decompression tables (1908) that were generated as a result, provided the desperately needed turning point in this disaster-ridden profession.

References

1 Francesco da Marchi. *Della Architettura Militare*. 1st edition. Brescia. 1599
2 Culpepper N. Culpepper's Complete Herbal. W Fousham & Co Ltd
3 Maule J. Notes concerning diving and working under water. Royal Society. 1663
4 Bartholin T. On Divers. *Acta Medica et Philisophica Hafniensia*. 1677
5 Halley Sir Edmond, Evance Sir Steven, Tyssen F and Holland J. *Engine for conveying air into a diving vessel. Patent No 279*. 1691 Oct 7
6 Halley E. The Art of Living Underwater or a Discourse concerning the Means of Furnishing air at the Bottom of the Sea, in any Ordinary Depths. *Philosophical Transactions Royal Society* 1716; 29 (349. Jul-Sep): 492-499
7 Halley E. *Correspondence and Papers of Edmond Halley*. London. 1932
8 Treiwald M. *Philosophical Transactions Royal Society*. 1736; 39 (444 Nov-Dec)
9 Desaguliers JT. *A Course of Experimental Philosophy. 3rd Edition*. 1763; 2: 214-215
10 Desaguliers JT. *A Course of Experimental Philosophy. 3rd Edition*. 1763; 2: 217
11 Desaguliers JT. *A Course of Experimental Philosophy. 3rd Edition*. 1763; 2: 218
12 *The Morning Post and Daily Advertiser*. 10th June 1783
13 Johnson RF. *The Royal George*. Charles Knight & Co Ltd. 1971. SBN 85314 103 7
14 *Document ADM/A/2790. National Maritime Museum*
15 *Philosophical Magazine*. 1799; 3: 90, 213, 300 and 418
16 Elliott J. Submarine Adventures. *Mechanics Magazine* 1832; 473 (1 September): 364-365
17 Foley. *The Engineer*. 22 January 1864
18 Esquiros A. *English Seamen and Divers*. Chapman and Hill. 1868; 278
19 *Annual Register*. January 1836; 78: 1
20 *Pasley Papers. Additional Manuscript 41988. 1838; 28*. British Library
21 *Pasley Papers. Additional Manuscripts 41961-41995*. British Library
22 *Hampshire Telegraph.* 30 August 1841
23 *Aide-Memoire to The Military Sciences*. 1846; 1: 360 Royal Engineers library
24 Buckland F. *The Logbook of a Fisherman and Zoologist*. 1875; 138
25 *Document ADM 1/5528. Admiralty Letters In. various. 22 June 1843.* Public Records Office
26 Green JB. *Diving with and without Armour*. Buffalo. 1856; 42
27 Davis RH. *A Few Recollections of an Old Lambeth Factory*. Siebe Gorman & Co Ltd. 1959; 33
28 Liddell J. On the Health of Divers etc. *Med Chi Rev New Series* 1842; 37: 633-636
29 *The Times*. 14 October 1854
30 *The Comet*. 19 October 1854
31 Bevan J. The Infernal Diver. Submex Ltd. 1992; 251

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OXYGEN TOXICITY
A BRIEF HISTORY OF OXYGEN IN DIVING

Chris Acott

Key Words

History, hyperbaric research, incidents, injuries, oxygen, medical conditions and problems, unconscious.

Introduction

The Earth was probably formed about 4,600 million years ago by the gravitational coalescence of cold material. Initially there was a tenuous atmosphere of hydrogen and helium which was lost because of a weak gravitational field. The secondary atmosphere was created by the thermal and radioactive decay of various Earth's constituents. Ammonia dissociated into nitrogen and hydrogen and water vapour into hydrogen and oxygen. However, by far the greatest source of oxygen was, and still is, from photosynthesis. There is some evidence to suggest that the atmospheric concentration of oxygen cannot have changed for the past 345 million years.[1]

Discovery of oxygen

Oxygen was not discovered until the 18th century, although its presence in air, as a gas which supported combustion, was postulated by Boyle and Hooke (1666), Lower (1669), Mayow (1673) and demonstrated by Joseph Black (1728-1799) in 1754. He showed that when a

substance burned it gained weight.[2] Boyle and Hooke demonstrated that respiration kept animals alive. Before this scientists believed Aristotle's theory that respiration cooled the blood.

In 1772 Carl Wilhelm Scheele (a Swedish chemist, 1742-1786) showed that air was a mixture of two gases, one which he called "fire air" because it supported combustion and the other "foul air" because it did not.[2]

Independently, in 1774, Joseph Priestley (1733-1804) also discovered oxygen by heating red mercuric oxide. He noted that a candle burned "with a remarkably vigorous flame". Priestley was unsure of what he had discovered but because he believed in the "phlogiston theory" he called it "dephlogisticated air".

The phlogiston theory had been devised by Johann Becher in 1669. It stated that during combustion or respiration a substance was liberated. This substance was called phlogiston (from Greek, meaning "burned") by Georg Ernest Stahl about 1700.[3]

In 1774 Priestley discussed his discovery with Antoine Lavoisier (1743-1794) who immediately appreciated its significance. In 1781 Lavoisier named dephlogisticated air "oxygen" (or acid producer) and in 1786 he refuted the phlogiston theory, despite opposition from Priestley and others.

Thomas Beddoe's publication *Considerations on the Medical use and on the production of Factitious Air* followed Lavoisier's work and was the first publication to postulate oxygen's therapeutic role.[4]

Central Nervous System oxygen toxicity

Soon after the discovery of oxygen, and the introduction of the combustion theory of respiration, it was suggested that oxygen might be toxic as an increased oxygen concentration would induce an increased respiratory exchange thus accelerating pulmonary circulation and congesting the lungs. In 1789 Lavoisier and Seguin challenged this view by stating there would not be an increase in the body's oxidative processes.[5] However, it was not until 1849 that the animal data of Regnault and Reiset showed that there was no evidence of an increase in oxidative processes due to an increased concentration of oxygen.[6]

In 1878 Paul Bert first described the central nervous system toxic effects of hyperbaric oxygen in his classic work *La Pression Barometrique*. This book was translated into English in 1943 and reprinted in 1973 by the Undersea Medical Society.[7] CNS oxygen toxicity is sometimes referred to as the "Paul Bert effect". He showed that oxygen was toxic to all living matter, insects, arachnids, myriapods, molluscs, earthworms, fungi, germinating seeds, birds and other animals. Among other experiments he transfused normal animals with blood from those that had convulsed while breathing oxygen. These animals did not convulse and so he concluded that the toxic properties of oxygen were direct effects. He described this toxicity as "A profound modification in the metabolism of tissues".[7]

He attempted to determine the most favourable oxygen partial pressure but concluded that *an increase in oxygen tension above its normal value in ordinary air seemed to bring no advantage,.....When any difference is noticeable it is in the favour of normal air*.[7] In many of his experiments, particularly on larger animals, the tension of carbon dioxide was not controlled.

Since his experiments, many other investigators, Lorrain Smith (1899),[8] Hill and MacLeod (1903),[9] Barach (1926),[10] Behnke (1934)[11] and Bean and Rottschafer (1939)[12] have shown the toxic properties of an increased partial pressure of oxygen in various species. Bean and Rottschafer exposed animals to hyperbaric oxygen until they convulsed and then continued the exposure. The animals repeatedly convulsed until they died.[12] This is referred to as the "Bean Effect".

The first human exposure to hyperbaric oxygen was recorded in 1910 by Bornstein. Two men breathed 2.8 bar oxygen for 30 minutes while Bornstein breathed it for 48 minutes, none showed any ill effects.[13] However, in 1912, Bornstein breathed 2.8 bar oxygen for 51 minutes when he developed cramps in both hands and legs, signs of oxygen toxicity.[14]

Pulmonary or whole body oxygen toxicity

Pulmonary oxygen toxicity was first described by Lorrain Smith in 1899 and is often called the "Lorrain Smith effect" or chronic oxygen toxicity.[8] He had noted the CNS effects of hyperbaric oxygen and postulated that other tissues, especially the lungs, would be affected by oxygen. His experiments on mice and birds showed that 0.42 bar had no effect but 0.74 bar of oxygen was a pulmonary irritant after a 4 day exposure. The liver, spleen and kidneys were also affected by prolonged oxygen exposures. There were intra- and inter-species variations in susceptibility to pulmonary oxygen toxicity. He showed that intermittent exposure allowed recovery, so delaying the development of toxicity.[8]

In 1926 Barach, using rabbits, investigated the use of normobaric oxygen for the treatment of pulmonary tuberculosis. He concluded that "the highest concentration of oxygen compatible with safety should be regarded as 60 per cent."[10]

CNS oxygen toxicity and diving

Oxygen's use in diving is limited by its toxicity. Fear of its acute toxic effects delayed its use in decompression and recompression treatment.[15]

With the development of the Davis Decompression Chamber in 1929 and the formation of the Admiralty Committee on Deep Diving Unit in 1930, the Royal Navy (RN) began experimenting with oxygen decompression from 60 fsw (18 m) for air dives to 300 fsw (90 m).[4] Both animal and human trials were conducted between 1930 and 1933.[16,17] Data from these human oxygen breathing trials are shown in Table 1.[18]

This research stimulated Behnke to begin his research into the human tolerance to oxygen in 1934.[19] Behnke believed that the toxic effects of oxygen had delayed its use in the treatment of decompression illness, although earlier workers, Zuntz[20] and Hill[9,21] had advocated its use.

Table 2 lists Behnke's oxygen exposure limits established by his experiments on oxygen toxicity between 1934-1936.[18] These time limits were used both by Behnke and Shaw[15] and by Yarborough and Behnke[22] in the development of air/oxygen treatment tables for decompression sickness and also by Van Der Aue, ten years later, in USN Treatment Tables 1-4.[23]

In 1941 Case and Haldane reported 8 exposures to very high pressures of oxygen. Two were to 7 bar for 4 minutes. Both resulted in oxygen toxicity, relieved by breathing air. The second exposure resulted in a convulsion, five minutes after coming off oxygen, during decompression. The other six exposures were to 6.15 bar. One subject exposed for 4 minutes had no symptoms. The other subjects were exposed for 5 minutes. One had no symptoms while the other five developed symptoms. None of these subjects convulsed.[21,24]

In 1942 JBS Haldane, Derrick and Donald undertook an exposure to 10 bar oxygen for 25-30 seconds with no signs of toxicity .[21]

In 1942 Donald was appointed as the Medical Officer to what became the Admiralty Experimental Diving Unit, based at the Siebe Gorman factory near London. He was to develop oxygen tolerance limits for Royal Navy (RN) divers to enable them to use oxygen rebreathing sets in combat. This research was a result of the successful attack by Italian divers, riding on torpedoes and using oxygen rebreathing sets, on the two battleships, HMS QUEEN ELIZABETH and HMS VALIANT, inside Alexandria Harbour, Egypt, in 1941.[25]

Between 1942 and 1945 Donald conducted 2,000 experimental dives using RN volunteers. Each experimental diver was monitored closely by 2 attendants.

TABLE 1

RN DEEP DIVING UNIT DRY CHAMBER OXYGEN TOLERANCE TRIALS 1930-33

Pressure	Subjects	Exposure	Symptoms
2 bar	12	60 minutes	None
3 bar	4	30 minutes	None
4 bar	1	13 minutes	Twitching face. Stopped with air breathing
4 bar	1	16 minutes	Twitching lips. Taken off O_2 then convulsed while breathing air

Table constructed from[18]

TABLE 2

DRY CHAMBER OXYGEN EXPOSURE LIMITS (BEHNKE et al.)

Pressure	Subjects	Time Limit
1 bar	10	4 hours
2 bar	3	3 hours
3 bar	4	3 hours
4 bar	2	<45 minutes

Table constructed from[18]

Working and non-working dives were conducted in both "dry" and "wet" environments and at various depths (25-100 fsw). These experiments produced the most extensive records of human acute (CNS) toxicity but war time conditions prevented their publication in full until 1992.[26]

Presenting symptoms in non-working dives (underwater) described by Donald are listed in Table 3.[27] When convulsions occurred they lasted for, on average, 2 minutes and were similar to Grand Mal seizures. Often there was no warning. The post-ictal stage lasted 15 minutes. There were no permanent residua. Lip twitching often progressed to a convulsion. Once this relationship was established the experiments were stopped when a diver developed lip twitching. One diver was accidentally kept on oxygen during and after his convulsion, when he restarted breathing he commenced fitting again within 30 seconds.[28]

The safe depth limit for oxygen-diving (no convulsions) was found to be 25 fsw (7.6 m), the shallowest depth tested, whether the diver was resting or active.[29] There was no time limit placed on this exposure although the longest exposures had only been for 2 hours. The exposure limit of the absorbers used by the RN at that

TABLE 3

SYMPTOMS AND SIGNS OF OXYGEN TOXICITY UNDERWATER IN 388 NON-WORKING DIVERS

Symptoms	Number	%
Lip Twitching	303	61.0%
Convulsions	46	9.3%
Vertigo	44	8.9%
Nausea	43	8.6%
Respiratory change	19	3.8%
Other twitching	16	3.2%
Drowsiness etc	16	3.2%
Visual disturbance	5	1.0%
Hallucinations	3	0.6%
Paraesthesia	2	0.4%
Total	**497**	**100.0%**

Table constructed from [27]

time was 90 minutes.[30] The results of Donald's experiments are listed in Table 4.

Donald's conclusions are best expressed in his own words:[28]

The most important finding in this large series of exposures was that the symptoms of oxygen poisoning vary enormously in different people and in the same person during different exposures. No list of warning signs or symptoms can be given that would ensure a safe and timely cessation to the exposure.

..variability of the group is independent of the depth."

..it is emphasised that no signs or symptoms can be given that would ensure a timely cessation of oxygen breathing in all cases. The variation of symptoms even in the same individual, and at times their complete absence before convulsions, constitute a grave menace to the independent oxygen-diver. The only possible conclusion is that such tensions of oxygen should be scrupulously avoided.

The variation of tolerance between individuals, the variation of tolerance of each individual, the impairment of tolerance with work and underwater, all make diving on pure oxygen below 25 ft of sea water a hazardous gamble.

In 1947 Dr Edgar End began using 3 bar oxygen in the treatment of decompression sickness in Illinois compressed air workers. The treatment tables were short, being 90-120 minutes long, and were apparently very successful. His results have not been published.[31]

TABLE 4

SUMMARY OF THE RESULTS FROM DONALD'S EXPERIMENTS

1	Individual variation of tolerance.
2	Susceptibility increased with depth, in a "wet" environment (this difference decreased with depth) and with exercise.
3	Susceptibility varied with water temperature. There was an increased susceptibility in water less than 9°C and above 31°C.
4	Convulsions can occur at anytime, frequently without warning.
5	The 5 epileptics tested did not show any increase in susceptibility.
6	The depth limit for oxygen diving should be 25 fsw (7.6 m) as no convulsions occurred at this depth. No time limit was suggested but the longest exposure was 2 hours.

In 1954 Lanphier developed the USN oxygen limits.[32] These were for exposures of between 25 (7.6 m) and 40 fsw (12 m). He ignored Donald's data and the limits were based on some experimental data, "educated guessing", "previous experience" and an arbitrary addition of 25% which became the "working limit".[33] These limits were subsequently revised in 1959 for the use with nitrox mixtures. Lanphier believed that toxicity was seen after a briefer exposure to a nitrox mixture than with an equivalent pressure of pure oxygen due to a change in gas density.[34,35] This was not Donald's opinion.[36] In the 1970s the USN Experimental Diving Unit had a time limit of 4 hours at 20 fsw (7.6 m).

In 1977 Hendricks et al. showed pulmonary tolerance of an exposure to 2 bar oxygen could be extended if air breaks were given.[37]

In 1993 Harabin and Survanshi analysed all the available data and showed that for all symptoms of CNS toxicity 1.3 bar oxygen was the threshold, with the threshold for convulsions at 1.7 bar.[38]

Summary

A brief summary of the important dates in the use of oxygen in diving and the studies on oxygen toxicity.

1669 The Phlogiston Theory developed by JJ Becher.

1733 Joseph Priestley born in Leeds, UK.

1742 Carl Wilhelm Scheele born in Sweden.

1743 A Lavoisier born in Paris.

1772 Scheele discovered oxygen.

1774 Priestley discovered oxygen which he named "dephlogisticated air". Lavoisier met Priestley in Paris and subsequently repeated Priestley's experiments.

1777 Scheele published his research.

1781 Lavoisier named "dephlogisticated air" oxygen.

1786 Scheele died.

1789 Lavoisier and Sequin demonstrated the pulmonary effects of prolonged use of normobaric oxygen.

1794 Lavoisier guillotined.

1796 Beddes and Watt published a paper on the medical uses of oxygen.

1804 Priestley died in the USA.

1878 P Bert published "La Pression Barometrique: Recherches de Physiologie Experimentale".

1880 Fleuss developed his oxygen rebreather for the use by miners.

1886 P Bert died.

1897 Zuntz used hyperbaric oxygen in the treatment of decompression sickness.

1899 J Lorrain Smith published his data on pulmonary oxygen toxicity.

1906 von Schrotter et al. suggested the use of hyperbaric oxygen in the treatment of decompression sickness.

1912 Bornstein and Stroink first recorded human exposure to hyperbaric oxygen.

1917 Karsnew published the animal pathological histology associated with pulmonary toxicity. He also showed post mortem data on toxicity in other organs.

1926 A Barach investigated the use of normobaric oxygen in the treatment of tuberculosis. He concluded "...the highest concentration of oxygen compatible with safety should be regarded as 60 %."

1929 Soper calculated that using oxygen during decompression may halve the time required for decompression. Davis developed a submersible decompression chamber.

1930 The 2nd RN Deep Diving Unit formed and started to use oxygen in decompression from 60 fsw (18 m) in air dives to 300 fsw (90 m) in the Davis Submersible Decompression Chamber.

1934-6 Oxygen tolerance experiments by Behnke et al.

1935 Behnke and Shaw investigated the use of hyperbaric oxygen in the treatment of decompression sickness.

1939 Behnke and Yarborough developed their air/oxygen treatment tables but these are rejected by the USN. The "John Bean" effect published.

1941 Italian oxygen divers attack HMS QUEEN ELIZABETH and HMS VALIANT, in Alexandria Harbour, Egypt.

1942-45 K Donald's experiments on underwater oxygen tolerance.

1947 Edgar End commenced using 3 bar of oxygen in the treatment of caisson workers with decompression sickness.

1954 Lanphier developed the USN oxygen tolerance limits. He revised these limits in 1959 when nitrox mixtures were used in diving.

1977 Hendricks et al. published their data that showed that pulmonary tolerance could be extended by using air breaks.

1992 A Behnke died.

1993 Harabin's analysis of the data published showed that the threshold for any symptom was 1.3 bar and for convulsions was 1.7 bar.

1994 K Donald died.

References

1 Nunn JF. *Applied Respiratory Physiology. 3rd Ed.* London: Butterworth, 1987

2 Historical Introduction. In *Respiration. 2nd Ed.* Haldane JS and Priestley JG. Oxford: Clarendon Press, 1935

3 *Encyclopaedia Britannica, Inc* 1996
4 Davis RH. *Deep Diving and Submarine Operations. 7th Ed*. London: St Catherine Press, 1962
5 Lavoisier AL and Seguin A. Premier memoire sur la respiration des animaux. *Hist Acad Sci Paris* 1789; 185: 566-584
6 Regnault TV and Reiset J. Recherches chimiques sur la respiration des animaux de diverses classes. *Annals de Chimie et de Phys* 1849; 26: 299-519
7 Bert P. *La Pression Barometrique: Recherches de Physiologie Experimentale*. Paris: G Masson, 1878. Translated by Hitchcock MA and Hitchcock FA and published as *Barometric pressure: Researches in Experimental Physiology.* Columbus, Ohio: College Book Company, 1943. Reprinted Bethesda, Maryland: Undersea Medical Society, 1978
8 Smith JL. The Pathological effects due to increase of oxygen tension in air breathed. *J Physiol* 1899; 24: 19-35
9 Hill L and MacLeod JP. The influence of compressed air on the respiratory exchange. *J Physiol* 1903; 29: 492-510
10 Barach AL. The effect of atmospheres rich in oxygen. *Am Rev Tuberc* 1926; 13: 293-361
11 Behnke AR, Shaw LA, Shilling CW, Thomson MW and Messer AC. Studies in the effects of high oxygen pressure on CO2 and O2 content, pH and CO2 combining power of the blood. *Am J Physiol* 1936; 107: 13-28
12 Bean JW and Rottschafer G. Reflexogenic and central structures in oxygen poisoning. *J Physiol* 1939; 94: 294-306
14 Bornstein A and Stroink M. Ueber Sauerstoff vergiftung. *Dtsch med Wschr* 1912; 38: 1495-1497
13 Bornstein A. Versuche uber die Prophylaxe der Pressluftkrankheit. *Pflug Arch* 1910; 4: 1272-1300
15 Behnke AR and Shaw LA. The use of oxygen in the treatment of compressed air illness. *Naval Medical Bulletin* 1937; 35: 61-73
16 Damant GCC. Physiological effects of work in compressed air. *Nature, London* 1930; 126 (2): 606-608
17 Thomson WAR. The physiology of deep sea diving. *Brit Med J* 1935; 2: 208-210
18 Donald KW. *Oxygen and the Diver*. Hanley Swan, Worcs: The SPA Ltd, 1992; 19-22
19 Behnke AR, Johnson PS, Poppen JR and Motley EP. The effects of oxygen on man at pressure from 1 to 4 atmospheres. *Amer J Physiol* 1935; 100: 565-572
20 Zuntz N. Zur Pathogenese und Therapie der durch rasche Luftdruckanderungen erzeugten Krankheilten. *Fortschr Med* 1897; 15, 633-639
21 Hill L. *Caisson sickness and the physiology of work in compressed air*. London: Edward Arnold, 1912
22 Yarborough OD and Behnke AR. The treatment of compressed air illness utilising oxygen. *J Ind Hyg and Tox* 1939; 21 (6): 213-218
23 Van Der Aue OE, Duffner GJ and Behnke AR. The treatment of decompression sickness: an analysis of 113 cases. *J Ind Hyg and Tox* 1947; 29 (6): 359-366
24 Case EM and Haldane JBS. Human physiology under high pressure. *J Hyg Camb* 1941; 41: 225-249
25 Donald KW. *Oxygen and the Diver*. Hanley Swan, Worcs: The SPA Ltd, 1992; 190-206
26 Donald KW. *Oxygen and the Diver*. Hanley Swan, Worcs: The SPA Ltd, 1992
27 Donald KW. *Oxygen and the Diver*. Hanley Swan, Worcs: The SPA Ltd, 1992; 69-70
28 Donald KW. Oxygen poisoning in man. Part 2. *Brit Med J* 1947; ii: 712-717
29 Donald KW. *Oxygen and the Diver*. Hanley Swan, Worcs: The SPA Ltd, 1992; 27-29
30 Thalmann ED. If you dive Nitrox. *Alert Diver* 1997; May/June: 32-40
31 Kindwall EP. Management of diving accidents, historical review. In *Diving Accident Management. 41st UHMS Workshop*. Bennett PB and Moon RE. Eds. Bethesda, Maryland: UHMS, 1990; 1-11
32 Lanphier EH and Dwyer JV. *Diving with self-contained underwater breathing apparatus (oxygen tolerance). US Navy Experimental Diving Unit Report 8/54*. 1954
33 Donald KW. *Oxygen and the Diver*. Hanley Swan, Worcs: The SPA Ltd, 1992; 87
34 Lanphier EH. The use of nitrogen oxygen mixtures in diving. In *Proceedings of the First Symposium on Underwater Physiology. Publication* 377. Washington, DC: Nat Ac Sc and Nat Res C, 1955; 74-78
35 Lanphier EH. *Nitrogen-oxygen mixture physiology. Phases 1 and 2. Report 7-55*. Washington, DC: US Navy Experimental Diving Unit, 1955
36 Donald KW. *Oxygen and the Diver*. Hanley Swan, Worcs: The SPA Ltd, 1992; 119-140
37 Hendricks PL, Hall DA, Hunter WH and Haley PJ. Extension of pulmonary oxygen tolerance in man at 2 ATA by intermittent oxygen exposure. *J Appl Physiol Resp Environ Exercise Physiol* 1977; 42: 593-599
38 Harabin AL and Survanshi SS. *A statistical analysis of recent Navy Experimental Diving Unit (NEDU) single-depth human exposures to 100-percent oxygen at pressure. Report NMRI 93-95.* Bethesda, MD. Naval Medical Research Institute , 1993

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