

B. Fischer K. K. Jain E. Braun S. Lehl

# Handbook of Hyperbaric Oxygen Therapy



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# Handbook of Hyperbaric Oxygen Therapy

With 33 Figures



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## Preface

Hyperbaric medicine involves the use of barometric pressure greater than that at sea level for the treatment of diseases. The term makes no distinction between air, oxygen or any other gas used as a medium of compression. Hyperbaric oxygenation (HBO) refers to the use of pure oxygen for breathing in a hyperbaric chamber via a mask or similar device or breathing freely in a monoplace chamber pressurized with oxygen. HBO is an intermittent, high-dose oxygen inhalation therapy. We have confined ourselves to the subject of HBO therapy and have not included oxygen therapy at normobaric pressures. With the exception of decompression sickness we have made no attempt to cover diving medicine as many excellent treatises are available on this subject.

Literature on HBO is extensive, and we estimate that the total number of publication on the subject of hyperbaric medicine during the past 150 years exceeds 20000, nearly half published during the past 30 years. No comprehensive textbook on this topic has ever been written in English, nor is there any bibliography more up to date than 1965. The books on the subject have consisted of monographs, reports of symposia and proceedings of the various international congresses on hyperbaric medicine. No definitive work has been published in the past 10 years.

We have attempted to fill the gap with a comprehensive review of the current state of knowledge and an assessment of the future potential. We have drawn freely on the published literature and interspersed it with our comments, observations and conclusions. Ideas for future research are presented. Our interest is oriented mainly toward rehabilitation. Our rehabilitation program has three goals:

1. To facilitate recovery of lost function and return the patient to work or other normal activity as soon as possible.
2. If the function of a part of the body, e.g., a group of muscles, does not recover, to compensate for it by improving the function of a complementary part, e.g., another group of muscles. This can be achieved by means of exercise and retraining of movement patterns.



3. If working patients are unable to return to their original job, to improve their general physical and mental status and retrain them for another job.

HBO as an adjunct to physical therapy and brain jogging (a system of mental exercises) plays an important role in all three phases. An example is provided by a patient with stroke. HBO has important beneficial effects on exercise physiology and potential future applications in sports medicine, and may revolutionize our methods of training athletes for high performance.

We hope that this book will be useful to all those who are interested in the topic of HBO and serve as a source of up-to-date information on this subject. We have included a bibliography with 1650 entries, but must still apologize to those investigators whose work has not been cited due to lack of space.

Finally, we acknowledge the financial support of the Vinzenz V. Paul Stiftung of Basel, without which this work would not have been possible. We would also like to thank Frln. Doris Eble for her help in compiling the bibliography, Frln. A. Schnaiter for her help in the research and all our colleagues on the staff of the Schwerpunktklinik Klausenbach, too numerous to list here.

Nordrach, Federal Republic of Germany  
January, 1988

B. Fischer  
K. K. Jain  
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## Part I

# Basic Aspects

Interest in hyperbaric oxygenation can be traced back to antiquity. Aristotle mentioned a diving bell used by the Greeks in the year 337 BC at the battle of Tyre. The first recorded use of compressed air in medicine was the first to use compressed air in medicine was an English room called "domicilium" in which variable climatic and pressure conditions could be produced. According to Henry, "in times of good health this domicilium is proposed as a good expedient to help digestion, to promote insensible respiration, to facilitate breathing and expectoration, and consequently, of excellent use for the prevention of most affections of the lungs." However, there is no account in the literature of any application of Henry's proposed treatment.

A few years later in 1667, Hook (B. 1634-1695) demonstrated that something essential to life passes from the inspired air into the blood and that once this substance is extracted the expired air is no longer fit for breathing. A few years later, in 1674, John Mayhew defined the "serial something" that supports combustion as a specific compound of the air that he called "spiritus nitro-aereus." Oxygen was discovered by Priestley in 1775 and independently by Scheele in 1777. Priestley immediately pointed to a possible therapeutic use in medicine and also to possible dangers.

From the greater strength and vivacity of the flame of a candle in the pure air it may be conjectured that it might be peculiarly salutary to the lungs in certain morbid cases, when the common air would not be sufficient to carry off the phlogistic period effluvia fast enough. But perhaps we may also infer from these experiments, that though pure air might be very useful as a medicine, it might not be so proper for us in the usual healthy state of the body; for as a candle burns out much faster in dephlogisticated than in common air, so we might, as may be said, live out too fast and the animal power be too soon exhausted in this pure kind of air. A mortal at last may say that the air which nature has provided for us is as good as we desire.

The toxic effects of oxygen, observed by Lavoisier and Laplace in 1785, were a hindrance to the development of therapeutic uses. Half a century went by before there was renewed interest in oxygen therapy in England. Around the same time, in 1834, the Frenchman J. J. Berthollet constructed a copper chamber and used pressures of 2-4 atm to treat patients, particularly those with pulmonary affections. He attributed the sense of well-being experienced by the patients to the improvement in cerebral circulation. In the mid-nineteenth century the use of such chambers spread across Europe, that of Berthollet (1833) being the most famous. The dominant figure of that period was Paul Bert, known as the "father of pressure physiology." He studied the effects of various gas mixtures. An exhaustive review of the literature

# Chapter 1

## Historical Aspects of Hyperbaric Oxygenation

Interest in hyperbaric therapy can be traced back to antiquity. Aristotle mentioned a diving bell used by Alexander the Great in the year 332 BC at the battle of Tyre. The British physician Henshaw (1664) was the first to use compressed air in medicine. The chamber he used was an airtight room called "domicilium" in which variable climatic and pressure conditions could be produced. According to Henshaw, "In times of good health this domicilium is proposed as a good expedient to help digestion, to promote insensible respiration, to facilitate breathing and expectoration, and consequently, of excellent use for the prevention of most afflictions of the lungs." However, there is no account in the literature of any application of Henshaw's proposed treatment.

A few years later in 1667, Hooke (Bayliss 1924) demonstrated that something essential to life passes from the inspired air into the blood and that once this substance is extracted the expired air is no longer fit for breathing. A few years later, in 1674, John Mayhew defined the "aerial something that supports combustion" as a specific compound of the air that he called "Spiritus-nitro-aereus". Oxygen was discovered by Priestley in 1775 and independently by Scheele in 1777. Priestley immediately pointed to a possible therapeutic use in medicine and also to possible dangers:

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The toxic effects of oxygen, observed by Lavoisier and Sequin in 1789, were a hindrance to the development of therapeutic uses. Half a century went by before there was renewed interest in oxygen therapy in England. Around the same time, in 1834, the Frenchman Junod constructed a copper chamber and used pressures of 2-4 atm to treat patients, particularly those with pulmonary afflictions. He attributed the sense of well-being experienced by the patients to the improvement in cerebral circulation. In the mid-nineteenth century the use of such chambers spread across Europe, that of Bertin (1855) being the most famous. The dominant figure of that period was Paul Bert, known as the "father of pressure physiology." He studied the effects of various gas mixtures. An exhaustive review of the litera-



ture on this subject up to that time is to be found in the publication by Artzenius in 1887, which contains 300 references.

By 1860 the first therapeutic compression chamber in North America was built in Oshawa, Canada and later moved to Toronto. Dr. Corning of New York published a paper in 1891 dealing with the use of compressed air in the treatment of nervous and mental afflictions.

The best-known use of hyperbaric air as a form of therapy was that by Cunningham of Kansas City (Sellers 1965) during the epidemic of Spanish influenza that swept the USA during the closing days of World War I. Cunningham made the observation that the mortality was higher in areas of higher elevation and that there was a barometric factor. He constructed a therapeutic pressure tank and achieved remarkable improvement in patients who were cyanotic and unconscious. After a loss of compression owing to mechanical failure one night, all the patients died. This could be interpreted as proof of the effectiveness of this form of therapy. Dr. Cunningham, however, went too far in his enthusiasm and started to treat diseases such as syphilis, cancer and diabetes mellitus with compressed air. In 1928 he constructed the largest chamber ever built - five stories high and 64 feet in diameter. Each floor had 12 bedrooms with all the amenities of a good hotel. This project was condemned by the American Medical Association and during World War II the chamber was dismantled and scrapped.

It was not until 1920 that oxygen was used therapeutically to prevent the bends (Behnke 1942). In the 1950s the toxic effect of oxygen was utilized to induce convulsions as a substitute for electroconvulsive therapy (Lambertson 1955).

From 1956 Boerema carried out considerable pioneering work using high oxygen pressures in cardiac surgery (Meijne 1973). His studies on the use of physically dissolved oxygen to support life in the absence of hemoglobin and the application of hyperbaric oxygen in clostridial infections earned him his preeminence in the modern history of hyperbaric oxygen therapy.

A landmark in organizing and getting together the various workers in this field was the First International Congress on Clinical Applications of Hyperbaric Oxygenation, held in Amsterdam in 1963. The subsequent congresses are listed below. Since 1981 the meetings have been held at 3-year intervals.

- |      |      |                    |
|------|------|--------------------|
| II   | 1964 | Glasgow, Scotland  |
| III  | 1965 | Durham, USA        |
| IV   | 1969 | Sapporo, Japan     |
| V    | 1973 | Vancouver, Canada  |
| VI   | 1977 | Aberdeen, Scotland |
| VII  | 1981 | Moscow, USSR       |
| VIII | 1984 | Long Beach, USA    |
| IX   | 1987 | Sydney, Australia  |

After a review of the history of hyperbaric therapy during the previous several years, Jacobson et al. (1965) concluded, "If this form of therapy is to achieve a worthwhile and lasting place in the medical armamentarium, it can only do so on a firm basis of accurate physiological data on the effects of both pressure and oxygen obtained in experiments, as well controlled as clinical medicine will permit."

## Chapter 2

# Physical and Physiological Basis of Hyperbaric Oxygenation

Oxygen is essential for sustaining life in nearly all known organisms, the exception being anaerobic bacteria. The properties of oxygen are listed in Appendix 2. A brief description of the oxygen transport in the body and the basic physical laws governing it is essential for understanding the clinical applications of hyperbaric oxygenation (HBO).

### Physical Basis

The atmosphere is a gas mixture containing by volume, 20.94% oxygen, 78.08% nitrogen, 0.04% CO<sub>2</sub> and traces of gases like argon, helium and krypton. For practical purposes we will consider air to be a mixture of 21% oxygen and 79% nitrogen. The total pressure of this mixture at sea level is 760 mm Hg. Dalton's law states that in a gas mixture, each gas exerts its pressure according to its proportion of the total volume:

partial pressure of a gas = absolute pressure  $\times$  proportion of total volume of gas. Thus, the partial pressure of oxygen (pO<sub>2</sub>) in air is  $760 \times 21/100 = 160$  mm Hg.

Pressures exerted by gases dissolved in water or body fluids are different from those in the gaseous phase. The concentration of a gas in a fluid is determined not only by the pressure but also by the "solubility coefficient" of the gas. Henry's law formulates this as follows:

concentration of a dissolved gas = pressure  $\times$  solubility coefficient. The solubility coefficient is different for different fluids and is temperature-dependent, with solubility inversely proportional to temperature. When concentration is expressed as volume of gas dissolved in each volume of water and pressure is expressed in atmospheres, the solubility coefficients of the important respiratory gases at body temperature are as follows:

|                 |       |
|-----------------|-------|
| Oxygen          | 0.024 |
| CO <sub>2</sub> | 0.57  |
| Nitrogen        | 0.012 |

From this one can see that CO<sub>2</sub> is 20 times more soluble than oxygen, and oxygen twice as soluble as nitrogen.

## Physiological Basis

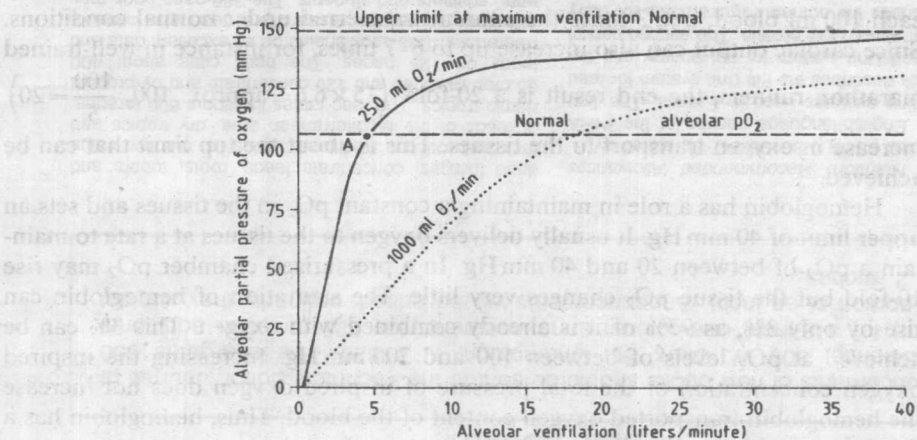
The physiological aspects of oxygenation can be discussed in three phases: ventilation, transport and utilization.

### Ventilation Phase

The function of respiration is to meet the demands of the metabolism for the supply of oxygen and the removal of  $\text{CO}_2$ . Oxygen is continually absorbed into the blood in the lungs and new oxygen is constantly entering the pulmonary alveoli. Figure 2.1 shows the effect of alveolar ventilation on the rate of oxygen absorption from alveoli on alveolar  $\text{pO}_2$ .

At a normal ventilatory rate of 4.2 l/min and oxygen consumption of 250 ml/min, the normal operating point in Fig. 2.1 is point A. During moderate exercise, when 1000 ml/min oxygen is absorbed, the rate of alveolar ventilation must increase four-fold to maintain the alveolar oxygen tension of 104 mm Hg. This figure also shows that alveolar ventilation can never increase the alveolar  $\text{pO}_2$  above 149 mm Hg as long as the subject is breathing atmospheric air at normal pressure, as this is the maximum oxygen content in the humidified air.

Carbon dioxide is constantly being formed in the body and discharged into the alveoli and is continually removed by ventilation, and these two factors determine the partial pressure ( $\text{pCO}_2$ ) and alveolar concentration of  $\text{CO}_2$ . The normal rate of  $\text{CO}_2$  excretion is 200 ml/min and  $\text{pCO}_2$  is 40 mm Hg. Alveolar  $\text{pCO}_2$  increases directly in proportion to rate of  $\text{CO}_2$  excretion and decreases in inverse proportion to alveolar ventilation.



**Fig. 2.1.** Effect of alveolar ventilation and rate on oxygen absorption from the alveoli on the alveolar  $\text{pO}_2$  (Guyton 1986)



## Transport Phase

The difference between  $pO_2$  in the alveoli (104 mm Hg) and  $pO_2$  of the venous blood (40 mm Hg), which amounts to 64 mm Hg causes oxygen to diffuse into the pulmonary blood. It is transported, mostly in combination with hemoglobin, to the tissue capillaries, where it is released for use by the cells. There the oxygen reacts with various other nutrients to form  $CO_2$ , which enters the capillaries to be transported back to the lungs.

During strenuous exercise, the body oxygen requirement may be as much as 20 times normal yet oxygenation of the blood does not suffer, because the diffusion capacity for oxygen increases four-fold during exercise. This rise results in part from the increased number of capillaries participating, as well as dilatation of the capillaries and also the alveoli. Another factor is that the blood normally stays in the lung capillaries about 3 times as long as is necessary to cause full oxygenation. Therefore, even during the shortened time of exposure on exercise, the blood can still become nearly fully saturated with oxygen. Normally 97% of the oxygen transported from the lungs to the tissues is carried in chemical combination with hemoglobin of red blood cells and the remaining 3% in a dissolved state in plasma. 1 g hemoglobin can combine with 1.34 ml oxygen. The normal concentration of hemoglobin is 15 g/100 ml blood. Thus, when hemoglobin is 100% saturated with oxygen, 100 ml blood can transport about 20 ( $15 \times 1.34$ ) ml oxygen in combination with hemoglobin. Since the hemoglobin is usually only 97% saturated, the oxygen content to the blood is actually 19.5 ml. However, in passing through tissue capillaries this amount is reduced to 14.5 ml ( $pO_2$  40 mm Hg and 75% oxygen saturation). Thus, under normal conditions, 5 ( $19.5 - 14.5$ ) ml of  $O_2$  is transported to the tissues by 100 ml blood. On strenuous exercise, which causes the interstitial fluid  $pO_2$  to fall as low as 15 mm Hg, only 4.5 ml oxygen remains bound with hemoglobin in each 100 ml blood. Thus 15 ( $19.5 - 4.5$ ) ml oxygen is transferred by each 100 ml blood, i.e., 3 times the amount transferred under normal conditions. Since cardiac output can also increase up to 6-7 times, for instance in well-trained marathon runners, the end result is a 20-fold  $\left(15 \times 6.6 = \text{approx. } 100, \frac{100}{5} = 20\right)$  increase in oxygen transport to the tissues. This is about the top limit that can be achieved.

Hemoglobin has a role in maintaining a constant  $pO_2$  in the tissues and sets an upper limit of 40 mm Hg. It usually delivers oxygen to the tissues at a rate to maintain a  $pO_2$  of between 20 and 40 mm Hg. In a pressurized chamber  $pO_2$  may rise 10-fold but the tissue  $pO_2$  changes very little. The saturation of hemoglobin can rise by only 3%, as 97% of it is already combined with oxygen. This 3% can be achieved at  $pO_2$  levels of between 100 and 200 mm Hg. Increasing the inspired oxygen concentration or the total pressure of inspired oxygen does not increase the hemoglobin-transported oxygen content of the blood. Thus, hemoglobin has a tissue oxygen buffer function.