

cave before trepidation at negotiating the entrance squeeze urged them to return. They later produced a beautiful map of their finds.

Three other divers, including one who had been there in the 60s, returned and, after a false start, made a 240 m dive up what is now known as the B tunnel using twin 50 cu ft cylinders. After some time, with a change of owners, the cave was closed. Subsequently, in the early 1990s the property was sold again and the present owners initially allowed access restricted to a few divers, but now all suitably qualified CDAA divers can access the site through a rigorously managed guide system. Since the early days the entrance chamber has been cleared, the squeeze widened by removal of some rock so that the cave could be entered using twin 90s or larger cylinders. The entrance is gated for security. The known extent of the cave now exceeds 8 km of passage.

David Doolette, PhD, is a physiologist attached to the Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, the University of Adelaide, North Terrace, Adelaide SA 5000 Australia. He has been a member (No 1310) of the Cave Divers Association of Australia since 1984. Phone +61-(0)8-8303-5157. Fax +61-(0)8-8303-3909.

E-mail ddoolett@medicine.adelaide.edu.au .

Philip Prust is a founding member (No 3) of the Cave Divers Association of Australia and has been cave diving since 1965.

JS HALDANE, JBS HALDANE, L HILL AND A SIEBE: A BRIEF RESUME OF THEIR LIVES

Chris Acott

Key Words

Decompression illness, equipment, history, general interest, physiology.

Introduction

In the 19th century there were numerous attempts to explain the symptoms of decompression sickness which ignored Boyle's and Bert's bubble theories. These explanations included: spinal cord damage caused by cold or exhaustion; frictional tissue electricity caused by compression-or decompression-induced organ and vascular congestion. Allbutt's "System of Medicine", published in 1900, reported that decompression sickness "was attributed to the mechanical effect of pressure on the circulation". However, despite these controversial views,

all the salient clinical features of decompression sickness were described between 1870-1910.¹⁻³

Haldane, Hill and Siebe are names synonymous with the development of diving and diving medicine. This paper is a brief outline of the lives of the J S Haldane, J B S Haldane, Leonard Hill and Augustus Siebe.

John Scott Haldane (1860-1936)

J S Haldane was born in Edinburgh on the 3rd May 1860 and died in Oxford during the night of March 14/15th 1936. His family (whose motto was "Suffer") was affluent and influential. He studied at the University of Jena after graduating from the University of Edinburgh Medical School.⁴⁻⁷

He taught physiology at the Universities of Dundee and Oxford and was noted mainly for his work on respiratory physiology. His research demonstrated great intellectual curiosity and often involved self experimentation. He was assisted by his son (JBS Haldane) in much of his work. He developed several procedures and apparatus for the physiological study of breathing and gas exchange; these included the haemoglobinometer and the Haldane-Henderson Gas Analysis apparatus.⁷

In 1893, following self experimentation, he concluded that respiration was regulated by carbon dioxide. With a colleague he remained in an air tight box (named "the coffin") for up to 8 hours rebreathing the atmosphere and noting their reactions. "At 7 percent (oxygen concentration in air) there is usually distinct panting, accompanied by palpitations, and the face becomes a leaden blue colour. At the same time the mind becomes confused." This data were not published until 1905.⁹ In 1914 the "Haldane Effect" was published. His book *Respiration* was first published in 1927.⁸

In 1895 he investigated a serious accident in which 5 London sewer workers died. He discovered "sewer gas" (hydrogen sulphide) by descending into the shaft to take samples of the air and sewerage. He recommended aerating the sewers and attaching safety ropes to all sewerage workers.⁷

In 1911 he led an expedition to Pikes Peak, Colorado, USA, to study the physiological effects of low barometric pressure.^{5,6}

In 1913 he became a director of a mining research laboratory. He found that "fire damp" (a mixture of CO, CO₂ and N₂) was far more lethal than the effects of an underground explosion. As a result of his firedamp experiments he recommended: "In view of the difficulty of recognising by ordinary means the presence of poisonous amounts of this gas (carbon monoxide), I propose the plan

of making use of a small warm-blooded animal [a mouse or very small bird] as an indicator of carbon monoxide".⁷

He was also a philosopher.⁷ He tried to clarify the philosophical basis for biology and its relationship to physics and chemistry. His publications included *Materialism* (1932) and *The Philosophy of a Biologist* (1935).

Haldane also conducted extensive research into the physiological effects of hyperthermia. He became known as "the father of the salt tablet" because he recommended salt replacement during excessive sweating.⁵

In diving he is best known for his "staged" decompression method. In 1908 with co-workers Boycott and Damant Haldane published his decompression tables. "In the ordinary diving table, therefore, the stay on the bottom is so limited that the diver can be decompressed safely in half an hour".⁹ These tables decreased the incidence of decompression sickness and are still used today, in a modified form, either as a decompression table or in a diving computer. Haldane had so much faith in these tables that he stated "compressed-air illness has now practically disappeared except in isolated cases where from one cause or another the regulations have not been carried out".⁹ He also suggested that oxygen should be used to shorten decompression provided the pressure was kept less than 2 bar (atmospheres absolute) because of the fear of oxygen toxicity.¹

However, Haldane made little contribution to the therapy of the decompression sickness although he recognised that recompression was the treatment of choice but stated "the trouble, however, about the use of recompression chambers is that it is often very difficult to get the patient out without the symptoms recurring".⁸⁻¹⁰

DECOMPRESSION EXPERIMENTS

Heller, Mager and von Schrotter believed that a uniform decompression at a rate of one atmosphere per 20 minutes was safe.^{1,10} Uniform decompression was also advocated by L Hill and M Greenwood (they had decompressed themselves without any serious symptoms from 5-6 atmospheres after short exposures).³ However, Haldane had doubts about the safety and efficacy of uniform decompression practice.⁹ At that time the Royal Naval decompression rate of 5 fsw/minute (1 atmosphere/6.5 minutes) had an unacceptable high rate of decompression sickness.⁹ Haldane was commissioned by the Admiralty to develop safe decompression procedures. (Haldane's brother, Richard Burdon Haldane, was the Secretary of State for War at this time and helped Haldane obtain this contract; L Hill had also applied for the same contract but was rejected.)¹¹

Haldane's experiments were conducted at the Lister Institute in a recompression chamber donated by Dr Ludwig Mond FRCS. Financial support was from both the Admiralty and private contributions.⁹ The diving company, Siebe Gorman, did not contribute financially to Haldane's experiments but did support the experimental work of L Hill and M Greenwood.^{12,13}

Haldane made the following assumptions:⁹

- 1 for bubbles to form, the pressure of gas in the tissues must exceed the external pressure;
- 2 that body tissues will hold gas in a supersaturated state unless a certain limit is reached;
- 3 that any decompression is free from risk only if the degree of supersaturation "can be borne with safety";
- 4 that tissue perfusion was the limiting factor in inert gas uptake.

His decompression experiments examined three different variables:⁹

- 1 the depth and pressure exposure;
- 2 the exposure duration;
- 3 the mode and decompression rate.

Initially, a few experiments were conducted on rabbits, guinea pigs, rats and mice but it was difficult to detect symptoms in these smaller animals and so the goat was chosen as the experimental model "because they were the largest animals which could be conveniently dealt with" and "those who are familiar with them can detect slight abnormalities with a fair degree of certainty". The dog was rejected because they had noted that Heller et al. had previously used them to produce "safe" decompression profiles that had failed in humans.⁹

Goats were excluded from the experiments if they were ill. Only 5-8 goats were used per experiment. The chamber was not ventilated because they thought that CO₂ had a minimal effect on the susceptibility to decompression sickness. The chamber temperature was not controlled and no allowance was made for any variation in atmospheric pressure. Large pressure variations were used to produce minor to severe symptoms. The compression time of 6 minutes was neglected in short exposures but included in longer, deeper exposures.⁹

THE 2:1 STAGING RATIO

At the time of the experiments, Haldane knew from Naval diving data that decompression from 2 bar produced no symptoms irrespective of the time of exposure, however, decompression from 2.25 bar produced the "occasional slight case". Examination of caisson workers' case histories showed that there were no deaths recorded in decompressions from 2.6 bar and previous experiments showed that a rapid decompression from 2.36 bar only produced "slight symptoms" in 1 of 22 goats while

decompression from 2.7 bar produced symptoms in 2 of 23 goats. An assumption was therefore made that decompression from 2.25 bar would be without risk. Goats could also be decompressed from 6 bar to 2.6 bar without producing any symptoms. However, if the goats were decompressed from 4.4 bar to 1 bar, only 20% escaped symptoms (in both of these decompressions the pressure difference is 3.4 bar). Hence the assumption that halving the pressure would not produce any symptoms.⁹

TISSUE PERFUSION HALF LIVES

Haldane used a “perfusion” mathematical model of gas uptake. These “perfusion half lives” were calculated from data available at that time⁹ and were not arbitrarily derived as has been reported. These data were from: Moir’s case histories of decompression sickness in caisson workers;¹ their own animal experiments;⁹ self experimentation by Hill and Greenwood³ and a mathematical model considering the body being as a uniform tissue.⁹

SYMPTOMS

The symptoms in goats were “protean in character”.

- 1 “Bends” : the bends, or limb pain, was the commonest symptom. The affected limb (commonly the foreleg) was raised.
- 2 “Pain”: pain was detected by “urgent bleating and continual restlessness” with the goat often gnawing at the affected area “such as the testicles”.
- 3 “Temporary paralysis”: The paralysis “may be of two kinds”. Both were noted about 15 minutes after decompression and had improved within 30 minutes, the animal being “quite well” the next day. Some goats showed general weakness, dyspnoea, foot drop and dragged their hind legs, while others were noted to be well but had foot drop or “palsy in one or more hind or fore limbs”.
- 4 “Permanent paralysis”: The hind legs were noted to be paralysed immediately post decompression. Any spontaneous improvement was followed by a permanent relapse. Urinary retention and an acute gut distension were also noted.
- 5 “Obviously ill”: The goats were noted to be apathetic and ill. They refused “to move or to be tempted with corn (of which goats are inordinately fond)”. Some were noted to be “castrated, of male habit”, presumably they were impotent.
- 6 “Dyspnoea”: Dyspnoea was a sinister symptom usually occurring just before the animal died.
- 7 “Death”.

This data showed that goats had an individual variability and susceptibility to decompression sickness.⁹

John Burdon Sanderson Haldane (1892-1964)

JBS Haldane, born at Oxford on 5 November, 1892, was considered a genius. He was educated at Oxford Preparatory School, Eton and New College, Oxford.¹¹ He went to Oxford to study mathematics and biology, but graduated in classics and philosophy.

From the age of 3 he assisted his father during his research by providing blood samples and recording the experimental data. In 1901 he co-authored, with his father, a paper on how haemoglobin combines with oxygen. In this paper he contributed the complex mathematical analyses.¹¹

While he was at Eton he worked closely with his father in the First Royal Navy Deep Diving Committee on the prevention of decompression sickness.^{11,13}

He assisted, and was used by, his father in his research into mine “fire damp”. He described one episode in a north Staffordshire mine: “.my father told me to stand up and recite Mark Antony’s speech from Shakespeare’s Julius Caesar.....and somewhere about “the noble Brutus” my legs gave way and I collapsed on the floor, where, of course, the air was alright. In this way, I learnt that firedamp is lighter than air”.¹¹

During World War 1 he served with the Black Watch in France and Iraq and was wounded twice.¹¹

He became the Reader in Biochemistry at Cambridge University (1922-32) and the Professor of Genetics at London University (1933-37). In 1930 he became the Fullerian Professor of Physiology at the Royal Institution.

His interest in genetics was stimulated by a lecture he attended on Mendel’s genetic principles in 1901. In 1912 he published his first paper on genetic linkage. He later published on the genetics of haemophilia and colour blindness. His book *The Causes of Evolution* was a landmark in population genetics. Among many scientific firsts he investigated the biochemistry of gene action, the genetic control of enzyme reactions, calculated mutation rates for genes, created linkage maps for human chromosomes and analysed human pedigrees to understand different modes of inheritance. While at Cambridge (1922-33) he formulated a mathematical model of natural selection.^{5,6}

He was a member of the Admiralty’s Second Deep Diving Committee in the 1930s. During World War 2 he assisted Kenneth Donald with some of his experimental work on oxygen toxicity in divers.¹⁴

A committed Marxist, he was chairman of the editorial board of the communist *Daily Worker* between 1940 and 1949. In 1956 he rejected the Marxist ideology

because of the Lysenko controversy and “Soviet interference in science”.⁴

In protest at Britain’s involvement in the Suez crisis he emigrated to India in 1957. He adopted Indian nationality and became Professor of the Indian Statistical Institute in Calcutta. However he resigned in 1961 because of personal differences with his colleagues. He became the Head of the Orissa State Genetics and Biochemistry Laboratory in 1962. He died in Bhubaneswar, India on December 1st, 1964.⁷

Leonard Hill (1866-1952)

Little has been written about Leonard Hill and his contribution to our understanding of decompression sickness and diving medicine. He advocated a linear or uniform decompression profile (this style of decompression is used in the saturation diving) as opposed to Haldane’s “staged” method. He was closely associated with the Siebe Gorman Diving Company which financed the majority of his experimental work.¹³

His book, *Caisson Sickness and the Physiology of Work in Compressed Air* was published in 1912 and is considered by some to be a classical work.¹⁰

His research included decompression sickness, oxygen toxicity and the effects of carbon dioxide in diving. He also experimented with nitrox and heliox mixtures.^{3,10,12,13} Much of his early experimental work involved self experimentation. For example, while trying to define the saturation rate of fast tissues with nitrogen, Hill and a colleague, W Greenwood, were pressurised to provide various samples of urine which were measured for nitrogen content.³ These data were used by JS Haldane to determine one of the tissue half lives for his staged decompression method.⁹ He also designed a unique experiment in 1900 that not only showed that bubbles caused decompression sickness but that recompression was the treatment of choice. Although recompression treatment had been advocated earlier by some clinicians but it was not universally accepted until 1924.¹⁵ Hill used a frog. “Thus, after keeping a frog in highly compressed air for some time and then rapidly decompressing the animal, bubbles of gas were seen to appear in the capillaries and stop the circulation. On recompression, the bubbles were observed to shrink until the circulation became re-established. The cause and cure of compressed-air sickness could thus be projected as a demonstration on the screen”.¹¹

In the 1930s he was a chairman of the Royal Navy’s 2nd Deep Diving Committee.^{12,13}

Leonard Hill was responsible for the original medical standards for deep diving. He stated that the original format for a diving medical “was similar to that employed

by the Air Ministry,” and “that the diver should be possessed of a very stable mentality,” and “that it is necessary for the deep-sea diver to be more fit than a diver engaged in shallow water.” His medical standards were refined over the following years.¹⁵

He was also a medical physiologist who noted the increased difference in systolic blood pressure between the legs and arms in aortic coarctation which is called “Hill’s sign”.¹²

Christian Augustus Siebe (1788-1872)

Siebe was born in Saxony, Southern Prussia. At a young age his family moved to Berlin where he was educated. He was a brilliant engineer and machinist who showed great skill in modelling, chasing and watch making. In 1802 and 1803 he was awarded medals for his workmanship.

He joined the Prussian army in 1812 and served as an Artillery officer during the Napoleonic Wars. In 1813 he was wounded during the Battle of Leipzig (also called the Battle of Nations). Following his medical discharge from the army he moved to Kiel, then in Denmark, where he obtained employment as a watchmaker.⁵

In 1816 he emigrated to Great Britain and obtained work as a watchmaker with Garrards of London (who were at that time Jewellers to the Crown). Although he lived in London for majority of his life he only applied for British citizenship in 1862. This was granted on the 12 December, 1862.

In 1819 he became the manager of an engineering firm based at 145 High Holborn, London. In that year he was also married. He had 3 sons and a daughter.

In 1828 he produced a successful rotary water pump that gained him public notice. He also designed a breech loading rifle and a weighing machine. In 1851 he manufactured a Galvanic battery.

In 1830 the Deane brothers brought their design for an improved diving apparatus to his shop in Denmark Street and asked him manufacture it. George Edwards, another engineer, also modified the Deane diving equipment and brought his ideas to Siebe. In May 1837 Siebe tried to establish himself as an independent supplier of diving equipment and approached the British Admiralty. In 1840 Siebe adopted Edward’s modified version and released the diving suit as his own.

During the salvage from, and destruction of, the ROYAL GEORGE wreck in Spithead, Siebe’s closed diving rig became the preferred suit because the helmet could not flood when the diver leaned forward. The operation

also required the highest standard of air pump and Siebe's pump was superior, hence his diving rig became the preferred one for the Royal Navy. From here the saga of the origins of the closed diving dress started. Siebe's son, William Henry Siebe, and son-in-law, William Augustus Gorman, and later Sir Robert Davis, a chairman of Siebe Gorman Company, were probably responsible for the propagation of the story that A Siebe was the first to design the "standard" diving dress. For greater detail on the origin of the "standard" diving dress the reader is referred to Bevan's excellent historical book *The Infernal Diver*,¹⁶ from which these details have been taken.

Augustus Siebe died on the 15 April, 1872 from "chronic bronchitis". His son, Henry, and son-in-law, William A Gorman, had taken managerial control of Siebe's business in 1868 which became known, in 1870, as the Siebe Gorman Company.^{12,16}

References

- 1 Behnke A. Some early studies of decompression. In *The Physiology and Medicine of Diving and Compressed Air Work*. Bennett PB and Elliott DH. Eds. London: Bailliere Tindall Cassell, 1969; 226-251
- 2 Bert P. *Barometric pressure* (1878). Translation by Hitchcock MA and Hitchcock FA. Columbus, Ohio: College Book Company, 1943. Republished Bethesda, Maryland: Undersea Medical Society, 1978
- 3 Hill L and Greenwood M. The influence of increased barometric pressure on Man 1. *Proceedings of the Royal Society of London* 1906; LXXVII (June): 442-453
- 4 *Chambers Biographical Dictionary*. Magnusson M and Goring R. Eds. New York: Chambers, 1990
- 5 *Encyclopaedia Britannica*. 1996
- 6 *Microsoft Encarta 96 Encyclopedia*. Microsoft Co and Funk and Wagnalls Co, 1996
- 7 Duin N and Sutcliffe J. *A History of Medicine. From Prehistory to the Year 2020*. London: Simon and Schuster, 1992
- 8 Haldane JS. *Respiration*. New Haven: Yale Univ Press, 1927
- 9 Boycott AE, Damant JCC and Haldane JS. Prevention of compressed air illness. *J Hygiene* 1908; 8: 342-425
- 10 Hill L. *Caisson Sickness and the Physiology of Work in Compressed Air*. London: Edward Arnold, 1912
- 11 *Everyman's Encyclopedia Vol 7*. Ridgway A and Holmyard E. Eds. London: J M Dent, 1931-2
- 12 Davis RH. *Deep diving and submarine operations. 7th Ed*. London: St Catherine Press, 1962
- 13 Davis RH. *Deep Diving and Submarine Operations, Part 1 & 2. 9th Ed*. Gwent : Siebe Gorman & Co Ltd, 1995
- 14 Donald K. *Oxygen and the Diver*. Hanley Swan, Worcs: The SPA Ltd, 1992; 194
- 15 Thalmann ED. Principles of US Navy recompression treatments for decompression sickness. In *Diving Accident Management. 41st UHMS Workshop*. Bennett PB and Moon RE. Eds. Bethesda, Maryland: UHMS, 1990; 194-221.
- 16 Bevan J. *The Infernal Diver*. London: Submex, 1996

Dr C J Acott, FANZCA, DipDHM, a Past President of SPUMS, is a Senior Specialist in the Hyperbaric Medicine Unit, Department Anaesthesia and Intensive Care, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia 5000. Phone +61-8-8222-5116. Fax +61-8-8232-4207.

BILATERAL DEAFNESS ASSOCIATED WITH DIVING

Alfred Buchner and Matthias Hepe

Key Words

Case report, ENT.

Case report

A 61 year old male German diver went to Egypt and the Red Sea for a live-aboard diving holiday. He had 30 years experience and more than 500 dives in the former German Democratic Republic. Before he went he had medical examinations for fitness to dive and was considered "fit to dive without any restrictions".

During the first day on board he performed two relaxing dives without any strenuous events. The profiles were 10 to 15 m (30 to 45 ft) for about 30 minutes. He did not use a diving computer and there were no records available.

On day 2 he performed three dives. The first dive was 30 minutes at 14 m (42 ft). After a surface interval of three hours the second dive was 30 minutes at 20 m (60 ft). The surface interval was 3 hours 30 minutes before the third dive, which was to 35 m (105 ft) for 20 minutes. Dives 1 and 2 were in a group of divers with a divemaster while dive 3 was a camera dive with a buddy. He did regular safety stops on all dives. The last dive was "not very much controlled" according to the diver's report. He had no equalisation problems at all and did not do any Valsalva manoeuvres during the dives.