Before discussing the possibility of using oxygen under high pressure (OHP) clinically in circulatory or respiratory emergencies it is essential that the basic physiology should be fully considered.

APPLIED PHYSIOLOGY

A patient is placed in a pressure chamber and breathes oxygen in order to increase the oxygenation of an organ or tissue which has been rendered anoxic or ischaemic by disease or accident. The theory is simple; successful practical application depends on the appreciation of certain difficulties.

Table I shows the “ideal” alveolar oxygen partial pressures which are attainable with oxygen at 1 and 2 atmospheres absolute. To reach these levels, however, the inspired gas must consist of 100 per cent oxygen and this is not easy in a patient breathing oxygen from a mask. McDowall et al. (1964) have shown that the commonly used B.L.B. mask when applied to an adult with a fresh gas flow of 8 litres a minute produces an alveolar oxygen partial pressure which is only 60 per cent of the maximum possible. This problem arises only in the larger pressure chambers which are pressurized with air; smaller chambers, such as that described by Hopkinson and Williams (1963) are usually pressurized with 100 per cent oxygen so that this particular difficulty is avoided. Very high alveolar partial pressures can, however, easily be attained in anaesthetized or unconscious patients who can be intubated.

Once the oxygen has been successfully introduced into the alveoli, the next step is its transfer to the arterial blood. It seems that at high alveolar partial pressures of oxygen, diffusion across the alveolar-capillary membrane is never a limiting factor in oxygen transport (Comroe et al., 1962); we can therefore assume that the pulmonary end-capillary blood has an oxygen tension equal to the alveolar partial pressure. If an alveolar-arterial oxygen difference occurs it must be the result of “shunting”. It has been shown that, at least at 2 atmospheres of oxygen, arterial oxygen tensions can reach the expected values in the conscious subject (McDowall et al., 1964). Pulmonary shunting does not then constitute an obstruction to the introduction of high pressures of oxygen into the blood of the conscious patient, provided, of course, that pulmonary oxygen toxicity is avoided. This is not, however, the case during general anaesthesia (McDowall, 1964).

AVAILABLE OXYGEN

To be therapeutically valuable, however, the oxygen in the arterial blood must be transported to the organ or tissue where it is required. This depends on the total cardiac output and on the peripheral distribution of the cardiac output. The product of the cardiac output and arterial oxygen

<table>
<thead>
<tr>
<th>Inspired gas</th>
<th>Alveolar $P_{O_2}$</th>
<th>Alveolar $P_{CO_2}$</th>
<th>Alveolar $P_{N_2}$</th>
<th>Alveolar $P_{H_2O}$</th>
<th>Total pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air 1 atmosphere</td>
<td>105</td>
<td>40</td>
<td>568</td>
<td>47</td>
<td>760</td>
</tr>
<tr>
<td>Oxygen 1 atmosphere</td>
<td>673</td>
<td>40</td>
<td>0</td>
<td>47</td>
<td>760</td>
</tr>
<tr>
<td>Oxygen 2 atmospheres</td>
<td>1433</td>
<td>40</td>
<td>0</td>
<td>47</td>
<td>1520</td>
</tr>
</tbody>
</table>
Table II

The oxygen content of arterial blood at a haemoglobin concentration of 14.5 gm/100 ml.

<table>
<thead>
<tr>
<th>Inspired gas</th>
<th>Haemoglobin saturation (per cent)</th>
<th>Oxygen dissolved in plasma (ml/100 ml)</th>
<th>Total oxygen content (ml/100 ml)</th>
<th>Extra oxygen (ml/100 ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air 1 atmosphere</td>
<td>97</td>
<td>0.3</td>
<td>19.1</td>
<td>2.4</td>
</tr>
<tr>
<td>Oxygen 1 atmosphere</td>
<td>100</td>
<td>2.1</td>
<td>21.5</td>
<td>2.1</td>
</tr>
<tr>
<td>Oxygen 2 atmospheres</td>
<td>100</td>
<td>4.2</td>
<td>23.6</td>
<td></td>
</tr>
</tbody>
</table>

content has been termed total available oxygen (Nunn and Freeman, 1964). From table II it can be calculated that the arterial oxygen content is increased by 12 per cent on breathing oxygen at 1 atmosphere and by 24 per cent at 2 atmospheres. However, there would be no increase in total available oxygen if oxygen breathing greatly depressed the cardiac output, i.e.:

Cardiac output × arterial oxygen content = available oxygen

e.g.
5000 ml/min × 19.1 ml/100 ml = 955 ml/min on air
4450 ml/min × 21.5 ml/100 ml = 955 ml/min on oxygen 1 atmos.
4040 ml/min × 23.6 ml/100 ml = 955 ml/min on oxygen 2 atmos.

In this example a reduction of 11 per cent in cardiac output at 1 atmosphere or a reduction of 19 per cent at 2 atmospheres would prevent any increase in total oxygen availability.

Changes in Cardiac Output

What evidence is available concerning the effects of oxygen on cardiac output? Daly and Bondurant (1962) found that 100 per cent oxygen at atmospheric pressure reduced the cardiac output of normal volunteers by 11 per cent; Eggers et al. (1962) obtained almost precisely the same result (a 12 per cent reduction). These reductions would almost completely negate the effects of any rise in arterial oxygen content on total available oxygen. Both groups found that most of the reduction in cardiac output was due to a decrease in heart rate and Daly and Bondurant made the important observation that atropine, by abolishing the change of rate, completely prevented an oxygen-induced depression of cardiac output (an observation, incidentally, which could have a bearing on the use of atropine in premedication). Although they did not study hyperbaric oxygen, Daly and Bondurant observed the effects of a number of different inspired oxygen percentages at normal pressure and found that the reduction in heart rate, and therefore probably the depression of cardiac output, bore a linear relationship to the inspired oxygen concentration. It would, however, be unwise to extrapolate this finding to supra-atmospheric oxygen pressures. McGuiness (1964) also, in a study of healthy volunteers, found a reduction in cardiac output with oxygen at normal pressure. He extended his observations to the effects of oxygen at 2 atmospheres and found that cardiac output was not reduced more than it was by oxygen at normal pressure.

In a subject with a cardiac output of 5 l/min, it is possible to calculate the total available oxygen using the data obtained from these studies:

Breathing air 5000 × 19.1 = 955 ml/min
at 1 atmos. O2 4450 × 21.5 = 955 ml/min
(i.e. an 11% reduction in cardiac output)

at 2 atmos. O2 4450 × 23.6 = 1050 ml/min
(i.e. no further reduction in cardiac output)

There is therefore an urgent need for studies of the effect of oxygen on the cardiac output of patients suffering from those conditions for which oxygen, either normo- or hyperbaric, may be indicated.

The Peripheral Vessels

The last link in the chain of tissue oxygen supply is the distribution of the cardiac output, and similar considerations of oxygen availability apply here. Vasoconstriction may so reduce the perfusion of a particular organ that the total amount of oxygen delivered to it may not be increased despite a rise in blood oxygen content. There is abundant evidence that oxygen increases total peripheral resistance (Daly and Bondurant, 1962; Eggers et al., 1962), but does vasoconstriction occur in the most important peripheral beds, i.e. in the brain and heart?
HYPERBARIC OXYGEN IN RELATION TO EMERGENCIES

Lambertsen et al. (1953) have shown that oxygen at 3.5 atmospheres absolute reduces cerebral bloodflow by 25 per cent. Jacobson et al. (1963) found that a 21 per cent reduction in the bloodflow through the cerebral cortex of anaesthetized dogs was produced by oxygen at 2 atmospheres absolute. I have used Lambertsen's results to calculate the available oxygen per 100 gm of brain (fig. 1). It will be seen that, by comparison with the increases in available oxygen which would have occurred had there been no change in cerebral bloodflow during oxygen administration, the actual increases observed were very modest (indeed at 1 atmosphere of oxygen there would appear to have been a small reduction in available oxygen).

![Available Oxygen](image)

**Fig. 1**

Changes in oxygen available to the brain during the administration of 100 per cent oxygen at 1 and 3.5 atmospheres absolute (based on the findings of Lambertsen et al., 1953). The white columns indicate the percentage changes which would have occurred had cerebral bloodflow not altered. The black columns show the actual changes observed. The discrepancies are due to the cerebral vasoconstrictive action of oxygen.

More direct assessment of the effects of oxygen administration can be made by measuring the oxygen tension in the venous blood draining from an organ under study or by measuring the actual tissue oxygen tension. In the case of the brain, Lambertsen et al. (1953) measured a rise in the oxygen tension of the jugular venous blood from 38 mm Hg on air to 76 mm Hg at 3.5 atmospheres of oxygen. Jacobson et al. (1963) reported a rise of 21 mm Hg in the blood in the superior sagittal sinus of the dog when breathing 2 atmospheres of oxygen. Jamieson and Van den Brenk (1963) have shown that brain tissue oxygen tension rises during exposure to hyperbaric oxygen. Therefore, as regards the brain, there is little doubt that, in the healthy volunteer or experimental animal, hyperbaric oxygen increases tissue oxygenation, but that the rise is much less than might be anticipated.

In the case of the coronary circulation, Sobol et al. (1962) reported that inhalation of 100 per cent oxygen at normal pressure reduced coronary bloodflow. Despite this reduction in flow, the oxygen content of the coronary venous blood rose from 4.4 ml/100 ml to 5.6 ml/100 ml due to the increase in arterial oxygen content. These results were, however, obtained from dogs deeply anaesthetized with thiopentone, some of which were hypotensive while others were receiving vasopressor drugs. There has been no study of the effects of hyperbaric oxygen on coronary bloodflow.

**CLINICAL APPLICATIONS**

The subject of the possible application of hyperbaric oxygen to circulatory and respiratory emergencies is very wide. For this reason the following discussion has been limited to those emergencies which are of particular interest to anaesthetists.

**PERIPHERAL CIRCULATORY FAILURE**

Since tissue hypoxia is a central feature of peripheral circulatory failure, it might seem logical to treat patients in this state with hyperbaric oxygen. However, clinical "shock" is so difficult a condition to analyze that critical assessment of the results of such treatment would be impossible, at least until a very large series had been collected. In the shocked patient many other well-proved forms of therapy must be used simultaneously, so that whether the outcome is favourable or not, it is still impossible to be sure what part, if any, is played by hyperbaric oxygen. Consequently, almost all the work on this subject has been done in experimental animals in whom carefully controlled shock can be produced and in whom the effects of different forms of therapy can be compared.

A commonly used type of experimental shock is produced by bleeding an anaesthetized dog to a low blood pressure, which is maintained for a period; thereafter the blood is reinfused and the survival rate measured. This preparation has
been used frequently for the assessment of many types of treatment and its effects are well documented. It is known that during the hypotensive period cardiac output falls, both arterial and venous oxygen saturations drop, a metabolic acidosis develops, coronary bloodflow falls and myocardial oxygenation is impaired. Work with this preparation has shown that raising the blood pressure with noradrenaline does not increase the survival rate (Catchpole, Hackel and Simeone, 1955), but administration of hypertonic solutions (Brooks et al., 1963) or the combination of correction of the metabolic acidosis and enrichment of the inspired air with oxygen (Manger et al., 1962) does improve the results. There is then evidence that oxygen has a beneficial effect in this situation; does oxygen under pressure produce an even greater improvement?

Attar et al. (1962) treated dogs with 3 atmospheres of oxygen during the hypotensive period and reported that the mortality rate was reduced from 83 per cent among the controls to 26 per cent in the treated group. Because of the small size of their pressure chamber, these workers were unable to make measurements on the dogs during the period of exposure to hyperbaric oxygen. They did find, however, immediately after decompression, that the blood pressure was 26 mm Hg higher in the treated group than in the controls and that the arterial and mixed venous oxygen saturations were higher. There was no difference in the degree of metabolic acidosis between the two groups, but the chamber animals hyperventilated more than did the controls, so that their fall in arterial pH was less. Clark and Young (1964, personal communication) have carried out a comparable study in a chamber large enough to accommodate the investigators and have found that even in the dog shocked by haemorrhage, hyperbaric oxygen causes a further rise in total peripheral resistance and tends to elevate the systemic blood pressure. In their series this increase in peripheral vasoconstriction resulted in a more severe metabolic acidosis in the treated group.

By what means could hyperbaric oxygen produce the dramatic improvement in survival claimed by Attar and his co-workers? Arterial desaturation occurs during hypotension as a result of an increase in physiological deadspace (Freeman and Nunn, 1963) but Freeman (1962) has shown that raising the oxygen percentage of the inspired gases to only 30 per cent is sufficient to correct this. The extra oxygen introduced into the plasma by OHP may help to elevate tissue oxygen tension, and Attar et al. (1962) did indeed show a rise in the tissue oxygen tension in both muscle and liver during haemorrhagic hypotension treated with oxygen under pressure.

As regards the brain, Attar et al. (1962) state that hyperbaric oxygen in the shocked dog "if anything, causes dilatation of the cerebral vessels because of the complicating effect of carbon dioxide". They say this because they measured a rise in mixed venous $P_{CO_2}$. Cerebral vasodilatation by carbon dioxide is, however, unlikely since Harper and Glass (1964) have shown that at these low levels of blood pressure the cerebral vessels are maximally dilated in an attempt to compensate for the reduced cerebral perfusion pressure and that carbon dioxide under these circumstances is unable to produce further dilatation.

Because of the known cerebral vasoconstrictor effect of oxygen, hyperbaric oxygen might indeed reduce, not increase, the already impaired cerebral bloodflow. Harper, McDowall and Ledingham (1964) have in fact studied this point by directly measuring cerebral flow in hypotensive dogs at 2 atmospheres of oxygen. They found that cerebral bloodflow fell with hypotension but that administration of oxygen at 2 atmospheres did not lower the flow further. Since the arterial oxygen content was increased and since flow was unchanged, OHP produced an improvement in the oxygenation of the brain during the hypotensive period. The rise in systemic blood pressure demonstrated by both Attar et al. (1962) and by Clark and Young (1964) would, of course, help by producing an increase in cerebral bloodflow. The oxygen tension of the myocardium has also been shown to drop drastically during haemorrhagic hypotension in the dog (Caliva et al., 1959), falling to near zero at a mean blood pressure of 20 mm Hg. Oxygen under pressure will therefore help here by the same mechanism as in the brain.

Treatment with hyperbaric oxygen has thus been shown to increase the tissue oxygen tension
in the brain, the liver and muscle during haemorrhagic hypotension. If low tissue oxygenation is the cause of death in these animals, then the improvement in mortality claimed by Attar et al. could be due to the partial reversal of such tissue hypoxia. However, most workers in this field believe that death following haemorrhagic hypotension in dogs is due to other causes, e.g. bowel necrosis following splanchnic vasocostriction (Marston, 1962). Furthermore, an earlier study of the effect of 3 atmospheres of oxygen on the course of haemorrhagic shock by Frank and Fine (1943) revealed no improvement whatever in the mortality rate.

One must therefore conclude that the experimental evidence on the application of hyperbaric oxygen to peripheral circulatory failure is inconclusive. Harper, McDowall and Ledingham (1964) have reported that in dogs rendered hypotensive by haemorrhage, the oxygen consumption of the cerebral cortex, which is low at this time, is increased by the administration of oxygen at 2 atmospheres absolute. This observation is encouraging in that it may indicate that tissue enzymes, partially inactivated as a result of hypotension, may be able to function again when they are exposed to the very high oxygen tensions which are attainable during hyperbaric oxygen administration.

TOTAL CIRCULATORY ARREST
Almost all the experimental and clinical work on circulatory arrest has been in relation to elective arrest with preliminary “drenching” of the tissues with hyperbaric oxygen. This obviously gives OHP the best chance to demonstrate any beneficial action it possesses because, during the time of the arrest, the tissues can draw on the small amount of extra oxygen in solution in tissue fluids. In addition, Norman (1964) has shown that exposure to hyperbaric oxygen reduces tissue oxygen consumption; this reduced oxygen requirement might be protective during the arrest. In emergency circulatory arrest, by definition there will be no time for preliminary hyperbaric oxygen “drenching”. However, a study of the elective arrest data may give pointers to the best that can be achieved with OHP.

CEREBRAL ANOXIA
It is often stated that the brain is the organ most vulnerable during circulatory arrest and that the period for which the circulation can be interrupted is equal to the duration of arrest after which the majority of cerebral neurones survive. Evidence has recently accumulated which challenges this view. If the circulation to the brain alone is interrupted by inflating a cuff round the neck while the oxygenation and perfusion of the rest of the body are continued unimpaired (artificial ventilation of the lungs is, of course, necessary) then the period of arrest without neurological damage is raised from 4 to 8 minutes in the rabbit (Schneider, 1963). The reason for this is that with total body circulatory arrest, the myocardium is not oxygenated, vasodilator substances accumulate in the tissues, and a metabolic acidosis builds up; consequently, when an attempt is made to restart the circulation, cardiac output and blood pressure are very low. Neurological damage in these circumstances is then the result of a period of total circulatory arrest followed by a period of inadequate cerebral perfusion.

The use of hyperbaric oxygen at the time when the circulation is being re-established may be beneficial to the brain, not only by virtue of the increase in the oxygen content of the blood, but also because cardiac output may be elevated as a result of improved myocardial oxygenation and the blood pressure raised on this account and because of an increase in the total peripheral resistance. Several groups of workers have measured the increase in time for which animals survive total circulatory arrest under high pressures of oxygen. Smith, G., et al. (1963) found that oxygen at 2 atmospheres increased the time of safe circulatory arrest from 4 to 8 minutes at normal body temperature. It may be of significance that this prolongation to 8 minutes with circulatory arrest under hyperbaric oxygen is the same as the time determined for the revival time of the brain when only the cerebral circulation is arrested. In other words, hyperbaric oxygen by improving the post-arrest cerebral perfusion may lengthen the revival time for the whole body to that of the brain alone. This is a point of the greatest practical importance to the present
discussion, for if OHP exerts part or all of its beneficial effects after and not prior to or during the arrest then it must have a part to play in resuscitation from emergency, in contradistinction to elective circulatory arrest. This question might be settled if a series of experiments was performed in which a group of animals was exposed to OHP before and during but not after the arrest, and compared with another group receiving OHP only after the termination of the arrest.

OTHER CONSIDERATIONS

Is there evidence of benefit from OHP in other ways after circulatory arrest? From further experiments on elective cardiac arrest, it seems certain that the incidence of ventricular fibrillation in the post-arrest phase is much lower in animals treated with OHP and defibrillation, if necessary, is much more likely to be successful (Meijne et al., 1962). Meijne and his co-workers believe that their success with defibrillation under OHP is due to the much better myocardial oxygenation which can be achieved during cardiac massage in these circumstances; indeed, in their series, spontaneous defibrillation occurred on several instances during cardiac massage.

Harper (1964, personal communication) has shown that after a period of circulatory arrest the oxygen consumption of the cerebral cortex is depressed for a considerable period. This depression in oxygen uptake is not due to inadequate oxygen availability because the uptake remained low long after adequate cerebral perfusion had been re-established. The failure to utilize oxygen was clearly indicated by a rise of about 10 per cent in the oxygen saturation of the cerebral venous blood. A similar continued depression of oxygen utilization occurs in the myocardium after a period of haemorrhagic hypotension despite full correction of blood pressure and blood volume (Edwards et al., 1954). These observations lead to speculation similar to that in the section on peripheral circulatory failure—that intracellular enzymes, unable to utilize oxygen supplied at normal tensions, may be able to do so when the oxygen is supplied at supra-atmospheric tensions.

After a period of circulatory arrest, cerebral oedema may produce cerebral hypoxia by imparding oxygen transport between capillaries and cells. The high arterial oxygen tensions attainable during hyperbaric oxygen exposure will partially overcome this by increasing the oxygen diffusion gradient (Pinkerton, 1962). Hulbert (1964), in a mathematical analysis, has shown that oxygen penetration into tissues after a period of tissue anoxia is increased by 40 per cent when the arterial oxygen tension is doubled.

Koch and Vermeulen-Cranch (1962) have reported on the use of hyperbaric oxygen in a patient after the occurrence of cardiac arrest on the operating table. This treatment was not carried out until 6 hours after the arrest so that the immediate post-arrest circulatory difficulties were over; the hyperbaric oxygen was therefore administered in an attempt to increase cerebral oxygen availability at a time when perfusion of the brain was imperilled by cerebral oedema and by posthaemorrhagic anaemia. This 1-hour period at 3 atmospheres of oxygen, in the opinion of the authors, improved the condition of the patient and prevented irreversible brain damage. However, the patient's condition was apparently already improving following the administration of urea and the transfusion of packed cells. As the authors very fairly conclude, the clinical improvement "was very suggestive of an active contribution of this treatment towards total recovery" (my italics).

LOCAL CIRCULATORY ARREST

Discussion on the use of hyperbaric oxygen in local circulatory emergencies such as myocardial infarction, cerebral infarction or limb embolism has been omitted. However, there is one local circulatory emergency which is of primary concern to anaesthetists and that is the arterial insufficiency which follows the inadvertent intra-arterial administration of thiopentone. There would appear to be good reason for treatment with hyperbaric oxygen in these rare but disastrous cases, since the resultant increase in the oxygen content of the arterial blood will partially compensate for the reduced arterial flow and so limit the loss of tissue which results. OHP has been shown to be of value in the rather similar vascular situation which follows traumatic arterial injury (Smith et al., 1961).
HYPERBARIC OXYGEN IN RELATION TO EMERGENCIES

RESPIRATORY EMERGENCIES
Every respiratory emergency results in one or more of the four basic derangements of respiratory function, i.e. alveolar hypoventilation, ventilation-perfusion imbalance, diffusion defect or pulmonary shunting. The effects on arterial oxygen tension of alveolar hypoventilation, ventilation-perfusion imbalance and reduced oxygen diffusing capacity become insignificant at high though subatmospheric alveolar oxygen tensions. Therefore, in the treatment of emergencies involving only these mechanisms OHP is not necessary. Pulmonary shunting is the one disturbance of pulmonary function in which OHP may be indicated, and even here lesser degrees of shunt will be corrected by 100 per cent oxygen or less. If significant arterial desaturation is still present after the institution of efficient oxygen administration at normal pressure then the use of hyperbaric oxygen would seem logical.

When pulmonary shunting is present the cardiac output can be considered to consist of two components: one component passes through ventilated alveoli and reaches the Po2 of the alveolar gas; the other component passes through unventilated alveoli and leaves with the same Po2 as exists in the mixed venous blood. The final arterial oxygen content is the resultant of the redistribution of oxygen which occurs in the left heart between these two components. By utilizing this idealized scheme it is possible to state in quantitative terms the difference which hyperbaric oxygen will make to a patient with pulmonary shunting, by calculating the maximum shunts which will be fully corrected by oxygen therapy (there is no need to include a reduction in cardiac output due to oxygen since this probably does not occur when the arterial oxygen tension is not raised by the oxygen):

Cardiac output × arterial O2 content = oxygen availability
5000 ml/min × 19.1 ml/100 ml = 955 ml/min

In shunting:

\[
(\text{volume} \times \text{mixed venous} \text{ shunted oxygen content}) + \left( \frac{\text{cardiac output} - \text{vol. shunted}}{\times \text{pulm. end. cap. O}_2 \text{ content}} \right) = \text{oxygen availability}^* \\
\]

When breathing 100 per cent oxygen:

\[
\left( x \times 13.5 \right) + \left( \frac{(5000 - x) \times 21.5}{100} \right) = 955 \text{ ml/min} \\
\Rightarrow x = 1500 \text{ ml/min or 30 per cent of cardiac output (where } x \text{ ml = volume shunted).} \\
\]

*Comroe et al., 1962, p. 131.

The points used to construct the graph have been obtained from the literature.
1. Binger et al. (1927)
2. Paine et al. (1941)
3. Smith, C. W., et al. (1963)
4. Karasewitch (1963)
5. Smith, J. L. (1899)
When breathing 100 per cent oxygen at 2 atmospheres:

\[
\frac{(y \times 13.5) + ((5000 - y) \times 23.6)}{100} = 955 \text{ ml/min}
\]

\[y = 2230 \text{ ml/min or } 45 \text{ per cent of cardiac output}
\]

i.e. in this case 100 per cent oxygen at normal pressure will allow complete correction of a shunt equal to 30 per cent of the cardiac output; 100 per cent oxygen at 2 atmospheres will prevent a fall in total available oxygen despite a shunt of 45 per cent of the cardiac output.

There is no need to list all the respiratory emergencies which may lead to pulmonary shunting since these are well known to anaesthetists; such a list would include such emergencies as Mendelson’s syndrome, aspiration collapse and “postperfusion lung”. The exact aetiology is in any case largely academic to the question of whether to use hyperbaric oxygen since, if arterial desaturation is present despite the administration of 100 per cent oxygen, then the arterial oxygen content will be increased by OHP therapy.

**TOXIC EFFECTS OF OXYGEN**

A more important problem is the duration of safe administration of such high oxygen pressures since most, though not all, of the causes of pulmonary shunting require oxygen treatment for several days. The danger, of course, is of oxygen toxicity which occurs in two forms: cerebral, manifested clinically as convulsions; and pulmonary, which results in pulmonary consolidation. Toxic doses of oxygen can therefore actually produce pulmonary shunting. However, in the type of case under discussion here where OHP is used to return the arterial oxygen tension to its normal level and no more, only pulmonary toxicity is possible since only the alveoli are exposed to high oxygen concentrations. Despite the likelihood that man is more resistant to pulmonary damage than are experimental animals (Donald, 1946), exposures much in excess of those shown in figure 2 should not be exceeded without the greatest care. While even normobaric oxygen can be harmful (Comroe et al., 1945; Smith, C. W., et al., 1963), the toxicity of oxygen rises very steeply at supra-atmospheric pressures (fig. 2).

**CONCLUSION**

The applied physiology of hyperbaric oxygen is based primarily on the concept of available oxygen at different pressures of air and oxygen. The effects of oxygen on the cardiac output and on its distribution are of vital importance.

The clinical applications of hyperbaric oxygen are (1) in peripheral circulatory failure where the experimental evidence, though hopeful, is inconclusive and (2) in total circulatory arrest. It would seem that the incidence of ventricular fibrillation in the post-arrest phase is much lower in animals treated with OHP and defibrillation, if necessary, more likely to be successful. OHP has a potential value in the treatment of accidental intra-arterial injection of thiopentone and in respiratory emergencies involving pulmonary “shunting”. The therapeutic use of hyperbaric oxygen in circulatory and respiratory emergencies should be guided both by an appreciation of its potentialities and a respect for its toxicity.

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