

# **Introductory Remarks to the Fourth Session**

E. E. P. Barnard

Phil. Trans. R. Soc. Lond. B 1984 304, 151-152

doi: 10.1098/rstb.1984.0016

**Email alerting service** 

Receive free email alerts when new articles cite this article - sign up in the box at the top right-hand corner of the article or click **here** 

To subscribe to Phil. Trans. R. Soc. Lond. B go to: http://rstb.royalsocietypublishing.org/subscriptions

Phil. Trans. R. Soc. Lond. B 304, 151–152 (1984) [ 151 ]
Printed in Great Britain

## Introductory remarks to the fourth session

### By E. E. P. BARNARD

Monckton House, Institute of Naval Medicine, Alverstoke, Gosport, Hampshire PO12 2DL, U.K.

The subjects of this session range from physics through neurophysiology to epidemiology but can, without using too high a level of abstraction, be related by the underlying presumption that the separation of dissolved gas from solution upon decompression leads to the formation of bubbles in the body and that they, in turn, produce pathological effects.

An increasingly large workforce was exposed to raised pressures during the second half of the nineteenth century particularly in building the foundations of a bridge by the method of Triger (1841), a method that used compressed air to exclude water from the workings. The disturbances caused by the application of pressure and, more seriously, following the reduction in pressure were described by many authors from France, Italy, Germany, Great Britain and the United States of America. So there was a considerable body of observational data and hypothesis available by 1900 and the first experimental studies of decompression had been done by Bert (1878). He established that gas was indeed produced on decompression; that, after exposure to compressed air, the main constituent of bubbles was nitrogen and he thought it probable that such bubbles caused the signs and symptoms previously recognized as being associated with decompression.

When, therefore, just after the turn of the century, J. S. Haldane, F.R.S., was asked to study the problems of diving for the Admiralty, there was a satisfactory theoretical basis for the causes of what we now call decompression sickness and an extensive literature of symptomatology. His contribution was to provide a method for divers for avoiding decompression sickness, partly based on observation and partly on theoretical considerations. The diving schedules that Haldane (1909) introduced virtually replaced the slow compression—slow decompression method that had been recommended by Littleton (1855) and, with minor modifications, J. S. Haldane's schedules are still in use.

Some of the more serious cases seen in diving before the introduction of the Haldane schedules prompted Boycott & Damant (1908) (Damant was a Naval Officer) to do experiments with goats to study the type of paralysis caused by decompression. Their work seemed to show that certain parts of the spinal cord were more susceptible to damage; indeed most of the damage occurred in the portion containing large numbers of myelinated nerve fibres, known as the white matter. They correlated this difference in susceptibility to damage between white and grey matter with differences in the blood supply and with the greater solubility of nitrogen in tissues that were fatty compared with tissues that were more watery. The occurrence of gas embolism was also well documented and some similarities with decompression sickness were noted in a number of papers published from 1873 to 1892.

One of the best known symptoms arising from decompression is 'bends', pain usually felt in or near the joints and starting within minutes or hours after leaving pressure. Although this was the earliest consequence of a sudden pressure drop to be reported, (Pol & Watelle 1854), the recognition of bone damage in caisson workers, now called avascular necrosis of bone, was not far behind; indeed the first large series of cases to be reported was by Bornstein & Plate

### E. E. P. BARNARD

(1911-1912), who studied men engaged in building the tunnel under the Elbe River at Hamburg.

It has long seemed tempting to link these two types of disorder, bends and bone necrosis, for one might characterize bends as a symptom without an obvious pathology and necrosis as a lesion without obvious preceding symptoms. When one has sufficient data to relate these disorders, however, it is found that there is independent assortment of necrosis and bends; each is related to decompression but there is no correlation between them.

In this brief introduction the examples chosen to illustrate the topics to be presented may seem rather remote, but their choice was deliberate and the intention was to suggest a parallel between then and now. In the second half of the nineteenth century the expansion in the engineering industry using new technology led to new medical problems, consisting initially of observational data because the experimental manipulation of pressure required special facilities, which were available in very few places. Today the expansion of the oil industry in the North Sea has led to new problems; but there are still very few laboratories where these can be studied. The need to work at great depths has been largely met by path-finding research, with men to explore the problems of great depths; unlike, for example, space research in which manned flights were preceded by animal exposures. This situation is at least partly the result of the low levels of investment in high pressure facilities. The principal advantage of using men is the decrease in complexity that this allows in the equipment, for men can feed themselves and help to maintain the atmosphere. On the contrary, long exposures at great depths with animal subjects would need more complex machinery and greater resources than have been available. These observations are not meant to detract from the achievements of the past ten years, but to point to the relative lack of basic research to support this area of knowledge. As an example of this weakness, during 75 years of uptake and elimination calculations of inert gas in biological systems, we still lack a generally accepted model for the uptake and distribution of gas in the body and a clear understanding of the effects of decompression on the elimination of gas.

At a point in the development of deep diving where one might expect to turn to academic research for the answers to fundamental questions, universities are also short of resources and may not recognize the value of pressure as a tool in types of research that are primarily basic research.

In summary, I believe that we have reached the stage at which we need, not more experience, but more experiments, and I would hope that this meeting will stimulate more interest in this field, both in University departments and in industry.

#### REFERENCES

- Bert, P. 1878 La pression barométrique; recherches de physiologie expérimentale. (1168 pages) Paris: G. Masson.
- Bornstein & Plate 1911–1912 Über chronische Gelenkveränderungen enstanden durch Presslufterkrankung. Fortschr. Rontgenstr. 18, 197–206.
- Boycott, A. E. & Damant, G. C. C. 1908 Some lesions of the spinal cord produced by experimental caisson disease. J. Path. Bact. 12, 507-515.
- Haldane, J. S. 1909 Report of a committee appointed by the Lords Commissioners of the Admiralty to consider and report upon the conditions of deep water diving. London: H.M.S.O.
- Littleton, T. 1855 Effects of submarine descent. Ass. Med. J. 3, 127-128.
- Pol, B. & Watelle, T. J.-J. 1854 Mémoire sur les effets de la compression de l'air appliquée au creusement des puits à houille. Annls Hyg. publ. Méd. lég. Ser. 2, 1, 241-279.
- Triger 1841 Memoire sur un appareil à l'air comprimé pour le percement des puits de mines et autres travaux, sous les eaux et dans les sables submergés. C.r. hebd. Acad. Séanc. Sci., Paris 13, 884-896.