

RESPIRATORY FAILURE AND OXYGEN THERAPY

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Definition

Respiratory failure may be defined as the state due to disordered function of the lungs in which there is a substantial decrease of the partial pressure of oxygen (P_{O_2}) in the arterial blood with or without a substantial increase of the partial pressure of carbon dioxide (P_{CO_2}).

Respiratory failure may be acute or chronic, but patients most often present with an acute exacerbation superimposed upon a chronic failure.

More generally useful criteria for failure may be based upon normal values at rest and at sea level which are 80-110 mm Hg for oxygen, and 36-44 mm Hg for CO_2 . For clinical purposes, however, the following criteria for failure, which are somewhat beyond normal limits, are more useful so that the definition of respiratory failure may be re-stated as follows "Respiratory failure is present in a subject at rest and at sea level if because of impaired respiratory function the arterial blood P_{O_2} is below 60 mm Hg or the Blood P_{CO_2} is

above 49 mm Hg" (Campbell 1965, Scadding 1966).

Classification

In practice if air is breathed an increase in the alveolar and arterial P_{CO_2} is inevitably accompanied by some reduction in the alveolar and arterial P_{O_2} because carbon dioxide is some twenty times more diffusible than oxygen. A useful classification may be made of two types of respiratory failure based on the presence or absence of Hypercapnia.

Types of Respiratory Failure

- I HYPOXAEMIA WITH HYPERCAPNIA
This may be caused by:
 1. *Inadequate Ventilation* due to
 - a) Inadequate neural drive or muscular power
and/or
 - b) Inadequate pulmonary or thoracic mechanics.
 2. Ventilation: Perfusion imbalance,

particularly reduced perfusion of adequately ventilated lung — (increased physiological dead space).

This type of respiratory failure is produced by ventilatory failure. A good clinical example of this type of failure is chronic bronchitis with or without pulmonary emphysema which produces both inadequate pulmonary mechanics and ventilation: perfusion imbalance.

II HYPOXAEMIA WITHOUT HYPERCAPNIA

This may be caused by:

1. Inadequate oxygen transfer due to
 - a) An inadequate alveolar-capillary membrane and/or
 - b) An inadequate pulmonary capillary bed.
2. Ventilation: perfusion imbalance particularly reduced ventilation of perfused lung — (increased shunt-like effect).

This type of failure is associated with defects of diffusion and/or distribution. A good clinical example of this type of failure is pulmonary congestion and oedema. A further example is the “alveolar capillary block syndrome” seen in such conditions as interstitial lung disease and in the diffuse pulmonary fibrosis of sarcoidosis.

Two important points which are very closely related and influence oxygen therapy in Respiratory failure must be mentioned. These are the Normal Control of Ventilation and the Oxygen Dissociation Curve.

Control of Ventilation

The volume of breathing (pulmonary ventilation) is normally influenced or controlled by two factors. These are the Medullary centres (Respiratory Centre) and the Aortic and Carotid Chemoreceptors.

The “Medullary centres” are chiefly influenced by the chemical state of the blood particularly its CO_2 Tension (P_{CO_2}); the higher the P_{CO_2} the greater the stimu-

lus on the medullary centre resulting in increased pulmonary ventilation.

The aortic and carotid chemoreceptors are chiefly influenced on the other hand by anoxia which produces reflex stimulation of breathing through the medullary centres.

The Oxygen Dissociation Curve

The “S” shape of the oxygen dissociation curve is very important clinically. A considerable reduction of the O_2 tension (P_{O_2}) below the normal arterial value does not significantly reduce the oxygen saturation of the arterial blood. It is seen from the curve in Fig. 1 that a reduction of oxygen saturation below 90% does not occur until arterial blood oxygen tension (P_{O_2}) has fallen to 60 mm from its normal value of 80-110 mm Hg. It is also seen from the curve that the change in shape below 60 mm Hg becomes very steep, which means that any further reduction in P_{O_2} causes a disproportionately severe desaturation. Conversely as a result of this sudden change in shape of the oxygen dissociation curve below 60 mm Hg slight increases in the oxygen tension in a patient with dangerous hypoxaemia in the region 30-50 mm Hg causes a disproportionate increase in the oxygen saturation of the blood with considerable benefit to the patient.

Oxygen Therapy in patients who have both hypoxaemia and hypercapnia

The importance of the above physiological observations is apparent when considering oxygen therapy in this type of patient. The “respiratory centre” of these patients has become insensitive to the high P_{CO_2} and they rely on anoxic stimulation of the carotid and aortic bodies to maintain their ventilatory drive. If these patients are given oxygen in high concentration, the anoxic stimulation is removed and hypoventilation with further increase of P_{CO_2} will result. Progressive loss of consciousness due to CO_2 Narcosis may follow. Mental changes following oxygen therapy were first reported by Barach

(1938) and he was the first to suggest the use of low concentration of oxygen. Donald (1953) suggested the administration of oxygen by tent (approx. 35-40%) with intermittent brief periods breathing air. Campbell (1960) elaborated Barach's original concept and suggested that the continuous administration of an oxygen mixture controlled with an accuracy of + or - 1% in the range 24-35% would allow the relief of dangerous hypoxaemia without running the risk of a serious rise in P_{CO_2} . This is possible because a slight increase in the oxygen tension in the inspired air of a

patient with dangerous hypoxaemia (arterial P_{O_2} below 50 mm Hg) produces a disproportionate increase in the oxygen saturation of the blood to a safer level as can be seen from Fig. 1.

An arterial P_{O_2} of 50 mm Hg or less is considered by Hutchinson *et al.* (1964) as dangerous hypoxaemia and they propose that the aim of controlled oxygen therapy should be to provide a P_{O_2} of at least this level.

There are several methods available for giving controlled oxygen therapy:

1. Venturi Apparatus. (Oxygenaire

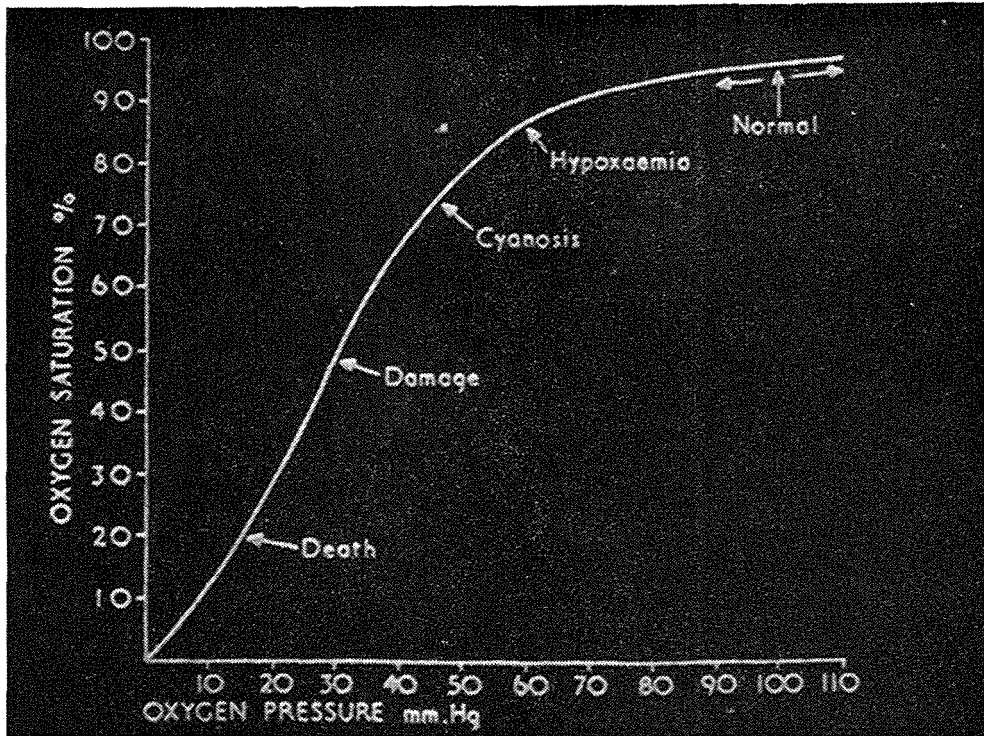


Fig. 1 (E. J. M. Campbell)

The oxygen dissociation curve relates the saturation of the haemoglobin to the partial pressure of oxygen (P_{O_2}) in the blood. In a normal subject at sea level the arterial P_{O_2} is 90-110 mm. Hg. and saturation 95-98%. The threshold of hypoxaemia is at the shoulder of the curve (60 mm. Hg., saturation 90%). Cyanosis can be confidently recognised only when the saturation falls to 70-75% implying an O_2 tension of 50 mm. Hg. (Comroe and Botelho, 1947; Medd *et al.* 1959). Hypoxic damage begins to appear in various organs at 30 mm. Hg. An arterial P_{O_2} of 20 mm. Hg. seems to be insupportable (Refsum, 1962; Campbell, 1965).

CAMPBELL, E.J.M. (1965) *Ann. N.Y. Acad. Sci.*, 121, 861.

COMROE, J.H. and BOTELHO, S. (1947) *Amer. J. Med. Sci.*, 214, 1.

MEDD, W.E., FRENCH, E.B. and WYLLIE, V. MCA. (1969) *Thorax*, 14, 247.

REFSUM, H.E. (1962) *Scand. J. Clin. Lab. Invest.*, 14, 545.

Ltd.) (Campbell 1960). Fig. 2.

This consists of a light alloy cone, the wide end being supported loosely before the nose and mouth by a perforated plastic face piece. In the base of the cone are two orifices for the admission of oxygen, the central one being of narrow bore, through which oxygen emerges as a high speed jet. The velocity of this jet is set by one of the calibrated gauges. By the Venturi principle this jet entrains atmospheric air through adjacent intake holes and the resultant mixture of oxygen and air is delivered to the patient. Campbell claimed that an air flow of 50 litres per minute is obtained when the jet is passing the calibrated 1.8 litres per minute. The additional wide bore oxygen pipe in the base of the cone is used to add oxygen, which does not entrain any further air, in order to vary the oxygen concentration the patient receives. Although the makers claim that the apparatus can deliver controlled amounts of oxygen between 24-35% with an accuracy of 1%, the error is nearer + or - 5% (Flenley *et al.*, 1963). They are however good enough for practical purposes.

2. The Venti Mask (Fig. 3). (Oxygenaire Ltd.) is a very cheap modification of the Venturi apparatus designed for use with conventional type of oxygen flow metre. This mask is in principle a single concentration Venturi mask. Two models are available which are claimed to provide two inspired concentrations of 24% and 28% respectively with a flow rate of 4 litres per minute. These approximations are suitable for most cases of respiratory failure.

3. The Edinburgh Mask (Fig. 4) (British Oxygen Co. Ltd.) (Flenley *et al.* 1963).

This is even simpler and less expensive. It consists of a loose fitting plastic face piece with a 5 cm (2 in) diameter orifice opening to the atmosphere. Into the centre of this orifice projects an oxygen delivery tube which emits oxygen at the desired flow rate into the mask at right angles to the air stream caused by the patient's ventilation. The inspired oxygen

concentrations which are obtainable at various flow rates when the minute volume of pulmonary ventilation is between 6 and 7 litres per minute are shown in Table I (Flenley *et al.* 1963).

TABLE I

Edinburgh Mask:
Inspired Oxygen Concentrations

Oxygen Flow (l./min)	Inspired Oxygen (per cent.)	
	Mean	Standard Deviation
0.5	23.4	1.6
1.0	27.4	4.1
2.0	29.9	7.7
3.0	35.2	5.8

The figures shown are only approximate and depend on the patient's minute volume, the concentrations being higher at lower levels of minute volume.

4. Nasal Catheters.

The single nasal catheter should be replaced by two types of double nasal catheter.

(1) Tudor Edwards spectacle frame. (Fig. 5). This has the disadvantage that it may be ill fitting and uncomfortable to wear.

(2) Addis nasal catheter. (Addis 1963) (Fig. 6).

This is made of soft translucent polythene tubing and consists of a long tube 7 mm in diameter which is worn over the ears like a spectacle frame and slung under the nose. It is held loosely in place by elastic behind the head. One end is sealed off, and oxygen is fed into the other end. This flows out through two smaller tubes placed about 12.5 mm ($\frac{1}{2}$ inch) inside the nares. Initially oxygen is given at a flow rate of 1 litre per minute and increased gradually.

The polymask and pneumask or any other similar type of mask are not to be used in this type of respiratory failure because they allow a lot of rebreathing of CO_2 .

When giving oxygen by any of the above methods treatment must be controlled by frequent estimations of the



Fig. 2



Fig. 3

blood gas tensions. For clinical purposes estimation of the P_{CO_2} is probably more important and more useful and also easier to perform than estimation of P_{O_2} . Oxygen must never be withheld from these patients since hypoxaemia is more dangerous for the patient than hypercapnia. If the P_{CO_2} is increasing with oxygen therapy this must be continued and other measures taken to increase pulmonary ventilation. Such measures would include: the use of respiratory stimulants, tracheostomy with or without intermittent positive pressure respiration. Other measures that must be considered are control of infection by antibiotics and treatment of heart failure by digitalis and diuretics.

Oxygen therapy in conditions not associated with retention of carbon dioxide

In acute pulmonary conditions where parts of normal lung can compensate by hyperventilation for the diminished excretion of CO_2 in the diseased portion — for example lobar pneumonia, pneumothorax, pulmonary embolism — there is little danger of CO_2 retention and the administration of 100% oxygen for limited periods is not only safe but relieves tissue hypoxia by increasing the amount of oxygen carried in solution. Hundred per cent. oxygen administration does not mean 100% oxygen in the inspired air but owing to inefficien-

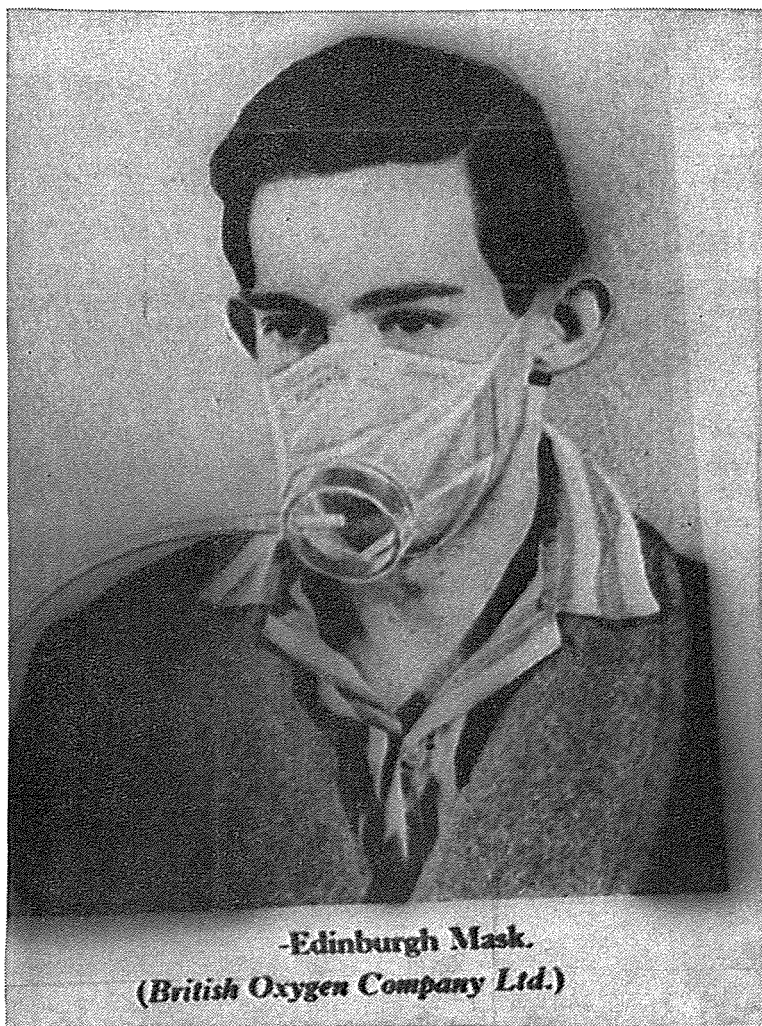


Fig. 4

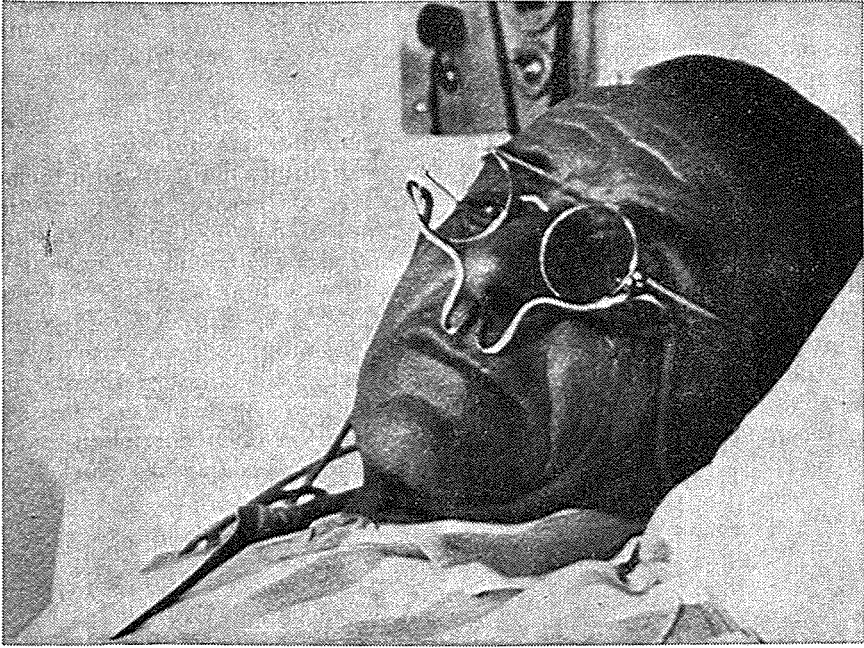


Fig. 5



Fig. 6

cies of the apparatus used the concentration in the inspired air seldom rises above 60% and is more usually round 40%. This is an advantage because it has been shown that prolonged use of oxygen in high concentration may be harmful (Dickens, F. 1964).

Humidification

This is essential in all the methods of oxygen administration except those based on the Venturi principle (Venturi apparatus and ventimask). This should be done by incorporating an efficient nebulizer in the system. Bubbling the oxygen through water is not sufficient.

Conclusion

The physiological basis and rationale of oxygen therapy in respiratory failure has been explained. The importance of controlled oxygen administration in patients who have anoxia combined with hypercapnia has been emphasized and the dangers of uncontrolled oxygen therapy stressed. Various apparatus suitable for oxygen administration have been described.

The most suitable of the various apparatus mentioned is the Venturi apparatus (Oxygenaire Ltd.) but this is expensive because of the necessity of having a gauge.

The Venti mask and the Edinburgh mask are much cheaper modifications. At a first glance there appears to be very little difference between them but the Edinburgh mask has the disadvantage that the oxygen concentration of the inspired air varies with the minute volume of the patient's respiration. The oxygen for use in the Edinburgh mask also has to be humidified by an appropriate humidifier. The Venti Mask is based on the Venturi principle like the Venturi apparatus and does not have these disadvantages; it is therefore to be preferred.

The danger of anoxia for the patient cannot be overstressed and this must be given prime importance during the treatment of a patient having anoxia combined with hypercapnia. A check on the P_{CO_2} must be kept, however, while oxygen is being administered to the patient by means of all forms of apparatus. There are several ways of checking the P_{CO_2} , some more complicated than others, but the Rebreathing method of Campbell and Howell (1962) is simple enough (although giving only a rough calculation) and suitable for use in a sideroom of a ward.

The management of respiratory failure must rest on physiological principles and in conclusion I wish to quote R. V. Christie who stated in the Frederick Price Lecture for 1963 to the Royal College of Physicians of Edinburgh that "death from respiratory failure can almost always be delayed or prevented, provided the physician or surgeon is aware of the hazards involved and provided that adequate facilities are available."

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References:

- ADDIS, G.J. (1963) *Lancet* 1, 1084.
 BARACH, A.L. (1938) *Ann. Int. Medicine*, 12, 454.
 CAMPBELL, E.J.M. (1960). *Lancet*, 2, 10.
 CAMPBELL, E.J.M. (1965). *Brit. Med. J.*, 1, 1451.
 CAMPBELL, E.J.M. and HOWELL, J.B.L. (1962) *Brit. Med. J.*, 2, 630.
 DICKENS, F. (1946) *Biochemical Journ.* 30, 145.
 DONALD, K.W. (1953), *Lancet* 1, 495.
 FLENLEY, D.C., HUTCHINSON, D.C.S., DONALD, K.W. (1963) *Brit. Med. J.*, 2, 1081.
 HUTCHINSON, D.C.S., FLENLEY, D.C., DONALD, K.W. *Brit. Med. J.*, 2, 1159.
 SCADDING, J.G. (1966) *Lancet* 1, 701.

