit to shatter. It will be apparent therefore that an automatic pressure regulator is much to be preferred. Another reducing valve supplied by British Oxygen Co. in common use in hospital practice provides an outlet pressure of 60 lbs./sq. in., (maximum), and this requires flow regulation by an additional needle valve.

181. The rate of flow of oxygen from the reducing valve can be measured either by a dry bobbin flowmeter, reading from 1 to 10 litres/minute, and controlled by a needle valve, (as with the hospital type of reducing valve mentioned above) or alternatively one of two predetermined fixed flow rates can be selected. The latter system, when incorporated with an automatic pressure regulator, is known as a fixed flow regulator, and was orginally developed by the Medical Research Council. We are of the opinion that this type of device is most useful for domiciliary practice, for an unsupervised patient may easily use more oxygen than is necessary, or wise, if supplied with a more variable control valve. The present commercial forms of the fixed flow regulator deliver a flow of 2 litres/minute on the "Med" setting and 4 litres/minute on the "High" setting. The term "low" is presumably avoided on psychological grounds.

182. We feel that there is a case for reconsideration of these oxygen flow rates. As shown in Chapter 4, domiciliary oxygen therapy is usually required either to provide high concentrations of inspired oxygen, as in treatment of

pulmonary oedema, cardiogenic shock etc. or to provide lower concentrations of oxygen in the patient with chronic respiratory disease. In the first case, a flow rate of oxygen of 6 litres/minute is often needed, whereas in the latter, 2 litres/minute is adequate, and should not be exceeded. We propose that the fixed flow regulator be altered to provide 2 and 6 litres/minute in the "Med" and "High" positions. Furthermore, we think that it is desirable to provide some form of key to operate the switching system between the "Med" and "High" positions, so that the patient, who is not supplied with this key, cannot alter it himself. The regulator would, of course, still have an off position which the patient can use. The chemist who dispenses the oxygen could adjust the selector control with the key so that the patient could receive either 2 or 6 litres/minute according to the doctor's prescription. The chemist would, of course, provide an appropriate mask for use with the recommended dose of oxygen. These flow rates of 2 and 6 litres of oxygen/minute would be adequate for all the types of oxygen mask and nasal catheters discussed in this chapter. These lower flow rates, particularly the 2 litres/minute, for use with low concentration devices which one so often indicated in domiciliary practice (Chapter 4), will obviously have the important advantage of prolonging the duration of therapy obtained from one cylinder.

183. The tubing which conveys the oxygen to the face mask, or other device, is often very unsuitable. If this tubing is of thick walled rubber, it is often so heavy that the oxygen mask is dragged from the patient's face. The alternative of latex lightweight tubing suffers from the disadvantage that it is easily compressed by pillows, etc., and the flow of oxygen obstructed. An ideal tubing would probably be in lightweight plastic, not easily squashed, and yet not rigid enough to pull the mask from the face (such as the Argyle tubing, supplied by Eschmann Bros. and Walsh Ltd.). Lack of attention to this small detail often prevents the patient receiving the correct therapy. We suggest that manufacturers of oxygen therapy equipment should be encouraged to provide suitable tubing for use with their apparatus in both hospital and domiciliary practice, and to provide suitable connectors for oxygen outlets only, so that the tubing cannot be used easily for other purposes.

184. Since Joseph Priestly, who discovered oxygen, first recommended its use in treatment of human disease, numerous devices have been described for administering the gas to patients. The following discussion is confined to apparatus in common use in the United Kingdom in 1967.

Oxygen Masks: High Concentration Devices

185. The oronasal mask described by Boothby, Lovelace and Bulbulian (1938) consists of a close fitting face piece, with a rebreathing bag of about 500 mls. capacity. It is commonly known as the B.L.B. mask, and a mask which operates on the same principles, but fits over only the nose is also available. This mask yields high concentrations of oxygen with a flow rate from 6 to 8 litres/minute (Ball, 1963). The flow rate should be adjusted until the reservoir bag just does not collapse at the end of inspiration. According to Ball (1963), "significant carbon dioxide retention does not occur". However, this author notes that "rubber becomes very irritating to the skin after continuous contact for more than one hour". The mask needs to be applied firmly if high concentrations of oxygen are to be obtained. The rubber breathing

bag requires careful cleaning and powdering after use, whereas with disposable masks, there is no danger of infection. Commercial models of the B.L.B. mask are available with a plastic facepiece, (Oxygenaire), in addition to those with a rubber facepiece (J. G. Franklin and Sons Ltd.). The B.L.B. mask is not intended for the treatment of patients with chronic respiratory failure who may have retention of carbon dioxide.

186. A mask consisting of a double bag of polyvinyl chloride, with an elastic head cord, and malleable wire stiffener in the free edge which fits over the nose (Burns and Hall, 1953) is in wide use, and our survey of oxygen therapy in domiciliary practice showed that it was by far the most commonly used device for oxygen therapy in the home (Chapter 4). It is intended to provide high concentrations of oxygen. If the mask is properly fitted, as shown by the fact that the bag partially deflates with each breath, studies have shown that an inspired oxygen concentration of at least 60% is obtained at an oxygen flow of 6 litres/minute (Flenley, Hutchison and Donald, 1963; Green, 1967). Commercial models of these masks (Polymask, British Oxygen Co.) are cheap, and the mask is therefore regarded as disposable. Green (1967) found that it was relatively comfortable in use, and this appears to us to be the general opinion of patients who use it. Catterall and Snow (1960) have noted that a heavy delivery tube can cause kinking of the mask above the entry tube, particularly when the patient moves, and they found that this could result in an oxygen concentration of 21·6% at 6 litres/minute, instead of the 70% when the mask was in the ideal position. We also noted that kinking or twisting of the entrance to the mask is common when heavy tubing is used. Green (1967) has recorded a minimum carbon dioxide concentration of 0·4% when 6 litres of oxygen per minute were given, when using a mask of this type. We do not regard this as any particular disadvantage, as the mask is not intended for use in patients who may have carbon dioxide retention.

187. Another type of mask which is commercially available is a plastic bag with an inflatable rim, the oxygen supply being so arranged that the rim deflates and the mask falls from the face if the supply fails. This mask is marketed as the Oxygenaire "Pneumask". When 6 litres of oxygen per minute were given to 4 patients with this mask, Green (1967) reported that a mean inspired concentration of 69% was obtained, and this figure rose to 73% with 8 litres/minute. Bethune and Collis (1967) found similar values for alveolar oxygen concentration. Green (1967) reported a minimum CO_2 concentration in the inspired gas of 1·8%, and mean values "as high as 3%", with oxygen flow rates of 4 or more litres/minute. Bethune and Collis (1967) reported that the mask had an effective dead space of 70 mls. at a flow of 8 litres of oxygen per minute, and 170 mls. at 4 litres of oxygen per minute. In our opinion the rebreathing of CO_2, which these studies reveal, when the mask is used at these flow rates, is not desirable.

188. The M.C. mask (BXL Ltd.) consists of a transparent plastic cone, padded with plastic foam at the face, with oxygen entering at the apex of the cone (Catterall, 1960). Holes around the in-flow pipe allow for the inhalation of room air and for expiration. The oxygen concentration inside the mask is reported to rise to a mean value of 61% at a flow of 6 litres of oxygen per minute, with a range in 12 subjects from 53% to 74% (Catterall, Kazantis and Hodges, 1967). An alveolar oxygen concentration of 40% at 4 litres of oxygen

per minute, rising to 60% at 8 litres of oxygen per minute, was reported by Bethune and Collis (1967) with a tidal volume of 500 mls. Catterall (1960) reported that the carbon dioxide concentrations inside the mask were less than 1% during use. Catterall, Kazantis and Hodges (1967) reported that the carbon dioxide concentration within the mask fell rapidly to zero during inspiration, even when the mask was worn with the oxygen supply cut off. The oxygen delivery tube in this mask is held firmly, so that obstruction to in-flow of oxygen does not occur. The clear transparent plastic allows the patient's lips to be clearly seen during treatment. In our opinion, the reports described above show that the M.C. mask is an effective means of delivering a high concentration of oxygen of the order of 60% at 6 litres of oxygen per minute. The range of oxygen concentration within the mask (22% to 38%, mean 28% at 1 litre of oxygen per minute, and 26% to 52% at 2 litres of oxygen per minute) (Catterall, Kazantis and Hodges, (1967)) do appear to us to be rather too wide at these low rates of flow. We also note that Bethune and Collis (1967) found that this mask had an effective dead space of 110 mls. at 1 litre of oxygen per minute, and 80 mls. at 2 litres of oxygen per minute, which is similar to the findings of Catterall, Kazantis and Hodges (1967). The slight increase in ventilation necessary to remove carbon dioxide, is undesirable in the patient suffering from carbon dioxide retention. For these reasons we do not think that this mask is so suitable for use in controlled oxygen therapy of patients with respiratory failure.

Oxygen Masks: Low Concentration Devices

189. As explained in Chapter 3, patients with chronic respiratory disorders and CO_2 retention should be given oxygen by a device capable of delivering 25% to 40% oxygen, with an accuracy of $\pm$ 5% at any given level. The Venturi mask (Oxygenaire) was the first mask designed to achieve these aims (Campbell, 1960). It consists of a light aluminium cone, connected to the face by a plastic facepiece and elastic cord. The apex of the cone contains a small jet through which oxygen enters and air is entrained through adjacent ports, by the venturi effect. Supplementary oxygen is given through an adjacent wide bore pipe to vary the inspired oxygen concentration as required. Two expensive special gauges are required but the mask can be re-used if the plastic facepiece is renewed. The performance of this mask has been examined by Flenley, Hutchison and Donald (1963), who found that it produces concentrations of inspired oxygen within a range of $\pm$ 5%, from 25% to 35%. It now appears that several other devices which are cheaper and less complex can achieve a control of the inspired oxygen concentration which is at least as adequate as that given by this mask.

190. The Ventimask (Oxygenaire) has been developed from the original Venturi mask. It achieves adequate control of the inspired oxygen concentration, is cheaper, and requires no special gauges. The mask uses the venturi principle and entrains air efficiently so that it will deliver a fixed concentration of oxygen to within $\pm$ 1% of the stated level when the oxygen flow rate varies between 1 and 4 litres/minute, (Green, 1967). Three separate models of the mask are required to cover the range of 24%, 28% and 35%. The mask can be cleaned for further use, but is probably cheap enough to be regarded as disposable. It is comfortable, and CO_2 does not accumulate because of the high flow rate of air and oxygen through the mask (Campbell and Gebbie, 1966). Although

the manufacturers advocate that 4 litres of oxygen/minute be used with the 28% Ventimask, in practice both Green (1967) and Campbell and Gebbie (1966) show that this concentration of inspired oxygen is obtainable with a flow rate of 2 litres/minute. The 35% Ventimask will produce the stated inspired concentration (± 1%) at a flow rate of 6 litres/minute. These masks, therefore, could be used with the modified fixed flow regulator to provide either 2 or 6 litres of oxygen/minute (para. 182).

191. The Edinburgh Oxygen Mask (British Oxygen Co.) consists of a loose plastic face-piece, supporting a two inch diameter plastic orifice before the lips, into which an oxygen pipe projects laterally (Flenley, Hutchison and Donald, 1963). The mask gives an inspired oxygen concentration from 25%-29% at 1 litre/minute, 31%-35% at 2 litres/minute, and 33%-39% at 3 litres/minute of oxygen. The Edinburgh mask, in our opinion, provides sufficiently accurate control of the inspired oxygen concentration for the treatment of patients with CO_2 retention. One mask provides all the aforementioned concentrations. Carbon dioxide does not accumulate and the inspired oxygen concentration is not greatly influenced by the position of the mask on the face. The mask has been found to be comfortable in use (Green, 1967), and is cheap enough to be completely disposable.

Nasal Catheters: Anterior Nares

192. Administration of oxygen directly to the nose was the first method used to provide controlled oxygen therapy and the method is particularly useful in treating bronchitic patients with CO_2 retention. Various devices are available; Argyle, (distributed by Eschmann Bros. and Walsh Ltd.); Aldington (Aldington Laboratories Ltd.); Addis (Portland Plastics Ltd.) (Addis, 1963); Bardic nasal cannulae (Bard-Davol Ltd.); the Tudor Edwards spectacle frame (British Oxygen Co.); and a simple intranasal oxygen inhaler (British Oxygen Co.). Although these devices differ in detail, they all deliver oxygen through twin tubes into the anterior nares. They produce changes in the arterial oxygen tension equivalent to an inspired oxygen concentration of 25% to 30% at 1 litre of oxygen per minute, 30% to 35% at 2 litres/minute, and 32% to 38% at 3 litres/minute (Catterall *et al*, 1967, and Green, 1967). These measurements are difficult to make, for the inspired gas stream cannot be sampled adequately when nasal catheters are used. Observations must be made of the arterial PO_2 when using the device, and then compared with the arterial PO_2 values obtained when breathing a known concentration of oxygen (Flenley, Hutchison and Donald, 1963). Nasal catheters are less satisfactory as a means of delivering high concentrations of oxygen for even with high flow rates of oxygen the inspired concentration is not as high as that achieved at similar rates with a mask. They remain particularly suited for controlled oxygen therapy in cases of respiratory failure and they are generally very comfortable in use (Green, 1967), provided that the oxygen flow rate does not exceed 2 to 3 litres/minute. The studies of Bethune and Collis (1967) suggest that the actual inspired oxygen concentration produced by such catheters into the anterior nares does depend to a considerable degree upon whether the patient breathes through the nose or mouth. However, the literature is not clear on this point, and we feel that further studies on this topic are required.

Nasopharyngeal Catheters

193. Intranasal catheters, projecting through the nose into the nasopharynx, have also been used for administering oxygen. An oxygen flow of 2 litres/minute again provides an inspired concentration of about 30% but the oxygen must be humidified, as the moist surfaces of the nose are bypassed in this technique (paras. 24-31). Accidental rupture of the stomach has been recorded following this form of oxygen therapy (Walstad and Couklin, 1961).

Oxygen Tents

194. Oxygen administration by tents or incubators is extensively used in paediatric practice. In adult practice, oxygen tents are usually indicated only when the patient cannot, or will not, tolerate a mask or nasal catheter. In addition to the regulation of oxygen concentration of the inspired gas, oxygen tents must provide for the control of temperature and humidity, and the disposal of carbon dioxide. Due to the larger volume of the tent, the fire risk is greater than with masks or nasal catheters.

195. The "Universal Oxygen Tent" (Oxygenaire) is a non-electrical, non-mechanical, ice-cooled oxygen tent suitable for adults and children. Under ideal conditions, following flushing of the tent by oxygen as described by the makers (20 litres/minute for 20 minutes followed by 20 minutes at the given flow rate), the tent produced oxygen concentrations in samples taken from within one inch of a child's lips, of 40% to 52% at flow rates of oxygen from 2 to 10 litres/minute. In random samples taken during actual use, where the tent was being opened for feeding and nursing purposes, similar samples ranged from 28% to 52% at flow rates from 2 to 20 litres of oxygen per minute. For example, at 10 litres of oxygen per minute 10 random samples varied in oxygen concentration from 29% to 36% (Simpson and Russell, 1967).

196. The "Humidaire" Tent (Oxygenaire) is a non-electrical, non-mechanical, ice-cooled, high humidity tent to fit any cot in paediatric practice. Tented under the ideal conditions described above, this tent gave oxygen concentrations of 39% to 43% at 2 litres of oxygen per minute, rising to 61% to 67% at 10 litres/minute, and 70% to 81% at 20 litres/minute. In random samples during use the concentrations of 22% to 30% were obtained at 2 litres of oxygen per minute rising to 50% to 56% at 10 litres/minute (Simpson and Russell, 1967). This largely confirms the manufacturers claims. The tent will also provide a relative humidity up to 100% if this is required.

197. The "Mark V Refrigeration Oxygen Tent" (Oxygenaire) consists of a tent with a refrigerator within the canopy. Oxygen and air are circulated by a fan, and the high ventilation rate can be used to "wash out" carbon dioxide if this is desired. The tent is suitable for adults or children, and it has been studied in detail by Freedman (1964). After preliminary flushing at 10 to 12 litres/minute for 20 minutes, samples were withdrawn during occupation of the tent by bronchitic patients undergoing the usual nursing procedures. With the carbon dioxide "wash out" control off, a flow rate of 8 litres of oxygen per minute produced an oxygen concentration between 50% and 57%, and a carbon dioxide concentration from 0·8% to 1·1%. With the carbon dioxide "wash out" on the oxygen concentration varied between 26% and 31% as the oxygen flow rate rose from 4 to 10 litres/minute, and the carbon dioxide concentration was always less than 0·4%. Relative humidity varied between

40% and 45% (Freedman, 1964). It would appear that the Oxygenaire, "Mark V" tent is suitable for controlled oxygen therapy in patients with carbon dioxide retention, if used with the carbon dioxide "wash out" on. Under ideal conditions Simpson and Russell (1967) found an oxygen concentration with the carbon dioxide "wash out" off rising from 40% to 41% at 2 litres of oxygen per minute to 50% to 52% at 10 litres/minute.

198. The Venturi Head Tent (Oxygenaire) consists of a small canopy suspended over the head and shoulders with the lower edge free. Oxygen is supplied through a Venturi jet in the dome from which the canopy is suspended. The amount of air entrained by this Venturi jet, and therefore the oxygen concentration delivered to the patient, is varied by a throttle on the air intake (Campbell and Gebbie, 1966). As the patient is breathing mainly room air, there is no need for humidification or cooling. The tent gave inspired oxygen concentrations of 23·5% to 24·5% at a setting of 24·5%, and 26·5% to 27·5% at a setting of 28%. Carbon dioxide levels were below 0·5% (Simpson and Russell 1967). Although some patients may find the tent rather warm, this can be overcome by positioning and removal of obstruction to the outlet or by exposing the forearms. This tent is intended, by the inventors, to be used only for the controlled oxygen therapy of respiratory failure (Campbell and Gebbie, 1967). The accurate control of the inspired oxygen concentration and the minimal carbon dioxide retention makes the tent eminently suitable for this purpose in patients who cannot tolerate a "low concentration" oxygen mask, or nasal catheters.

199. The Croupette Type D (Air-Shields (U.K.) Ltd.) is an oxygen tent for paediatric use. Under ideal circumstances (flushing at 10 litres of oxygen per minute for 30 minutes, with samples taken after a further 20 minutes at the given flow rate, in an undisturbed tent) oxygen concentrations ranged from 40% to 52% at a flow rate of 2 to 10 litres/minute. When the "Universal" Croupette (Air-Shields (U.K.) Ltd.) was tested in a similar fashion concentrations of 28% to 31% were obtained at flow rates of 10 and 20 litres of oxygen per minute. The Type D Croupette yielded concentrations of 27% to 49% at oxygen flow rates from 2 to 20 litres/minute, when random samples were taken (Simpson and Russell, 1967). The Universal Croupette, with the icebox uncovered, gave oxygen concentrations of 22% to 37% at flow rates of 2 to 20 litres of oxygen per minute (Simpson and Russell, 1967).

Incubators

200. The "New Oxygenaire" Incubator (Oxygenaire) is designed to conform to British Standard 3061:1965. This incubator produced inspired oxygen concentrations from 27% to 35% as the oxygen flow increased from 1 to 6 litres/minute, with the air intake open. With this intake occluded the concentrations rose to 63% after 30 minutes at 6 litres/minute. The Portable Incubator Mark III (Oxygenaire) is intended to provide isolation and warmth for a small infant during transport from home to hospital. With flow rates of 2 litres/minute oxygen concentrations of 25% are produced, rising to 31% at 4 litres/minute (Simpson and Russell, 1967). Higher flow rates would not be used in practice with this incubator.

201. The Intensive care Isolette Incubator (Air-Shields (U.K.) Ltd.) gives concentrations of oxygen of 26% to 44% as the oxygen flow rate rises from

1 to 4·5 litres/minute. When the red handle of the apparatus is raised, the oxygen concentration at the baby's lips was found to vary between 58% and 71% (mean 63%), with a flow of 4·5 litres of oxygen per minute. (Simpson and Russell, 1967).

Humidification

202. Under the optimal conditions of 50% relative humidity and 18 °C, air has a water concentration of about 7 mg. water/litre. After passing through the nose, the water concentration increases to about 35 mg./litre in the larynx, and 45 mg./litre in the alveoli. During mouth breathing, however, the concentration rises only to 26 mg./litre in the larynx (Cole, 1953; Sara, 1965). The figures apply to a normal adult at rest. Oxygen from cylinders or piped supplies is dry, and humidification is usually considered desirable before the gas is administered to the patient. The exact need for this can be calculated, if the aim is to produce a normal concentration of water in the upper tracheal gas. If a patient who is breathing a total of 10 litres/minute of air is given 2 litres of oxygen/minute and 8 litres of air, and he breathes through his nose, the nose must add about 250 mg. of water/minute to the inspired gas, as opposed to the 230 mg. of water/minute when air alone is breathed. These figures apply to ambient air with a water concentration of 7 mg./litre. This extra 20 mg. of water/minute is well within the capacity of the nasal mucosa for humidification, and it follows that in these circumstances oxygen does not need to be humidified.

203. On the other hand, similar calculations show that if 10 litres/minute are breathed through the mouth, of which 6 litres are provided as dry oxygen, the gas in the upper trachea will be deficient in 270 mg. of water, each minute. Although these calculations do not allow for the rebreathing of expired gas which is saturated with water vapour, it is obvious that humidification of the oxygen is required. Prolonged inhalation of dry gas can disturb ciliary action, and indeed metaplasia of ciliated to squamous epithelium has been observed (Sara, 1965). If the humidifying action of the upper airway is completely bypassed, by tracheostomy, or by an endotracheal tube, then air at 34°C, fully saturated with water vapour (a concentration of 35 mg./litre) is required.

204. In acute laryngo-tracheo bronchitis in infants, in mucoviscidosis, in asthmatic attacks, and in chronic bronchitis (Palmer, 1960), an increase in the humidity of the inspired gas is used to reduce sputum viscosity, and to promote expectoration. Oxygen is often indicated in these conditions and therapy may include bronchodilators or mucolytic agents given as aerosols in the inspired gas. Nebulisers are used to produce aerosols, which consist of a suspension of droplets, ranging from 100 microns to under 1 micron in diameter. The larger droplets tend to coalesce and fall out of suspension, but particles under 0·5 microns in diameter may not be deposited at all in the respiratory tract. The water content of a gas, therefore, depends both upon the water vapour content (which is a function of the temperature and pressure, Fig. II) and the amount of water suspended as droplets.

205. In an attempt to increase the water concentration of inspired gas, some use has been made of high humidity oxygen tents, particularly in paediatric practice. It should be remembered, however, that high temperatures and high humidity reduce heat loss from the body by evaporation. High temperatures

also produce skin vasodilation, tachycardia, a rise in cardiac output and an increase in pulmonary ventilation. These all put an additional strain on the cardiovascular system, which may be serious in the ill patient. It is recommended that an ambient temperature of 25°C, with a water concentration of less than 14 mg./litre (60% relative humidity), should not be exceeded in treating patients with heart disease.

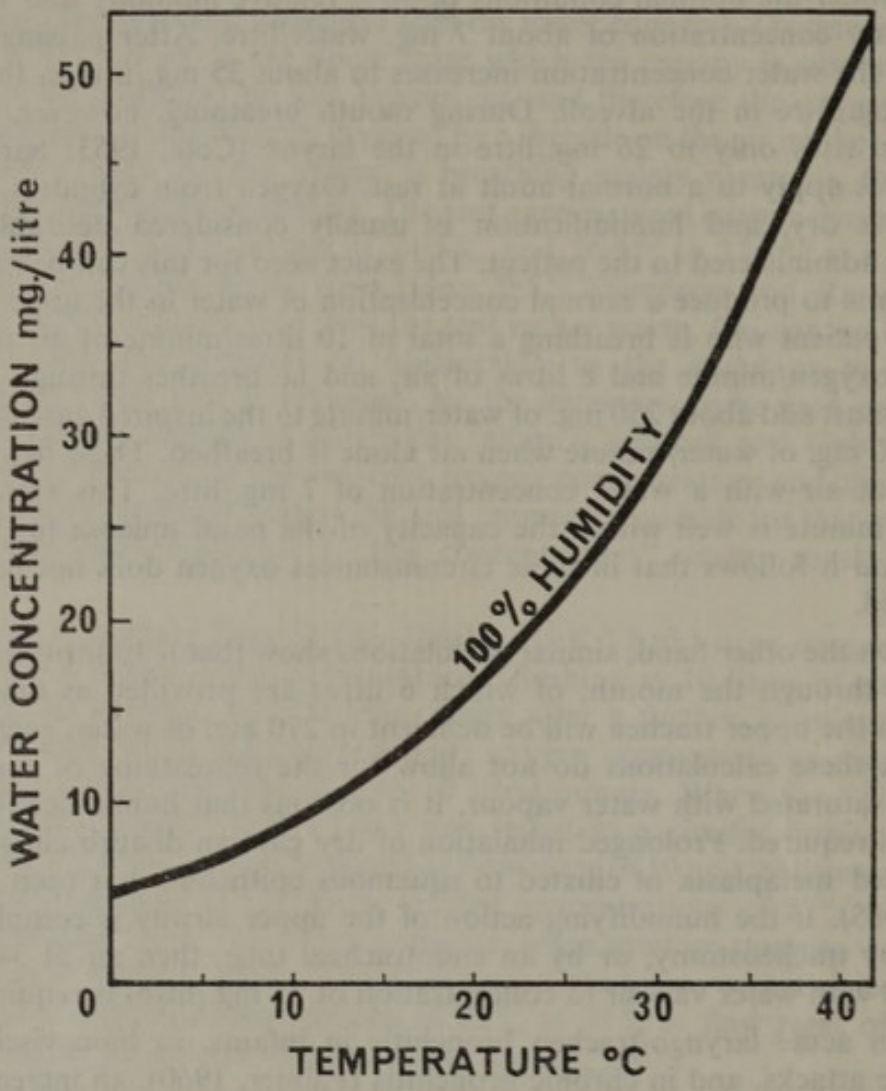

FIGURE II

Illustrating the relationship between the concentration of water in fully humidified air and its temperature.

206. Water can be added to the inspired gas by:

(a) bubbling the gas through water at room temperature;

(b) passing the gas over heated water;

(c) nebulising water at room temperature;

(d) nebulising heated water;

(e) condenser-humidifiers.

The efficiency of a simple bubbling humidifier, the Woulfe bottle, has been studied by Freedman (1967). As water evaporates into the gas stream, the water cools, and the amount of water added is decreased, (Fig. II). At a flow

of 3 litres of oxygen/minute, at a room temperature of about 18°C, a water concentration of approximately 13 mg./litre is achieved. As this is higher than the 7 mg./litre found in air under optimal conditions, it provides adequate humidity at this flow rate. If the patient is a persistent mouth-breather, and 6 litres of oxygen/minute are given, the water concentration rises to 12 mg./litre which is probably not sufficient unless some re-breathing occurs. This apparatus is certainly not suitable for humidifying the inspired gas for delivery to a tracheostomy.

207. In the humidifiers described by Marshall and Spalding (1953), and Spalding (1956), the inspired gas passes over the surface of water which is raised to a thermostatically controlled temperature. The water concentration of the gas delivered depends upon the temperature, and water will condense out if the gas cools before it is delivered to the patient. A water concentration of 35 mg./litre, at 34°C which is suitable for patients with a tracheostomy, can be obtained by these devices. The water in these bulk humidifiers can easily become contaminated with bacteria and careful sterilisation is important to avoid cross infection (Kundsin and Walter, 1962).

208. Nebulisers which operate at room temperature are primarily intended for the administration of drugs as aerosols, but they are also useful as humidifiers. The Wright nebuliser (Wright, 1958), requires a pressure of between 10 and 20 lbs./sq. in., and passes 10 litres of oxygen/minute at 20 lbs./sq. in. It is not effective as a nebuliser at 2 litres of oxygen/minute, as the pressure is then too low. Cushing and Miller (1958) have shown that most nebulisers operating at room temperature produce a water concentration of between 12 and 18 mg./litre, but some of the droplets may fall out of suspension before delivery to the patient. Heated nebulisers can be very efficient humidifiers (Sara and Currie, 1965) and are widely used in patients with tracheostomy during artificial ventilation. Ultrasonic nebulisers, which have been introduced recently, are expensive but extremely efficient. They may provide water far in excess of the need for humidification. This may be helpful for some patients with tenacious secretions, but there is need for caution because there is evidence from experimental work in animals that prolonged exposure may give rise to pulmonary atclectasis (Modell *et al*, 1968). The condenser humidifier is only applicable to rebreathing systems, such as are used with a tracheostomy, but it can be very effective (Mapleson, Morgan and Hillard, 1963). The respirations pass through the device, which has a large surface area, and the moisture of expired gas condenses out, and serves to humidify the gas following inspiration.

209. In summary, humidification of inspired oxygen is not usually required with low concentration devices, but may be indicated when high concentrations are given by a device in which rebreathing is avoided. Dryness of the ambient air, and mouth breathing are both possible indications for humidification and this may be necessary in some respiratory diseases. The most efficient humidifiers are the heated nebulisers, and the greatest need for humidification is in treating patients with a tracheostomy. The simple Woulfe bottle appears to be adequate for routine humidification of oxygen given in high concentration by a mask which allows some rebreathing (Polymask, M.C. Mask).

Apparatus for Portable Oxygen Therapy

210. Portable oxygen equipment which is intended to be carried by the patient is not available for prescription on form E.C.10, but is available through

the National Health Service, on the recommendation of a Consultant. Equipment available consists of a lightweight cylinder, with reducing valve, and mask.

211. The Portogen (British Oxygen Co.) weighs 3 lbs. 1 oz., contains 100 litres of oxygen, and delivers 4 litres/minute to a B.L.B. type mask with rebreathing bag. Cylinders can be recharged in the home from a 120 or 48 cu. ft. cylinder, using a special adaptor. The D.H. series (Walter Kidde Ltd.) is supplied in 3 sizes of 120, 170 and 230 litres capacity which weigh 3·8, 4·0 and 5·0 lbs. respectively, and give a flow of 2 or 4 litres of oxygen/minute. The portable oxygen set manufactured by Siebe Gorman & Co. Ltd. contains 99 litres, weighs 4·25 lbs., and gives an oxygen flow of 10 litres/minute. The 2 flow portable oxygen therapy set made by Draeger Normalair Ltd. weighs 4·9 lbs. for 120 litres, or 6·1 lbs. for 200 litres, with a 2 or 4 litre/minute automatic flow control. A mask with rebreathing bag is supplied with this set, and a recharging assembly and cylinder key are also supplied. Valu-Air Ltd. provided the portable oxygen set described by Campbell (1965). The set weighs 2 lbs. 8 oz., and supplies 80 litres of oxygen at a flow rate of 4 litres/minute, through a reducing valve to a plastic facepiece, with reservoir bag and valve. The apparatus produces an inspired oxygen concentration of about 35%, which can be reduced to 30% if the air entrainment port is uncovered.

212. Dr Cotes, of the Medical Research Council suggests that the most appropriate mask for use with portable equipment is the Siebe Gorman breathing mask with a non-return valve which is available from Walter Kidde Ltd. An alternative device to a mask is a "palm breathing device" (Walter Kidde Ltd.) which can reduce the social embarrassment associated with wearing an oxygen mask in public (Cotes, Matthews and Tasker, 1963). This device delivers oxygen into the cupped hand during inspiration, as the control button is depressed by the patient's thumb. Intermittent oxygen during exercise, given by this palm breathing device, needs to be given at a flow rate of 8 litres/minute, to secure the same effect on the prolongation of exercise time as 4 litres/minute given continuously by the Siebe Gorman mask with reservoir bag (Cotes, Matthews and Tasker, 1963).

213. In Chapter 4, we recommend that general practitioners should be encouraged to carry their own supplies of oxygen and equipment. It was suggested that apparatus capable of giving a flow of 6 litres of oxygen/minute for at least 1 hour was needed. From the above descriptions, the D.H. series (Walter Kidde Ltd.), holding 230 litres in the largest size, and weighing 5·0 lbs. appears to approach this requirement most closely, but the highest flow rate is only 4 litres/minute. The Draeger Normalair set of 200 litres capacity, weighing 6·1 lbs., also approaches the requirements of our proposal. If this recommendation is accepted, manufacturers may wish to produce equipment which meets these requirements more closely.

Oxygen Therapy Equipment in the Ambulance

214. The St. Andrew's Scottish Ambulance Service at present provides the following equipment for resuscitation in its ambulances:

(1) The Stephenson Minuteman Resuscitator.

(2) Bag and Mask Resuscitators.

(3) Foot operated aspirators.

(4) A 48 cu. ft. cylinder of oxygen, fitted with a pressure regulator and flowmeter, of the bobbin type, is being supplied at present in some ambulances.

215. The Stephenson Minuteman Resuscitator (British Oxygen Co.) is a pressure cycled automatic ventilator, which can inflate the lung with 100% oxygen 6 to 7 times/minute, or with 50% oxygen 15 times/minute. The apparatus operates from a 72 gallon (327 litre) oxygen cylinder which provides automatic ventilation for 35 minutes. The complete apparatus, with one full cylinder weighs 26 lbs. In addition to artificial ventilation the apparatus can be set to deliver a continuous flow of 6 to 8 litres/minute of oxygen for about 55 minutes. The machine incorporates an aspirator operated by the pressurised oxygen supply. The Automan Resuscitator (Siebe Gorman and Co. Ltd.) is an automatic time cycled ventilator, operated by a self-contained 72 gallon oxygen cylinder (327 litres). The unit weighs 27 lbs,. and can be set to provide automatic ventilation, manual ventilation, or to give a constant flow of 8·4 litres of oxygen/minute.

216. The Ambu Universal Resuscitator (Ambu International) consists of a non-return valve and a self-expanding bag lined with sorbo rubber. The lungs are inflated by compression of the bag through the valve to an anaesthetic type of mask. Various sizes of mask and extension tubes which allow the bag to be compressed by the foot, are available accessories. The bag has a high filling rate so that the victim can be hyperinflated, and hand ventilation has been carried out for up to 8 hours with this apparatus (Ruben and Ruben, 1957). The resuscitator and a foot operated suction pump can be carried in an "ever ready pack". The Air-Viva resuscitator (British Oxygen Co.) also consists of bag, valve and mask, the bag being constructed of plastic. A carrying case, with the resuscitator, and two sizes of masks weighs 3 lbs. 5 oz. The Resusci Folding Bag (Vickers Ltd.) is a plastic bag and mask resuscitator which folds into a small volume. Although these devices provide ventilation with air, all of them have provision for an oxygen supply to be attached. When 4 litres of oxygen/minute are used the lungs can be inflated by an oxygen concentration of between 50% and 60% (Birt, 1965).

217. After discussion with the Scottish Ambulance Service, we consider that resuscitation equipment in the ambulance should, in future, concentrate on a bag and mask resuscitator, with various sizes of masks, and oropharyngeal airways. This equipment should be light in weight and easily carried to the victim. A foot operated aspirator is also desirable. We recommend that the Service should not purchase more automatic ventilators at present although the existing machines should continue to be used. As we have discussed in Chapter 8, we feel that additional oxygen has a place in respiratory resuscitation. Oxygen can be provided by tubing from an oxygen cylinder, either in the ambulance, or brought to the scene of the accident. In addition, however, we recommend that manufacturers should be encouraged to provide a self-contained oxygen supply with a bag and mask resuscitator. A supply of even 40 litres of oxygen could provide 10 minutes of artificial ventilation by hand, with 50% oxygen, from a bag and mask resuscitator. In this period, evacuation of the casualty, or provision of further supplies of oxygen, should be possible. If not, ventilation should continue with air.

218. At the risk of repetition, we feel it wise to repeat our warnings of the psychological danger of excessive reliance on any apparatus in resuscitation.

Seconds are vital, and time spent in bringing the simplest apparatus may lose lives. Mouth-to-mouth artificial ventilation needs no apparatus, and must be the first measure to be employed once the airway is cleared.

219. Oxygen is needed in the ambulance during the transport of medical, surgical and obstetrical cases to hospital. In our view, the medical indications for oxygen therapy in the ambulance fall clearly into two groups (Chapter 3). In cases of pulmonary oedema, haemorrhagic shock, cardiogenic shock, pneumonia, pulmonary fibrosis, thrombo-embolic pulmonary disease, pulmonary collapse, and carbon monoxide poisoning, oxygen in high concentration is indicated. This can be given by a Polymask or M.C. Mask at a flow rate of 6 litres of oxygen/minute. In cases of chronic bronchitis and emphysema, and bronchial asthma, oxygen in the ambulance should be given in low concentrations, by means of an Edinburgh mask or Ventimask with 2 litres of oxygen/minute. We recommend that all ambulances which carry medical, obstetrical, or surgical emergencies should carry a 48 cu. ft. cylinder with a fixed flow regulator which can be set by the ambulanceman to provide either 2 or 6 litres of oxygen/minute. When the "Med" setting is used (2 litres of oxygen/minute) and Edinburgh mask or Ventimask is used, and with the "High" setting (6 litres of oxygen/minute) the Polymask or M.C. Mask. These recommendations are incorporated in the proposed amendments to Instruction to Staff of the St. Andrew's Scottish Ambulance Service, on Oxygen Therapy Equipment (Appendix IV).

Testing of Oxygen Therapy Equipment

220. Despite numerous enquiries, we were unable to discover any organisation which was charged with responsibility for testing equipment for oxygen therapy. The results which we have given were obtained by individual workers, and published in the medical press. The British Standards Institution has published 4 standards in this field; B.S. 1319: 1955 (Medical Gas Cylinders and Anaesthetic Apparatus); B.S. 3806: 1964 (Breathing Machines for Medical Use), B.S. 3849: 1965 (Breathing attachments for Anaesthetic Apparatus); and B.S. 3061: 1965 (Electrically Heated Incubators for Babies). This last standard (B.S. 3061: 1965) states in paragraph 21 that "an oxygen flow control shall be required to limit the oxygen concentration under usual working conditions to 35%". No details of tests to meet this requirement are given in this standard, and we understand that the British Standards Institute has not carried out any tests on incubators from the point of view of oxygen concentration.

221. "If the concentration of oxygen which a patient receives is not known with a reasonable degree of accuracy then the situation is analagous to the administration of an unknown quantity of a drug which may do harm if given in excess or provide insufficient benefit if the dose is too small." For this reason we are of the opinion that apparatus for oxygen therapy should be required to conform to certain standards, in particular with regard to the concentration of oxygen delivered to the patient in actual use. We propose that some national agency, such as the Medical Research Council or the British Standards Institute, should be asked to establish standards and routine methods for testing oxygen therapy equipment. Responsibility for ensuring that commercially available apparatus conforms to the required standards would, we suggest, be a suitable task for the adviser in Inhalation Therapy which we have proposed as a member of the staff of each Regional Hospital Board.

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CHAPTER 12

Summary of Recommendations and Conclusions

Oxygen Therapy in Adult Hospital Practice (Chapter 3)

222. The clinical assessment of the degree of hypoxaemia is unreliable. Objective measurement in terms of arterial blood oxygen tension can be of great help in many cases and should be more widely available (paragraphs 33 and 35).

223. Controlled oxygen therapy with an inspired oxygen concentration of the order of 30% is required in the treatment of acute exacerbations or other infective complications of generalised obstructive respiratory disease (chronic bronchitis and emphysema). The administration of oxygen in high concentrations may cause or increase under-ventilation and carbon dioxide retention (paragraphs 36 and 37).

224. In severe respiratory failure arising from neuromuscular disorders or from a crushed chest, oxygen therapy alone is quite inadequate and mechanical ventilation is required (paragraphs 39, 40 and 42).

225. Oxygen is required in high concentrations in conditions such as pneumonia, bronchial asthma and pulmonary oedema where the arterial blood oxygen tension is low and the carbon dioxide tension is normal or low (paragraphs 41 and 42).

226. A controlled trial should be instituted to ascertain the value of administering high concentrations of oxygen in uncomplicated cases of myocardial infarction. We would at present recommend the administration of oxygen in high concentration in such cases except in those with severe chronic bronchitis (paragraph 44).

227. In right ventricular (congestive) failure complicating rheumatic heart disease, arterial blood oxygenation is usually adequate while breathing air. In view of the low cardiac output in this condition, high oxygen therapy is indicated at an early stage if there are any pulmonary complications except severe chronic bronchitis where controlled oxygen therapy is indicated.

228. High concentrations of oxygen are strongly indicated in cardiogenic shock (paragraph 45). We also recommend the routine use of such therapy in haemorrhagic and traumatic shock (paragraphs 49, 50 and 51).

229. The dangers and ominous significance of carbon dioxide retention in severe and prolonged attacks of bronchial asthma is emphasised (paragraph 46). Blood gas monitoring is highly desirable in such cases. Active research is needed on the management of such patients in whom high concentrations of oxygen, although initially necessary, may become dangerous.

230. Oxygen can be given in high concentrations without danger in alveolar capillary block and in cyanotic congenital heart disease and may be needed in treatment of superadded chest infections (paragraphs 47 and 48).

231. 30% oxygen should be given to all patients with a history of bronchitis or with any chest complications for 24-48 hours following general anaesthesia for upper abdominal surgery (paragraphs 52 and 53).

232. Hyperbaric oxygen therapy has a place in the treatment of carbon monoxide poisoning but its other therapeutic uses are still being evaluated (paragraphs 54 and 55).

233. The dangers of prolonged administration of high concentrations of oxygen by mechanical ventilators is discussed. Such therapy should aim to produce a normal level of arterial PO_2, and not one that is highly elevated (paragraph 56).

234. Anaemic patients are more vulnerable to hypoxaemia from any cause and appropriate oxygen therapy should be commenced early if hypoxaemia is suspected (paragraph 58).

235. After careful deliberation, we considered that the great advantages of piped medical gas supplies in hospitals definitely outweighs the not inconsiderable dangers. We also considered the proposal that nitrous oxide should not be piped in hospitals but only oxygen. Again we considered that the logistic and therapeutic advantages greatly outweighed the potential dangers and that the present practice of piping both these gases in hospitals should continue although with greatly increased precautions (paragraphs 60 and 61).

236. The Code of Practice on the installation and testing of such piped supplies, which has been adopted by the Western Regional Hospital Board, Scotland, is commended for further detailed consideration by other hospital authorities in Scotland (paragraph 63).

237. The post of "gas technician", with clearly defined duties (paragraph 64) in each major hospital should be established and each Regional Hospital Board should establish a post of Technical Adviser on Inhalation Therapy, with duties as described in paragraph 65.

Oxygen Therapy in Domiciliary Practice (Chapter 4)

238. The clinical indications for oxygen in emergencies in the home and in transport to hospital are the same as in hospital practice (paragraphs 79 and 80).

239. Oxygen is recommended as an adjunct to mechanical artificial ventilation with a Bag and Mask resuscitator, and we recommend that general practitioners should carry such equipment (paragraph 81).

240. The problems of evaluating long term oxygen therapy are considered (paragraph 82). The need for such therapy could increase about three fold if its value were more clearly established. The standard apparatus for oxygen administration in domiciliary practice should provide approximately 30% oxygen (paragraph 83).

241. After consideration of the value and problems of patient-carried oxygen supplies, we do not recommend that apparatus for this treatment should be available on the Drug Tariff, but that this equipment continue to be provided on the recommendation of a hospital consultant only (paragraphs 84-88).

242. General practitioners should be encouraged and helped to carry their own supplies of oxygen for emergency use (paragraphs 97 and 98). The cylinders should be lightweight, yet capable of delivering up to 6 litres/minute for one hour (paragraph 98), these cylinders being replenished by chemists on an exchange basis.

243. A working group should be established to advise on the technical problems involved in the provision of lightweight oxygen therapy equipment

for use by general practitioners in emergency, the associated problems of the supply, maintenance and sterilisation of such equipment, and on the problem of the provision of oxygen by means of bag and mask resuscitators (paragraph 100).

Oxygen Therapy in Obstetrical Practice (Chapter 5)

244. Analgesic and anaesthetic techniques that fail to ensure a normal inspired oxygen concentration for the mother in labour are condemned (paragraph 105). Entonox 50/50 mixture of nitrous oxide and oxygen should replace gas and air machines in obstetrical practice (paragraph 105).

245. There is no convincing evidence of maternal hypoxaemia in late pregnancy in normal women, and the routine use of oxygen therapy in labour is not recommended (paragraph 104).

246. Oxygen in high concentration should be given to any pregnant women, in or out of labour, whose arterial PO_2 is either known or suspected to be below normal (paragraph 102).

247. Oxygen in high concentration may be needed as an ancillary measure in the pulmonary aspiration syndrome, in amniotic fluid embolism and obstetrical shock (paragraph 106). We are unable, on review of evidence available to us, definitely to recommend the use of oxygen in treatment of foetal distress and research into this problem is urgently required (paragraph 108).

Oxygen Therapy in Paediatric Practice (Chapter 6)

248. There is considerable evidence that in acute infections of the lower respiratory tract during infancy and childhood there is no danger of producing carbon dioxide narcosis during uncontrolled (high) oxygen therapy. Further research is recommended to clarify the use and effects of oxygen therapy in this age group (paragraph 109).

249. The indications for oxygen therapy in (a) the neonatal period and (b) infancy and childhood are given (paragraphs 110-118).

250. District midwives and general practitioners conducting home confinements should carry simple apparatus for dealing with cases of asphyxia neonatorum (paragraph 111). In emergency, outside hospital, mouth to mouth breathing, properly carried out, is recommended as a possible means of saving life in this condition (paragraph 112).

251. The hyperbaric technique for the resuscitation of the severely asphyxiated newborn should be continued in the Glasgow Medical School. Other centres should be encouraged to appraise this technique against that of incubation and assisted ventilation (paragraphs 113 and 114).

252. In neonatal respiratory distress, oxygen is best given in an incubator. High oxygen concentrations must only be used under expert paediatric supervision and the levels should be carefully controlled by periodic measurements with an oxygen meter (paragraph 115).

253. The ill newborn infant should always be transported in a heated portable battery/mains-operated incubator (paragraph 116).

254. Measurements of oxygen concentration in oxygen tents used for older children should be made more frequently than they are at present in order to ensure that adequate oxygen concentrations of at least 40% are present (paragraph 118).

255. The concentration of oxygen in incubators should be permitted to rise above 35% only in cases of severe respiratory distress when cyanosis is still not abolished by 35% oxygen and this should be carried out if possible, under biochemical control. Oxygen therapy should be discontinued or lower levels sought as soon as possible (paragraph 119).

256. It is extremely important to avoid the use of open fires or naked lights where oxygen tents or incubators are in use (paragraph 120).

Oxygen and Travel by Air (Chapter 7)

257. 30%-35% oxygen should be given during flight in pressurised aircraft to any patient who is known to be hypoxaemic but who is comfortable at sea level (paragraph 124). Patients with acute or chronic respiratory failure should not travel by air.

258. Oxygen in high concentration should be recommended during flight for any patient in heart failure or with severe cyanotic congenital heart disease (paragraph 126). High level oxygen therapy should also be immediately available for any passenger who is subject to angina pectoris or known to be at risk of myocardial infarction. Any passenger in whom the latter condition is suspected should be given oxygen immediately.

259. Anaemic patients should receive 30% oxygen if they must travel with a haemoglobin concentration below 7 Grams/100 ml. (paragraph 129) and oxygen should be given to all patients with sickle cell anaemia when they travel in aircraft with cabin altitudes higher than 4,000 ft. (paragraph 129).

Oxygen in Resuscitation (Chapter 8)

260. Expired-air artificial ventilation ("the kiss of life") should be adopted as the primary first aid measure in restoring life to the apparently dead (paragraphs 132-134). To demand oxygen for resuscitation is dangerous if it prevents people from applying artificial ventilation with expired air immediately (paragraph 134).

261. The professional rescuer (i.e., doctors, nurses, ambulancemen and first aid workers) should carry an airway and a simple Bag and Mask resuscitator (paragraphs 135, 136 and 143).

262. Manufacturers should be encouraged to develop methods of providing high concentrations of oxygen from such Bag and Mask apparatus and this should be preferably from a self-contained source of oxygen (paragraph 136).

263. Automatic cycling ventilators should not replace Bag and Mask resuscitators in first aid work (paragraph 137).

264. Fire Service personnel should be equipped with Bag and Mask resuscitators because many victims of fire die from carbon monoxide poising (paragraph 140).

265. Oxygen enrichment of the gas used in artificial ventilation is highly desirable in the treatment of cardiac arrest (paragraph 141).

Oxygen Therapy in Industry (Chapter 9)

266. Artificial ventilation at first with expired air and later, with high concentrations of oxygen from a Bag and Mask resuscitator with added oxygen, should be applied to the victim of carbon monoxide poisoning who is not

breathing enough by himself. We recommend 100% oxygen in such treatment except where carbon monoxide is the only gassing hazard, as in coke oven plants etc. (paragraph 154) where 95% oxygen and 5% carbon dioxide (carbogen) is recommended.

267. The treatment of poisoning by other noxious gases is considered, (paragraphs 156-161). In cases where asphyxia has resulted, treatment by expired-air artificial ventilation and then by a Bag and Mask resuscitator with added oxygen is recommended.

268. Pulmonary oedema due to lung irritants requires oxygen therapy (paragraph 161) and this may also be necessary in treatment of poisoning with some miscellaneous chemical agents (paragraphs 162-164).

269. Oxygen therapy is not an important component in the treatment of cyanide poisoning and it should never delay the more effective inhalational and intravenous therapy (paragraph 162).

Oxygen Therapy in the Treatment of Drowning (Chapter 10)

270. Strong emphasis is placed on the need for immediate expired air artificial ventilation in near drowning with respiratory failure (paragraph 169). Only when expired air ventilation is satisfactorily initiated should time or effort be spent in obtaining oxygen or other first aid equipment (paragraph 173).

271. Closed chest cardiac massage should only be used in addition to artificial ventilation when two or more arterial pulses are absent, or severe central cyanosis is not corrected by six inflations of the lung with expired air (paragraph 169) and then only by professional rescuers who are trained in its use.

Apparatus for Oxygen Administration (Chapter 11)

272. It would be useful for manufacturers to indicate the capacity of oxygen cylinders in metric as well as imperial units (paragraph 177).

273. Large cylinders of oxygen (240 cu. ft.) should be used on the grounds of economy by hospitals without liquid gas supplies (paragraph 178). Industrial oxygen can be used for medical purposes in emergency (paragraph 179).

274. For domiciliary use, an automatic pressure reducing valve with a fixed flow regulator, modified to give either 2 or 6 litres of oxygen/minute, is recommended (paragraphs 181 and 182).

275. A device to allow fixed presetting at either 2 or 6 litres/minute, so that the patient cannot change the dosage of oxygen himself, is recommended (paragraph 182).

276. Lightweight non-kinking plastic tubing should be used for conveying oxygen to a face mask (paragraph 183).

277. The Polymask or the M.C. mask is most suitable for the delivery of oxygen at high concentration (paragraphs 186 and 188).

278. The Edinburgh oxygen mask (paragraph 191) and the Ventimask (paragraph 189) are recommended for the delivery of oxygen at moderately raised concentration (controlled oxygen therapy).

279. Nasal catheters appear to be a satisfactory method of providing controlled oxygen therapy; the type of catheter which projects into the anterior nares only is to be preferred (paragraph 192).

280. Of available oxygen tents, the Humidaire Oxygen Tent (paragraph 195) and the Mark V Refrigerated Oxygen Tent (paragraph 197) are suitable for providing a wide range of oxygen concentrations.

281. We are alarmed at the wide variation in oxygen concentrations provided by some oxygen tents. The infant incubators which we reviewed appeared satisfactory in this respect (paragraphs 200 and 201). The Venturi Head Tent will provide accurate control of the inspired oxygen concentration in the range 24%-35% (paragraph 198).

282. Humidification is not usually required with devices delivering moderate concentrations of oxygen but is needed when high concentrations are given by devices which do not allow re-breathing. Humidification is required in patients with mouth breathing and when the ambient air is very dry (paragraphs 202-208). The simple Woulfe bottle appears to be adequate for humidification with the Polymask and M.C. mask (paragraph 206).

283. There is no commercially available lightweight apparatus that can satisfactorily deliver up to 6 litres of oxygen/minute for one hour; as indicated in Chapter 4, such apparatus is most desirable for general practitioner use (paragraph 213).

284. Ambulances should carry a Bag and Mask resuscitator, with various sizes of masks and oropharyngeal airways, for use in artificial ventilation. Ambulance staff should be trained in their use (paragraph 217).

285. Manufacturers should be encouraged to provide at least 40 litres of oxygen in a self-contained lightweight unit for use with a Bag and Mask resuscitator.

286. The Scottish Ambulance Service should not purchase more automatic cycling ventilators (paragraph 217). Where indicated, oxygen should be given in ambulances from a 48 cu. ft. cylinder to patients during transport to hospital. A fixed flow regulator should be used. Six litres of oxygen/minute with a Polymask or M.C. mask should be given to patients with pulmonary oedema or cardiogenic shock (paragraph 219), *or* 2 litres of oxygen/minute with an Edinburgh mask or Ventimask to patients with chronic bronchitis and emphysema, or bronchial asthma (paragraph 219).

287. Some national agency should establish standards of performance for apparatus used in oxygen therapy. The adviser in Inhalation Therapy to each Regional Hospital Board (Chapter 3) should be responsible for ensuring that equipment meets these standards (paragraph 221).

Appendix I

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Appendix II

The Causes, Mechanisms and Treatment of Impairment of Oxygen Delivery to Body Cells

Condition	*Mechanism*	*Arterial Oxygen Partial Pressure*	*Arterial CO_2 Partial Pressure*	*Type of Oxygen Therapy Indicated*
Faulty anaesthetic and inhalation apparatus, low oxygen, oxygen in confined spaces, and high altitude.	Low inspired oxygen partial pressure	Low	Low	Concentration to restore an inspired PO_2 of 150 mm Hg. is adequate but no harm if it is higher.
Lung disease (Pneumonia, Pulmonary Fibrosis, Thrombo-embolic Pulmonary disease, Pneumothorax, Pulmonary Collapse, etc.)	Poor mixing of gas and blood in the lung	Low	Low or normal	High concentrations of oxygen are not dangerous
Chronic bronchitis and emphysema in an acute exacerbation	Hypoventilation and poor mixing of gas and blood in the lung	Low	High	Controlled oxygen therapy (see text)
Respiratory depressive drugs, Crushed chest, Neuromuscular disease (see text)	Hypoventilation	Low	High	Mechanical ventilation. Never use high concentrations of oxygen alone
Pulmonary oedema	Poor mixing of gas and blood in the lung	Low	Low	High concentrations of oxygen indicated
Anatomical shunt from the right to the left heart, "Cyanotic congenital heart disease", etc.	Some blood completely by-passing areas of gas exchange in the lung	Low	Low	Even 100% oxygen may not correct the arterial PO_2
Cardiogenic shock	Poor mixing of blood and gas and decrease in total and regional blood flow	Low	Variable	100% oxygen does not always completely correct but is valuable
Peripheral vascular disease	Locally decreased blood flow	Normal	Normal	Even hyperbaric oxygen does not necessarily restore normal oxygenation to the ischaemic area

Condition	*Mechanism*	*Arterial Oxygen Partial Pressure*	*Arterial CO_2 Partial Pressure*	*Type of Oxygen Therapy Indicated*
Congestive heart failure	Inadequate cardiac output, sometimes with secondary changes in mixing of gas and blood in lungs	Normal or slightly decreased	Normal or slightly decreased	If arterial PO_2 is low oxygen is indicated in high concentration
Anaemia	Insufficient oxygen carrying capacity of blood	Normal or slightly lowered	Normal or slightly decreased	Conventional oxygen therapy is of little value, unless pulmonary disease complicates the picture
Cyanide poisoning, Dinitrophenol poisoning, and Aspirin poisoning	Poisoning of various cellular enzymes involved in utilisation of oxygen	Probably normal	Probably normal or low	Conventional oxygen therapy is of no value

In previous terminology, the following were used:

(a) Anoxic anoxia, meaning any condition with a low arterial PO_2.
(b) Stagnant anoxia, meaning any condition with a poor blood flow.
(c) Anaemic anoxia, meaning a condition where the arterial PO_2 was normal, yet the oxygen content of the arterial blood was decreased.
(d) Histotoxic anoxia, meaning any condition where the cellular utilisation of oxygen was impaired.
(e) In general, we feel that the term "anoxia" is incorrect, as it is usually applied to conditions where oxygen is deficient, but not absent, and the term hypoxia is more appropriate.

Appendix III

Oxygen Therapy in Domiciliary Practice

Results of enquiry amongst General Practitioners in South-East Scotland.

1. Population (on doctors' lists) at risk

Urban	—	584,113
Rural	—	70,198
Mixed	—	141,497
Unknown	—	27,118
Total	—	822,926

2. Incidence of use (See also x^2 calculation at item 12 below).

		Urban	*Rural*
Number of cases in which oxygen therapy used	—	202	21
Population at risk	—	584,113	70,198
Incidence of use per 1,000 population at risk	—	.346	.299

3. Age of patients receiving oxygen therapy — Histogram 1

4. Number of cases in which oxygen therapy used (by disease) — Table 1

5. Type of apparatus used (by disease) — Table 2

6. Approximate periods patients received oxygen (by Patients' age groups) (Individual tables for each specified disease) — Table 3

7. Problems encountered in use or supply of oxygen equipment

A total of 257 doctors gave a positive reply to this question, and, of these, 73 (or 28%) had actually experienced difficulty under one or more of the categories listed. Of those recording difficulties, 36% encountered problems in the supply of oxygen during the day; 38% at night; 18% in the servicing of oxygen equipment; 29% in patients not understanding its use; and 12% in dangers from the use of oxygen.

A comparison of extracted figures for urban and rural practices only gave the following results:

		Urban	*Rural*
1. Number of doctors replying	—	171	33
2. Number of doctors recording difficulties	—	51 (29·8%)	11 (33·3%)
3. Percentage of (2) encountering problems in:			
(a) supply of oxygen during day	—	31·4%	63·6%
(b) supply of oxygen at night	—	35·3%	36·4%
(c) servicing of oxygen equipment	—	23·5%	9·1%
(d) patients not understanding use	—	37·3%	9·1%
(e) dangers from use of oxygen	—	13·7%	—

8. Use of oxygen in emergencies during six months prior to survey

		Urban	*Rural*
1. Number of doctors who returned questionnaire	—	255	39
2. Number of doctors who used oxygen in emergency	—	89 (34·9%)	16 (41·0%)
3. Percentage of (2) who:			
(a) carried oxygen with them	—	21·4%	50·0%
(b) obtained supply from ambulance service	—	67·4%	12·5%
(c) obtained supply from hospital	—	2·3%	12·5%
(d) obtained supply from chemist	—	7·9%	18·8%
(e) obtained supply from other source	—	1·1%	6·3%

9. Preferred source of emergency supply

		Urban	*Rural*
1. Number of doctors who stated a preference	—	242	37
2. Percentage of (1) who:			
(a) preferred emergency supply from ambulance service	—	50·4%	21·6%
(b) preferred emergency supply from chemist	—	19·0%	27·0%
(c) preferred emergency supply from hospital	—	10·3%	21·6%
(d) preferred to have their own equipment	—	40·5%	51·4%

10. Use of oxygen during transport to hospital in six months prior to survey — Table 4

11. Use of oxygen therapy for specific conditions in the two years prior to survey

1. Number of domiciliary deliveries of children in S.E.R.H.B. area during this period	—	8,159
2. Number of doctors who used oxygen therapy for:		
(a) the mother in domiciliary obstetric practice	—	14
(b) the domiciliary treatment of the new-born	—	42
3. Total population at risk	—	822,926
4. Number of doctors who used oxygen therapy in:		
(a) the long term management of chronic respiratory disease and cor pulmonale	—	211
(b) bronchial asthma	—	122
(c) the long term management of heart failure other than cor pulmonale	—	82

12. Incidence of Use

	Type of practice		
	Urban	*Rural*	*Total*
(i) Number of patients who did not receive oxygen	583,911	70,177	654,088
(ii) Number of patients who received oxygen	202	21	223
(iii) Total number of patients at risk	584,113	70,198	654,311
(iv) Percentage in each type of practice that had oxygen	.035	.030	.034
(i) Number of patients who did not receive oxygen	583,911	70,177	654,088
(ii) Expected number on hypothesis that incidence of use of oxygen and type of practice are unrelated	583,914	70,174	654,088
(iii) Difference, observed minus expected	−3	+3	—
(iv) (Difference)^2	9	9	—
(v) (Difference)^2 ÷ Expected number	.000015	.000128	—
(i) Number of patients who received oxygen	202	21	223
(ii) Expected number on hypothesis that incidence of use of oxygen and type of practice are unrelated	199	24	223
(iii) Difference, observed minus expected	+3	−3	—
(iv) (Difference)^2	9	9	—
(v) (Difference)^2 ÷ Expected number	.045	.375	—
Total —			
Observed	584,113	70,198	654,311
Expected	584,113	70,198	654,311

x^2 value = .50
P = 50.

HISTOGRAM 1

Number of Patients who received Oxygen Therapy by Age Groups

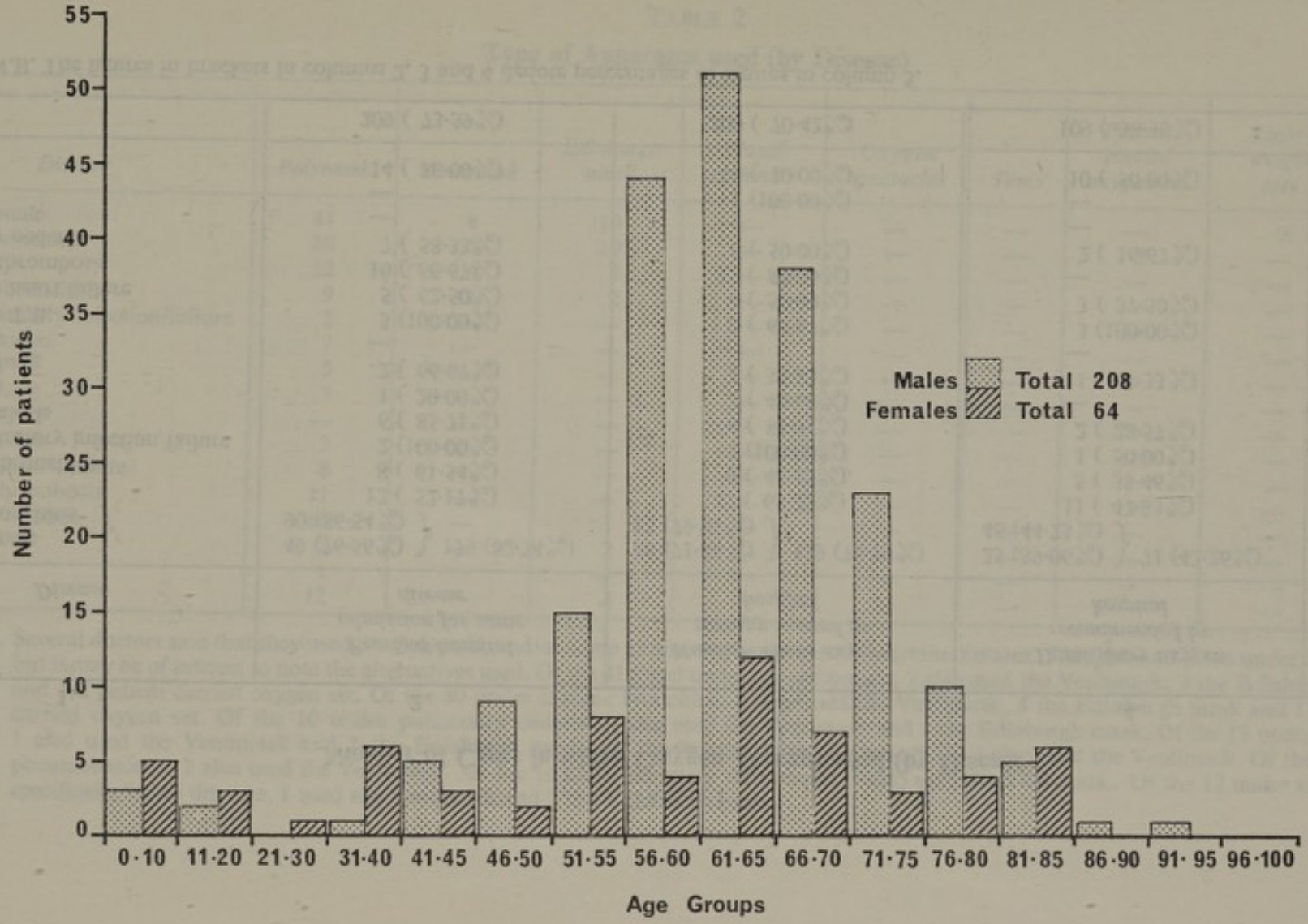

TABLE 1

Number of Cases in which Oxygen Therapy used (by Disease)

1.	2.	3.	4.	5.
Disease	*Previous hospital admission for same disease*	*Number whose oxygen therapy started in hospital*	*Domiciliary oxygen recommended by hospital*	*Total number of cases in which oxygen used*
Cor pulmonale	49 (76·56%) } 139 (82·74%)	46 (71·88%) } 129 (76·79%)	25 (39·06%) } 71 (42·26%)	64 } 168
Chronic bronchitis	90 (86·54%) }	83 (79·81%) }	46 (44·23%) }	104 }
Asthma	12 (52·17%)	15 (65·22%)	11 (47·83%)	23
Asthmatic bronchitis	8 (61·54%)	6 (46·15%)	5 (38·46%)	13
Acute respiratory infection/failure	2 (100·00%)	2 (100·00%)	1 (50·00%)	2
Pneumoconiosis	6 (85·71%)	6 (85·71%)	2 (28·57%)	7
Pneumonia	1 (20·00%)	2 (40·00%)	—	5
Cancer of lung	2 (66·67%)	1 (33·33%)	1 (33·33%)	3
Lobectomy	—	—	—	—
Pulmonary T.B.	3 (100·00%)	2 (66·67%)	3 (100·00%)	3
Congestive heart failure	5 (62·50%)	4 (50·00%)	3 (37·50%)	8
Coronary thrombosis	10 (66·67%)	12 (80·00%)	—	15
Pulmonary oedema	7 (58·33%)	6 (50·00%)	2 (16·67%)	12
Poisoning	—	—	—	4
Injuries	—	1 (100·00%)	—	1
Other	14 (70·00%)	14 (70·00%)	10 (50·00%)	20
Totals	209 (73·59%)	200 (70·42%)	109 (38·38%)	284

N.B. The figures in brackets in columns 2, 3 and 4 denote percentages of figures in column 5.

TABLE 2

Type of Apparatus used (by Disease)

Disease	*Polymask*	*Ventimask*	*Edinburgh mask*	*Nasal catheters*	*Oxygen spectacles*	*Tents*	*Patient carried oxygen set*	*Light-weight sets*	*Other or unknown type*
Cor pulmonale	41	8	10	—	—	—	—	3	2
Chronic bronchitis	80	5	10	—	—	—	2	—	7
Asthma	12	4	3	—	—	—	—	—	4
Asthmatic bronchitis	9	1	2	—	—	—	—	—	1
Acute respiratory infection/failure	2	—	—	—	—	—	—	—	—
Pneumoconiosis	7	—	—	—	—	—	—	—	—
Pneumonia	5	—	—	—	—	—	—	—	—
Cancer of lung	3	—	—	—	—	—	—	—	—
Lobectomy	—	—	—	—	—	—	—	—	—
Pulmonary T B.	2	1	—	—	—	—	—	—	—
Congestive heart failure	8	—	—	—	—	—	—	—	—
Coronary thrombosis	11	—	—	—	—	—	1	—	3
Pulmonary oedema	10	—	1	—	—	—	—	—	1
Poisoning	2	—	2	—	—	—	—	—	—
Injuries	1	—	—	—	—	—	—	—	—
Other	12	—	4	—	—	—	—	—	4

N.B. Several doctors said that they used the Polymask and/or some alternative equipment for certain diseases. They were all listed under Polymask but it may be of interest to note the alternatives used. Of the 41 listed under cor pulmonale, 7 also used the Ventimask, 3 the Edinburgh mask and 1 a patient carried oxygen set. Of the 80 under chronic bronchitis, 4 also used the Ventimask, 3 the Edinburgh mask and 1 a patient carried oxygen set. Of the 10 under pulmonary oedema, 1 also used the Ventimask and 1 the Edinburgh mask. Of the 12 under asthma, 1 also used the Ventimask and 1 the Edinburgh mask. Of the 9 under asthmatic bronchitis, 1 also used the Ventimask. Of the 7 under pneumoconiosis, 2 also used the Ventimask. Of the 11 under coronary thrombosis, 1 also used the Ventimask. Of the 12 under other (not specifically listed) diseases, 1 used the Ventimask and 2 the Edinburgh mask.

TABLE 3

Approximate Periods Patients received Oxygen (by Patients' Age Groups)

Disease	*Age groups*	*— 24 hours*	*— 1 week*	*— 4 weeks*	*— 3 months*	*— 1 year*	*Over 1 Year*	*Continuing*	*Period unknown*
All diseases	0–10	6	—	—	—	—	—	1	1
	11–50	6	1	3 (1)	3 (1)	3 (1)	6 (4)	9	1
	51–65	17	9	15 (3)	12 (5)	23 (14)	32 (22)	23	
	66–100	10	9	14 (1)	13 (2)	13 (7)	14 (13)	22	3
Cor pulmonale; chronic bronchitis; acute respiratory infection/failure	0–10	3	—	—	—	—	—	1	—
	11–50	1	1	2 (1)	2 (1)	1	3 (1)	5	1
	51–65	6	6	6 (1)	9 (4)	19 (12)	23 (17)	19	1
	66–100	1	3	8 (1)	11 (1)	4 (2)	13 (11)	14	2
Asthma	0–10	2	—	—	—	—	—	—	—
	11–50	1	—	1	—	—	1 (1)	3	—
	51–65	2	—	2	—	2 (2)	2 (1)	—	—
	66–100	1	1	3	—	—	—	1	—
Asthmatic bronchitis	0–10	—	—	—	—	—	—	—	—
	11–50	—	—	—	—	1	—	—	—
	51–65	2	—	3 (1)	—	2 (1)	1 (1)	—	—
	66–100	—	1	1	—	—	—	2	—

N.B. The figures in brackets denote the numbers of patients who have received oxygen for that particular period of time but continue to receive it.

TABLE 3—(*Continued*)

Disease	*Age groups*	— 24 *hours*	— 1 *week*	— 4 *weeks*	— 3 *months*	— 1 *year*	*Over 1 Year*	*Continuing*	*Period unknown*
Pneumoconiosis	0–10	—	—	—	—	—	—	—	—
	11–50	—	—	—	—	—	—	—	—
	51–65	—	1	—	—	—	1	—	—
	66–100	1	—	—	—	2 (2)	1 (1)	1	—
Pneumonia	0–10	—	—	—	—	—	—	—	—
	11–50	1	—	—	—	—	—	—	—
	51–56	1	1	—	—	—	1	—	—
	66–100	—	1	—	—	—	—	—	—
Cancer of lung	0–10	—	—	—	—	—	—	—	—
	11–50	—	—	—	—	—	—	—	—
	51–65	—	—	1	—	—	—	—	—
	66–100	—	—	—	—	1 (1)	—	—	—

TABLE 4

Use of Oxygen During Transport to Hospital in Six Months Prior to Survey

1.	2.	3.	4.
Disease	*Total number of cases in which oxygen used during transport to hospital*	*Number of cases in which oxygen used during transport to hospital*	
		Urban	*Rural*
Cor pulmonale	23 (10·65%)	17 (10·30%)	2 (11·77%)
Chronic bronchitis	23 (10·65%)	19 (11·51%)	1 (5·88%)
Asthma	19 (8·79%)	17 (10·30%)	1 (5·88%)
Asthmatic bronchitis	8 (3·70%)	6 (3·64%)	—
Acute respiratory infection/failure	10 (4·63%)	8 (4·85%)	—
Pneumoconiosis	—	—	—
Pneumonia	26 (12·04%)	21 (12·73%)	4 (23·52%)
Cancer of lung	—	—	—
Lobectomy	—	—	—
Pulmonary T.B.	—	—	—
Congestive heart failure	9 (4·17%)	5 (3·03%)	2 (11·77%)
Coronary thrombosis	44 (20·37%)	33 (20·00%)	4 (23·52%)
Pulmonary oedema	11 (5·09%)	6 (3·64%)	—
Poisoning	10 (4·63%)	8 (4·85%)	1 (5·88%)
Injuries	3 (1·39%)	2 (1·21%)	—
Other	30 (13·89%)	23 (13·94%)	2 (11·77%)
Totals	216	165	17

N.B. The figures in brackets in columns 2, 3 and 4 denote percentages of the total figures shown at the foot of the columns.

Appendix IV

Suggested Amendment to St. Andrew's Scottish Ambulance Service Instructions to Staff Regarding Oxygen Therapy and Resuscitation* Equipment

1. General

Ambulances operated by the Service are equipped with one or more of the undernoted items for use when resuscitation or oxygen therapy is required, and all officers, drivers and attendants are required to be thoroughly conversant with their operation:

(a) *Bag and Mask Resuscitators*

There are three types in use all basically similar in design. These are the AMBU, the RESUSCI and the AIR VIVA. Using free air they enable artificial respiration to be carried out for prolonged periods, with a minimum of effort. When oxygen is available in the ambulance, it should be added at a rate of 6 litres of oxygen per minute, on the HIGH setting of the regulator to the inlet valve of the bag and mask resuscitator in all cases where artificial respiration is required.

(b) *Stephenson Minuteman Resuscitator*

This is a compact, portable unit which operates on oxygen carried in two 72 gallon capacity cylinders. It can be used for resuscitation which is carried out automatically, for oxygen therapy, or for the aspiration of vomit, blood, etc., from the patient's airway. A properly used Bag and Mask resuscitator can be just as effective as the Minuteman Resuscitator, if oxygen is added to the inlet valve.

(c) *Oxygen Therapy Unit*

One 48 cu. ft. oxygen cylinder with a dry bobbin flow meter or alternatively a fixed flow regulator which gives a flow rate of 2 litres/min. on the MED setting, or 6 litres/min. on the HIGH setting. Oxygen is administered to the patient by either the Edinburgh Mask, when the MED setting is to be used, or by the Polymask when the HIGH setting is to be used. The decision as to which is to be used is described under paragraph 3.

(d) *Aspirator*

A portable foot operated suction pump is supplied with all Bag and Mask resuscitators for the purpose of removing vomit, blood etc. from a patient's airway. Aspirators should be thoroughly cleaned with soap and water after use.

2. Resuscitation:

(i) The main need in resuscitation is to get air in and out of the lungs and this can be achieved without special apparatus. Speed is of utmost importance if lives are to be saved. The mouth to mouth, or mouth to nose method of artificial respiration is excellent and no time must be lost in applying it. It is most important that a clear airway is provided

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* The word 'resuscitation' is used throughout this section to describe any form of assisted ventilation.

by tilting the patient's head back, and pulling his jaw forward. When mouth to mouth or mouth to nose artificial respiration is satisfactorily under way, then time can be given to bringing apparatus, or moving the victim whilst artificial respiration continues.

(ii) The Bag and Mask resuscitator, or the Stephenson Minuteman Respirator are used to continue artificial respiration. It is important to realise that oxygen is not a substitute for artificial ventilation in saving life. When it is available, as in the ambulance, oxygen should always be added to the inlet of the bag and mask resuscitator at a flow rate of 4 litres of oxygen per minute, or the HIGH setting of the regulator.

(iii) Resuscitation should be started in any unconscious patient who has irregular or absent breathing, or where there is a dusky blue colour to the lips or tongue (cyanosis). Special circumstances of resuscitation are:

(a) Respiratory obstruction, by blood, vomit, false teeth, or a paralysed tongue. Obstructions must be removed with the fingers, aided by suction, and the head positioned properly before resuscitation starts.

(b) Coal gas poisoning, where the patient is pink and unconscious. After opening windows and turning off the gas, mouth to mouth artificial respiration carries no risk to the rescuer, but he should double his depth of breathing. Oxygen added to a bag and mask resuscitator is very valuable.

(c) Drowning, where artificial respiration must be continued for long periods. If a victim of drowning does not become pink after 6 artificial breaths which caused his chest to expand, or he has an absent pulse at both wrists, then external cardiac compression should be given in addition to artificial respiration. Instructions for external cardiac compression are given in the First Aid Handbook.

3. Oxygen Therapy:

(i) Except when used for resuscitation purposes, as for example with oxygen added to the bag and mask resuscitator, or with the Stephenson Minuteman, oxygen should normally be administered only under professional advice. There are cases, however, in which it may be necessary for the ambulanceman to make a diagnosis and take a decision to use oxygen.

(ii) Oxygen can be given in high concentrations by a Polymask with the cylinder regulator set to the HIGH position, giving 6 litres of oxygen per minute. Alternatively, in other conditions it is advised that oxygen is only given in lower concentrations, by the Edinburgh Mask, with the regulator set to the MED position, giving 2 litres of oxygen per minute. The requirements for these two types of oxygen therapy are described below:

Oxygen in High Concentrations

(High setting, with Polymask)

(a) Pulmonary oedema, when the patient is very breathless, sitting upright, often coughing and maybe bringing up pink, frothy spit.

(b) Coronary thrombosis, where the patient complains of severe pain in the chest of sudden onset.

(c) Shock, from injury or loss of blood. The patient is pale and sweating and may have a rapid, feeble pulse.

Oxygen in Lower Concentrations

(Med setting, with Edinburgh Mask)

(a) Chronic bronchitis, where the patient or his relatives will tell of long-standing chest trouble, with winter cough and yellow spit, often for many years. The patient may appear blue (cyanosed).

(b) Asthma, where the patient is wheezy, and very breathless, and has usually had previous attacks.

(c) Pneumonia, where the patient has a fever, is sweating, may be cyanosed, and may complain of sharp pains in his chest.

Remember that it can be dangerous to give high concentrations of oxygen particularly to patients with chronic bronchitis. If in doubt, use the MED setting with the Edinburgh Mask.

4. Equipment: Operating Instructions:

(a) *Stephenson Minuteman Resuscitator*

Full instructions regarding operation are issued with each Minuteman, but the correct method of operation should also be demonstrated to all mobile staff by a Senior Officer. Any driver or attendant who has not been so instructed should immediately draw the attention of his superior to the fact.

(b) *Bag and Mask Resuscitators*

(i) When resuscitation is required the patient should be placed on his back and the operator should position himself behind the patient's head with the Resuscitator within easy reach of his right hand.

(ii) The operator should place his left hand under the patient's chin, and his right hand on the patient's head tilting it back as far as possible without using undue force. The last three fingers of the left hand should be kept under the patient's chin. After verifying that the tongue is well forward an airway may be inserted if available, in order to minimise obstruction by lips, teeth or base of the tongue, but on no account should this be done by an ambulanceman who has not been thoroughly trained in the correct method of insertion.

(iii) Keeping the head tilted with the left hand take hold of the valve on the bag with the right hand.

(iv) By holding the valve the mask is pressed against the patient's face making slight circular movements to ensure a good fit around the mouth/nose area.

(v) While still holding the valve and pressing the mask over the mouth and nose, place the left thumb and forefinger around the mask. The thumb and forefinger should form a "C" closely around the neck of the mask, so that it can be pressed and kept airtight over the mouth and nose of the patient. The last three fingers of the left hand should continue to be kept immediately under the chin of the patient, at the same time tilting the head backwards.

(vi) Compress the bag at about 15 times per minute with the right hand, releasing it suddenly and fully after each compression in order that the inflation valve can reverse and allow air from the lungs of the patient to escape to atmosphere.

(vii) Should oxygen be required the tubing from the oxygen cylinder should be connected to the air intake nipple on the ventilation bag, and the supply of oxygen opened to give a flow rate of 4 litres of oxygen per minute, proceeding thereafter as in (vi) above.

(viii) Resuscitation or oxygen therapy should be continued until the patient resumes normal breathing, recovers, arrives at hospital, or a doctor advises discontinuance. Should the supply of oxygen fail for any reason, resuscitation should be continued with free air after disconnecting the oxygen tubing.

5. Oxygen Therapy Units:

(a) *Regulator/Flowmeter*

(i) This instrument is very fragile and great care must be exercised when attaching to an oxygen cylinder. Before connecting the Regulator to a cylinder the cylinder valve should be opened momentarily to blow away any foreign matter which may have accumulated in the valve socket, and which might otherwise damage the mechanism of the unit.

(ii) On connecting the instrument to a fully charged 48 cu. ft. cylinder a reading of 2,000 p.s.i. (pounds per square inch) should be observed and this will give a supply of oxygen for approximately 4 hours at a flowrate of 6 litres per minute. A cylinder should be considered exhausted when the gauge shows a pressure of 500 p.s.i. and should be replaced immediately.

(iii) The oxygen flowrate is measured by means of a dry bobbin flowmeter which should be as nearly vertical in use as possible. Oxygen is led in at the lower end of the glass-domed flowmeter and the gas stream causes the bobbin to rise to a height which will depend on the rate of flow. An oxygen flowrate of 2 or 6 litres per minute is needed and should not be exceeded unless under medical or professional nursing supervision.

(iv) A fixed flow regulator is now being supplied with oxygen therapy units. These regulators are less fragile than the dry bobbin type and operate when the cylinder is lying on its side. A knurled knob selects one of two fixed positions, HIGH, giving 6 litres of oxygen per minute, and MED, giving 2 litres of oxygen per minute.

(b) *Operation*

(i) Connect the tubing to the outlet nipple.

(ii) Open the valve on the cylinder.

(iii) When using the bobbin flowmeter, adjust the valve on the flowmeter until the top of the bobbin is in line with the scale reading, of either 2 or 6 litres of oxygen per minute, as described in 3 above.

(iv) Ensure a free flow of oxygen through tubing and mask, explaining to the patient, if conscious, what you are doing before positioning the face mask.

(v) It is important that the mask be held firmly in position round the nose and mouth. If a Polymask is being used the chin should be inserted first, and the wire on the outer edge of the mask bent to fit closely round the nose and mouth. The Polymask and Edinburgh mask should be discarded after use by the patient, and the other types thoroughly cleaned with soap and water at the first opportunity.

6. Precautions with Oxygen Cylinders:

(a) Cylinders containing oxygen are identified by being painted black from base to shoulder, and white from shoulder to valve. On receiving a fresh cylinder the colour markings should be checked and no cylinder other than those painted black and white should be used when oxygen is required to be given to a patient.

(b) Cylinders should be protected from extremes of weather and kept in a ventilated place well away from combustible materials. At Depots they should preferably be stored upright in suitable stands, but if these are not available they should be stacked horizontally — each stack containing not more than 4 layers of cylinders.

(c) When being carried in an ambulance cylinders should not be allowed to roll about the floor, but should be placed in the oxygen bottle holder provided and securely clamped, or placed in the carrying box of the Minuteman, in the case of 72 gallon cylinders.

(d) Empty cylinders should be kept separate from full ones making it unnecessary to open valves to find out if they are full or empty. It is common practice to mark M T in chalk on the side of the empty ones, but any other distinctive sign can be used.

(e) All cylinders should be carefully handled to avoid metal fractures or damage to fittings. On no account should they be used as "rollers" for moving heavy objects.

(f) Cylinders and valves should not be lubricated with oil or grease, nor handled with oily or greasy hands, gloves or rags.

(g) Excessive force should never be used to open or close cylinder valves.

(h) Before fitting a regulator to a cylinder make sure that all dirt and dust is cleaned from the cylinder valve by opening the valve slightly and allowing a short blast of oxygen to escape, thereafter closing the valve promptly.

(i) Having fitted the regulator, test for any leakage before putting the cylinder into use. A faulty or suspect cylinder should never be used, but should be labelled immediately and returned to the British Oxygen Company.

(j) Oxygen under pressure is very dangerous when exposed to an open flame and should be kept away from any possible source of ignition, i.e. lighter, cigarettes or pipes, naked lights, etc., and this is particularly important when oxygen is being administered to a patient.

(k) In the event of fire in an Ambulance Station or in an ambulance, oxygen cylinders not directly involved in the blaze should be moved to safety as soon as possible. If any cylinders are heating up spray with cold water if at all possible, but make sure you are in a position of safety in case of explosion.

(l) Any leaking cylinders should be taken outside so that the gas can escape and disperse safely. If fire occurs it should only be extinguished by closing the valve or plugging the leak, but if it is unsafe to approach the cylinder to do this, no attempt should be made to extinguish the cylinder in any other way. All that can be done is to cool the cylinder with a water spray. Any cylinders or valves involved in a fire should not be used until they have been examined by the British Oxygen Company.

February, 1968.

Appendix V

Maternal Arterial PO_2 in Late Normal Pregnancy, breathing Air, unanaesthetised

Author	Number of Patients	Duration of Pregnancy (weeks)	Method of PO_2 determination	Mean PO_2 mm Hg	S.D. or range mm Hg
Vasicka *et al.* (1960)	31	"Normal Term"	Micro-cathode O_2 electrode	96.2	13.0
Rooth and Sjostedt (1962)	16	37–42	Micro-cathode O_2 electrode Patient sitting	Not given	89–110
Stenger *et al.* (1964)	5	38–40	Van Slyke S_aO_2	Not given	37–95
Wulf (1964)	63	Term	Dropping Hg. Electrode	90.7	75–125
Stenger *et al.* (1965)	61	14–44	Van Slyke S_aO_2	47	29–140+

NOTES: Calculations of PO_2 from the S_aO_2 (oxygen saturation) are made on the assumption of an arterial pH of 7.45 (Rooth and Sjostedt, 1962).
S.D. refers to standard deviation, where this is given.

Appendix VI

The Apgar Scoring System

Various methods have been devised to permit of greater accuracy in the assessment of the condition of an infant at birth. The most generally employed in paediatric practice was introduced by Dr Virginia Apgar (Apgar *et al*, 1958). It requires that a score (0-2) be given to each of five clinical observations (see table). The score is made at a stated time after complete delivery of the infant from the mother e.g. one or two minutes. Like all clinical methods of assessment the Apgar score is subject to observer variation and it does not correlate closely

Score	2	1	0
Heart rate	> 100/min.	< 100/min.	Nil
Respiration	Established or crying	Gasping only	Apnoeic
Colour	Good	Peripheral Cyanosis	Generalised cyanosis or pallor
Response to Stimulation (soles of feet or nostrils)	Good	Poor	None
Muscle tone	Good	Flexion movements only	None

with blood gas measurements. It also has the disadvantage that although each of the observations has the same scoring range they are not of equal importance. Thus the heart rate and state of respiration are much more significant of the infant's condition than muscle tone or response to stimulation. None the less, this system is of value in maternity units in that it points to those infants whose condition at birth should arouse anxiety and initiate some resuscitatory measures. An Apgar score of 5 or less is an indication that the infant should be seen promptly by a doctor. It is in this group also that difficulties in maintaining adequate respiration are likely subsequently to arise and the Apgar score is, therefore, of some value in pointing to those infants who merit continued and careful medical supervision. Some paediatricians think that the Apgar score taken 5 minutes after delivery is of greater prognostic value than after the more usual period of 1 minute.

Apgar, V., Holaday, D. A., James, L. S., Weisbrot, I. M., and Berrien, C. (1958). J. Amer, med. Ass., *168*, 1958.

Appendix VII

Manufacturers of Oxygen Therapy Equipment

Equipment manufactured by the following companies has been mentioned in Chapter 2.:

Air-Shields (U.K.) Ltd., 10 Towerfield Road, Shoeburyness, Southend-on-Sea.

Aldington Laboratories Ltd., Aldington Firth, Ashford, Kent.

Ambu International (Denmark), Scottish Representative, 41 Polmaise Road, Torbrex, Stirling.

Bard-Davol Ltd., Valley Bridge Road, Clacton-on-Sea, Essex.

The British Oxygen Co. Ltd., Medical Department, Hammersmith House, London, W.6.

BXL Ltd.

Draeger Normalair Ltd., 27/31 Minshull Street, Manchester, 1.

Eschmann Bros. & Walsh Ltd., 24 Church Street, Shoreham-by-Sea, Sussex.

J. G. Franklin & Sons Ltd., 51 Manchester Street, London, W.1.

The Walter Kidde Co. Ltd., Belvue Road, Northolt, Greenford, Middlesex.

Portland Plastics, Ltd., Hythe, Kent.

Siebe Gorman & Co. Ltd., Davis Road, Chessington, Surrey.

Valu-Air Ltd.

Vickers Ltd. (Oxygenaire), Medical Group, Basingstoke, Hants.

Printed in Scotland for Her Majesty's Stationery Office
by Highland Printers Ltd., Inverness. Dd. 242132 K

Three earlier reports from the Scottish Health Services Council

The Young Chronic Sick

The Report of a Committee appointed to consider the arrangements for the care and treatment of young people suffering from some form of chronic illness (other than mental illness). The Committee made twenty-one recommendations.

1*s.* 6*d.* (1*s.* 10*d.* by post)

Alcoholics

The health services for the treatment and rehabilitation of alcoholics are considered in this Report. 2*s.* 9*d.* (3*s.* 1*d.* by post)

Rheumatic Fever in Scotland

The Report of a Committee which was appointed to consider the problem of rheumatic fever and rheumatic heart disease in Scotland with particular reference to the incidence of these diseases and the means of prevention. 2*s.* 9*d.* (3*s.* 1*d.* by post)

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