HYPERBARIC

OXYGENATION

LEDINGHAM

HYPERBARIC OXYGENATION

PROCEEDINGS OF THE SECOND INTERNATIONAL CONGRESS

GLASGOW—SEPTEMBER 1964

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PREFACE

THE Second International Conference on Hyperbaric Oxygenation was held from 22nd to 26th September 1964, at the University of Glasgow. The University was glad to welcome as its guests the large number of visitors, from this country and overseas, who accepted our invitation to attend.

Preparing a Proceedings of such a Conference is notoriously a difficult and time-consuming task. We are greatly indebted to Dr Iain Ledingham for the vigour and assiduity he has applied to the task of editing it, and to his secretary, Miss Irene Russell, for her painstaking help. We owe our gratitude also to the publishers, Messrs E. & S. Livingstone, for their expedition in the production of the book.

CHARLES F. W. ILLINGWORTH.

Glasgow, 1965.

ACKNOWLEDGMENTS

I wish to thank Mr A. D. Bird and Dr A. B. M. Telfer for help in abstracting the original taped discussions, and Dr D. G. McDowall, Mr R. G. Clark and my wife for help with the final page proofs.

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UNIVERSITY OF GLASGOW

Second International Conference on Hyperbaric Oxygenation

OPENING ADDRESS

Professor SIR CHARLES ILLINGWORTH

FIRST of all, it is my very great privilege to welcome you all to this Conference. Some of you have been to Glasgow before and to these I would just say that we are very glad to see you back again and for those for whom this is their first visit, I would like to say that we all hope that you will find it both enjoyable and profitable. I am quite sure that all of you will find a very warm welcome from this hospitable, though rather murky, metropolis.

It is now one year since the First Hyperbaric Conference was held, very appropriately in Dr Boerema's clinic in Amsterdam, and I was delighted to get a letter from him two or three days ago to say that he will be here later with us this week on Thursday and Friday. We are very glad to welcome him here and also Dr Jacobson who arranged the very successful meeting in New York in January of this year.

Since those two meetings there has, of course, been more experience of hyperbaric oxygen and many new research projects are being started and carried through to completion. The time has come to present new findings and reassess some of the older ones. We have, as you have doubtless observed, a full and diverse programme. I think the diversity illustrates a very interesting phenomenon which we see quite often in the development of science. I was going to say, for the benefit of our visitors from Holland, that it was rather like one of the dykes bursting, but I think a better analogy would be, rather like some great river bursting its banks and flooding the alluvial country round about with its fertilizing influence. Since that very small gap appeared in Amsterdam seven or eight years ago the floods have spread and a tremendous volume of research experience has opened up and new territories are still being discovered. Accordingly this programme includes many new areas of research.

I think you will find the keynote is experimentation. We have reached the stage when it is very necessary to be sure of our facts. It is necessary to establish data which will stand up to a really critical

scientific investigation. This is, of course, extremely difficult in clinical applications and it will take a long time before the full value of hyperbaric oxygen can be determined in that field. Most of us are pretty certain about some aspects of it. Carbon monoxide poisoning is a case in point and also anaerobic infections. Many of us are pretty confident about others, but it will take a long time, many years, before we can get definite evidence. It is all the more important then that we should concentrate on the laboratory experience and that is what will be done during this Conference.

Another welcome development, I think, is that this flood of interest has taken us back to first principles and many of the papers in the next five days are related to basic subjects of physiology, biochemistry and the rest. As you know there are sections on respiratory physiology and many different papers on the vascular responses in the brain and different parts of the body and papers on related subjects of surgical shock and so forth. This is not to forget subjects that have been of interest for certainly 100 years—the subjects of decompression sickness and oxygen toxicity. It is a very diverse programme and I am sure you will find it interesting. I hope you will find it informative.

PART 1

DANGERS OF INCREASED ATMOSPHERIC PRESSURES

Chairman: Dr J. W. BEAN (Ann Arbor)

Introductory Remarks

I THINK each and every one of us fully recognize that it is a great privilege and honour to be present and participate in this Second International Conference on Hyperbaric Oxygenation which, I think, has been so appropriately dedicated to Sir Charles Illingworth, one of the great surgeons of our time. Of particular interest to us is his great enthusiasm in furthering the use of oxygen at high pressure clinically and surgically, and to him I am sure we owe a great debt.

The programme begins with a very important subject. It is not one which is the main theme of those of us who are interested in hyperoxygenation, but it is one of those ancillary problems which may involve stumbling blocks on the way to further use of oxygen clinically—the matter of the dangers of hyperbaric environments.

You will recognize that all of us who are concerned with this work are really compressed air workers, and we are thus subjected to the same hazards as the compressed air workers. It is as well for us to keep that in mind. A careless attitude to this aspect of the subject can lead us into a great deal of trouble and disrepute. This involves selection of personnel and the very careful recognition of the dangers of decompression. I am reminded of the report I read a few weeks ago that, in the period from January 1960 to August 1963, there were 37 fatalities in Florida waters alone, involving ' Scuba ' divers and skin divers, as compared with the U.S. Navy over a period of 15 years which, I think, had only six or seven fatalities and in which the exposures were much more severe. Admittedly, the ' Scuba ' fatalities are not all decompression fatalities, but nevertheless a great many of them do involve this particular aspect of hyperbaric environments.

SOME DANGERS OF A HYPERBARIC ENVIRONMENT

D. N. WALDER

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WITH every form of therapy, there are always associated risks. It is not often, however, that this risk applies to the medical personnel rather than to the patients, but this can be the case in hyperbaric oxygen therapy where medical attendants may be in a high pressure environment with the patient. It is essential to recognize these hazards and to ensure that the doctors are not laying themselves open to unreasonable danger. The particular danger to which I refer is that of decompression sickness. I like to think of decompression sickness as being of two types:

Type I. A pain in a joint with no constitutional upset, usually easily dealt with by recompression.

Type II. Which may take the form of a respiratory crisis known as the chokes with coughing and cyanosis, circulatory crisis similar to that seen following coronary thrombosis with retrosternal pain or neurological crisis with visual scotomata, paraesthesia or paraplegia. In Type II decompression sickness there is always an associated constitutional upset with peripheral vasoconstriction, nausea, sweating and hypotension and a feeling of being ill.

Now my experience of these conditions has been mainly in 'sandhogs,' that is men who dig under-river tunnels when compressed air has to be used to keep the water out. Some may believe that the two situations, that of compressed air workers in a tunnel and that of medical staff in a hyperbaric oxygen chamber are quite different. To some extent, this is true in that compressed air workers on shift work may be called upon to do eight-hour shifts in pressures up to 4 atmospheres absolute, whereas perhaps medical personnel supervising or operating on patients in a hyperbaric chamber may not be expected at the outside to spend longer than four hours at say 3 atmospheres absolute.

A look at the literature about tunnel workers, however, will show that even men who work for short periods in pressures up to 3 atmospheres absolute, *i.e.*, non-shift workers such as foremen, electricians, plumbers, engineers, tunnel surveyors, still have considerable trouble with decompression sickness. In Glasgow

where they have recently completed a tunnel under the Clyde, of 184 men who worked at less than 3 atmospheres absolute for less than four hours, approximately 1 in 5 men suffered from Type I bends at some time or another.

Although, as already stated, a Type I bend is easily treated by recompression and the man is in no danger of his life, the relationship between a bone pain and the subsequent development of caisson disease of bone is not yet clearly known.

Many factors have been suggested as influencing the susceptibility to decompression sickness, and one in which I have recently been interested is fatness. During some experiments in which pregnant guinea-pigs were being exposed to a hyperbaric environment for another purpose, it was noted that whether or not a particular guinea-pig foetus developed decompression sickness depended on its age, and this in turn was related to its fat content—young foetuses contain very little fat and are resistant to decompression sickness (Viotti & Walder, 1964).

As far as man is concerned, it has always been extremely difficult to determine fatness. Various attempts have been made to do this using height and weight, corporal S.G., linear density (wt./ht.), Ponderal index (wt./ht.^{3}) and the Sheldon index (based on body type) but none of these estimations has been clearly shown to be related to fatness nor indeed to susceptibility to decompression sickness. Recently, the Medical Research Council Decompression Sickness Panel has tried to relate skin-fold measurements (which are said to be closely related to a man's fatness) to susceptibility to decompression sickness, and preliminary results relating skin-fold measurements to the incidence of Type I bends look quite encouraging.

Now it may be thought that compressed air workers get bends because they are careless and do not take proper precautions, and that the simple way of avoiding decompression sickness is to use proper decompression procedures. But, except for isolated incidents, compressed air workers adhere closely to the regulations for decompression. The fact is that whether the British Decompression Sickness Tables or the American Naval Tables, or any other tables are used, it is recognized that for some individuals there is always a risk of decompression sickness developing in spite of proper decompression procedures.

A far more disturbing problem is that of Type II decompression sickness since men will rapidly die or suffer from permanent neurological damage unless it is promptly and efficiently treated. These men have air in their circulations. This type of catastrophe can occur after only short exposures to low pressures. (Six such cases

occurred during the construction of the Dartford Tunnel after exposures of less than four hours to pressures of less than 3 atmospheres absolute.) In some men this type of decompression sickness has been associated with the presence of lung cysts. Figure 1 is the post-decompression radiograph of a man who, after 45 minutes at less than $2\frac{1}{2}$ atmospheres absolute, fell unconscious after leaving the lock. There are two cysts to be seen, one partly concealed by the right border of the heart. Figure 2 shows the chest radiograph of a man who developed paralysis of the legs after an exposure to less than $2\frac{1}{2}$ atmospheres absolute. There is a cyst in the right lower zone, containing a fluid level. Figure 3 shows the post-mortem radiographic appearance of a man who, following exposure to 3 atmospheres absolute, collapsed and died. A large translucency in the right lower lobe can be seen. This was found to be a large pleural bleb.

As a result of these findings, a hypothesis was developed (Walder, 1963) that perhaps bronchial blocking was occurring during the period of compression, and that during decompression a volume of high air pressure was being locked in the chest to expand subsequently and burst its way into the circulation to give air embolism. At first guinea-pigs' bronchi were blocked at pressure with lead spheres, but this failed to produce either a cystic appearance of their lungs or air embolism on subsequent decompression. Attempts were then made to produce bronchiolar obstruction by spraying histamine into the chamber when the guinea-pigs were at pressure—subsequent decompression gave air embolism in 75 per cent. of guinea-pigs and in none of the controls.

An explanation was then sought for the failure to produce cysts in guinea-pigs' lungs. It appears that the structure of the guinea-pig's lung and, indeed, most laboratory animals is different from that of man. In man there are interalveolar septa which dip down into the alveolar areas of the lung from the visceral pleura (Fig. 4). At the bottom of each septum is a pulmonary vein radicle. It appears that pigs have a similar lung structure. Some experiments were therefore carried out in which a bronchus in pig lung was blocked and the pressure beyond the block raised at first to $1\frac{1}{8}$ atmospheres absolute. At this pressure air blebs arose on the visceral surface of the lung due to tearing along the lines of the interalveolar septa (Fig. 5). When the pressure beyond the block was raised to $1\frac{1}{4}$ atmosphere absolute, a spherical cyst developed (Fig. 6). On examining the interior of this cyst, it was found that at the bottom of the split septa were open pulmonary veins (Fig. 7) and it is therefore suggested that air trapped behind blocked bronchi at pressure may well form cyst-like blebs on