outcome (without influencing the shape of the distribution of X_{DCI}). Such knowledge may also allow tailoring of schedules to specific populations so that the intervening normal distributions of processes would be more compact as would the final distribution of sensitivity to DCI (for that population).

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THE NATURAL PROGRESSION OF DECOMPRESSION ILLNESS AND DEVELOPMENT OF RECOMPRESSION PROCEDURES

Richard Moon

Key Words
Decompression illness, history, treatment.

Beginnings

Whereas diving with an open bell, and hence exposure to compressed gas, dates back to at least the 16th century, when decompression illness was first observed is not known. However, when the vacuum pump was invented in the 17th century, there was a tremendous fascination with the effects of vacuum on living things. The first example of decompression illness (DCI) and subsequent recompression was described by Robert Boyle in 1670, when he described the effects on animals of decompression in a bell jar. An excerpt from one of his papers is reproduced below:

"We took a viper and including her in the greatest sort of small receivers, we emptied the glass very carefully, and the viper moved up and down, as if it were to seek for air, and after a while, foamed a little at the mouth, and left off that foam sticking to the inside of the glass: her body swelled not considerably, and her neck less, till a pretty while after we had left pumping; but afterwards the body and neck grew prodigiously tumid and a blister appeared on the back…The jaws remained mightily opened, and somewhat distorted…the air being readmitted after 23 hours in all, the viper's mouth was presently closed, though soon after it was opened again, and continued long so; and scorching or pinching the tail made a motion in the whole body, that argued some life".

This is probably the first written account of decompression sickness (DCS) in animals (and the partial effectiveness of recompression).[1]

Compressed Air Work and Diving

The observations of Robert Boyle remained a laboratory curiosity, with little practical relevance until the development of diving and compressed air work in the 19th century. In 1854 Pol and Watelle first described DCI in miners working in compressed air at Avaleresse-la-Naville in France.[2] A compressed air environment (caisson) in which men performed the excavation, was utilised to keep water and mud out of the working environment. At that site there were 64 men employed, of whom 16 are known to have suffered accidents, and there were two deaths.

After observing bends in compressed air workers during the construction of the bridge over the Seine at Argenteuil in 1861, Foley had recommended construction of a portable recompression chamber capable of sustaining a pressure of 2.5 atmospheres (described by Bert).[3] Presaging an era of questionable clinical use of hyperbaric air therapy, he suggested that this device could be used for treatment of conditions other than bends, such as croup, asthma, snakebite, smallpox and rabies.

The first American major construction job in which compressed air was used was during construction of the bridge (now named the Eads Bridge) over the Mississippi River at St. Louis. Both bridge piers and one of the abutments required compressed air work. There were around 600 men employed in compressed air work in this project. During the East and West Piers there were 91 reported cases of bends, of which 30 were classified as serious; two were crippled for life, and there were 13 deaths. Two thirds of those at the East Pier were attacked immediately on coming out, either on the stairs or as soon as the man had climbed to the top. Some of the severe cases were observed to improve spontaneously, as described in a volume published in 1881 by CM Woodward, Professor of Mathematics and Applied Mechanics at Washington University:[4]

"Pressure 48 lb. GL (age 30) had worked 5 months; was in the compressed air for two hours. Twenty five minutes after coming out he had epigastric pain, paresis of both legs, and retention of urine; also shooting pains in the legs and back. In three and a half months he could walk by the aid of sticks.

"Pressure 50 lb. LB (age 24) had not worked in the compressed air before. Half an hour after his first shift of two hours, he was attacked with paresis of legs and epigastric pain. He was better in a few days, and was warned by Dr Jaminet not to work in compressed air again. In spite of his advice, he worked one more shift of two hours. He afterwards suffered from paraplegia, and paralysis of the bladder and rectum. Cystitis afterwards developed, from which he recovered in two and a half months.".

Climbing the progressively longer staircase from the level of the work site to the surface immediately after decompression appeared to be detrimental, and at the East Abutment an elevator was installed. At that site there were 28 cases; 27 completely recovered and there was one death, the particulars of whom were described by Woodward:

"The comparative immunity enjoyed thus far at the abutment seems to have made the men reckless, and the doctor complains that they would not obey orders as to lying down after coming up from the air-chamber, and as to not drinking water for thirty minutes after coming up. (No reason is given for this last rule)

"Some half-dozen light cases, easily disposed of, occurred previously to April 14 [1871], when the pressure was 49 pounds. On that day a man was taken, who died two weeks later. This was the only death by compressed air at the East Abutment; and it would appear from the report of the doctor and of Superintendent McComas that the man brought his fate upon himself. He had failed to bring his dinner, so went home to eat it, contrary to orders. Then, on the way back, he 'filled himself' with beer. Moreover, on coming up from his second watch, he left the works before his hour of rest was up……On reaching home in the afternoon, the man was taken sick with vomiting. His dinner had evidently been eaten with great haste, and was still undigested. In a few minutes general paralysis supervened. The history of his case up to his death shows that the man's blood was in a bad state. He had worked in the air-chamber over three months."

Medical support for the St. Louis Bridge construction was provided by Eads' personal physician, Dr Alphonse Jaminet, who had a personal experience with bends after spending 2 hours at a pressure of 45 psig (approximately 31 msw). Decompression time was 3 minutes , after which he complained of epigastric pain. Since the lock was at the level of the river bed, he then had to ascend a staircase nearly 100 feet high, which he accomplished with difficulty. Despite leg weakness he was able to climb into his buggy, drive home and stagger into his office, where he became paralysed. It was reported that he was unable to speak for a time. Twelve hours later he began moving his legs. Notwithstanding the woefully inadequate decompression schedule (by today's standards) and the severity of his manifestations, he recovered completely.

Snell, describing the experience in digging the Blackwall Tunnel under the Thames River in 1896, noted that pain resolved after days to weeks; none longer than 5-6 weeks.[5]

"Paralysis usually passes off in from one to a few weeks, unless accompanied by bladder troubles. The fatal cases have usually died from cystitis and bed-sores…..When the bladder is involved some chronic trouble with micturition may remain; and impotence of a more or less lasting character may result from such an illness".

In the largest series of decompression illness in the medical literature, Dr. Frederick Keays, Medical Director of the Pennsylvania East River Tunnels, reported over 3,000 cases of DCS and 20 deaths in tunnelling work in New York. Manifestations are listed in Table 1.[6]

Decompression sickness in divers was first described in the medical literature by a French Navy physician, Leroy

TABLE 1

DECOMPRESSION SICKNESS IN NEW YORK CITY TUNNEL WORKERS

Manifestation		Number	%
Pain		3,278	88.78%
Pain with local manifestations		9	0.26%
Pain and prostration		47	1.26%
Brain symptoms (hemiplegia)		4	0.11%
Spinal cord		80	2.16%
Sensory	36		
Motor	34		
Sensory and motor	10		
Vertigo, "staggers"		197	5.33%
Dyspnea, sense of constriction of chest ("chokes")		60	1.62%
Partial or complete unconsciousness with collapse		17	0.46%
TOTAL		**3,692**	**99.98%**

Compiled from Keays [6]

de Méricourt in 1868, based upon observations made in 1867 on Mediterranean sponge divers, who shortly before had changed from breath hold diving to using the Denayrouze diving apparatus.[7,8] Additional details of bends in this population were reported by Dr. Alphonse Gal, and reprinted by Bert.[3] Forty years later, the disease was described in the English medical literature by Blick, who observed the disease in the pearl divers of Broome and reported 200 cases, of whom 140 lived.[9] Eleven of these cases died, 8 from septicemia due to cystitis and decubitus ulcers and 3 from meningitis. The prognosis of the remainder was surprisingly benign:

"The rest, after a longer or shorter time, recovered, most of them completely, about 10% being permanently affected with slight paresis, generally of the anterior muscles of the legs.

"I have had patients who have been twice, thrice, or even oftener paralysed, and who have more or less completely recovered.

"The treatment after the establishment of paralysis is that of all organic nervous disease-one can only wait on Nature's efforts, though in this disease Nature is kinder than usual....I have been often astonished at the way apparently hopeless paraplegics have recovered in the course of many months.".

Zografidi published further observations detailing the clinical course and autopsy findings of sponge divers.[10] Fig. 1 depicts a 23 year old sponge diver who dived to 70 meters for 25 minutes and made a rapid ascent. He quickly developed pain, followed by paralysis, and was treated with purging, intestinal antiseptic, application of ice to the head and spine, ergotamine, quinine, quinquina, kola and oxygen. He continued to deteriorate, and on the 8th day of the illness he developed decubitus ulcers in the regions of the sacrum, ribs, scapulae and elbows, which was followed by sepsis, complicated by encephalopathy. He died on day 36. The photograph in Fig. 1 was taken two days prior to death .

Green and Leitch reported spontaneous recovery in 8 of 187 cases of serious DCS in a series collected by the Royal Navy between 1965 and 1984 .[11]

Figure 1. A 23 year old sponge diver who dived to 70 m for 25 minutes and made a rapid ascent. The photograph, showing decubitus ulcers on his buttocks, elbows and back, was taken on day 34 of his illness, two days before his death [9].

Development of Recompression Tables for Compressed Air Work and Diving

In 1872 Dr. Andrew Smith, Surgeon to the New York Bridge Company and the man responsible for the welfare of the caisson workers at the Brooklyn Bridge, observed 110 cases severe enough to require treatment, which often consisted of ergot, whiskey or ginger.[12] Atropine was also used, most likely ineffectually, for spasms. Nausea and vomiting were treated with calomel (mercury chloride). The more mildly affected were reported to have prescribed their own treatment at the local saloon. The impression of the workers and engineers at the time was that most of the afflicted men recovered, irrespective of Smith's treatments. In fact, Smith was in favour of recompression treatment, and conceived a design for a medical lock specifically for this purpose. Unfortunately for the bridge engineer, it was never built. After John Roebling, the bridge designer, died of tetanus following a crush injury to his toes, his son, Washington, took over as Chief Engineer. After exiting the caisson on two occasions he suffered lower limb paresis due to DCS. Unfortunately he was not as lucky as Jaminet, and after the second attack, though eventually able to walk, remained impaired for the rest of his life.[12]

Recompression treatment, although noted anecdotally as early as the 1840s to be effective, was not implemented for several years. Pol and Wattelle had suggested recompression as a treatment,[2] although the idea was not well accepted, perhaps because for an injured man it was counter-intuitive to re-enter the environment that was responsible for his illness. Recompression treatment for bends was not, in fact, systematically utilised until the end of the 19th century. Ernest Moir, an engineer who in 1889 assumed responsibility for construction of the first tunnel under the Hudson River between Manhattan and New Jersey, should be credited with the installation of the first recompression chamber.[13] Injured men were recompressed using air to two thirds of the pressure at which they had been working, then slowly decompressed to atmospheric pressure at 1 psi per minute or slower, requiring 25-30 minutes. Moir recognised that recompression was less successful after a delay, a fact well known to 20th century diving doctors. Before instituting this treatment, Moir reported that approximately 25% of the workforce per year had been dying from DCS. Afterward, he reported that of 120 men at work, only two died during a 15 month period. This positive experience was confirmed by Snell in England in 1896.[5]

Keays used a recompression schedule that was slightly different from the one used by Moir. Keays used a recompression pressure equal to the one in which the man had been working.[6] In his algorithm, as soon as pressure equalled tunnel pressure, decompression was initiated, with the decompression time in minutes at least as great as twice the number of pounds of pressure. In the event of recurrences, the procedure was repeated up to two or three

times. For joint pain, Faradism (use of electrical stimulation to cause muscle contraction), hot compresses and hot baths were sometimes used to supplement recompression . Keays' results for joint pain in one knee are shown in Fig. 2. Additional details of the history of hyperbaric medicine of this era can be found eloquently detailed in volumes by McCullough[12] and Phillips.[14]
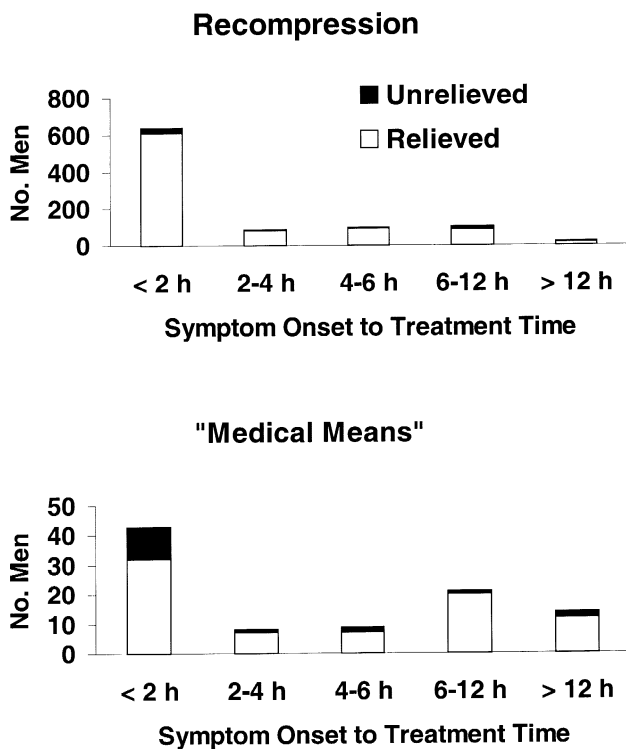

Figure 2. Keays' data comparing recompression with non-recompression therapy ("medical means") for 1,050 compressed air workers with joint pain in one knee.

Recompression therapy for divers took longer to catch on. In the Royal Navy, even as recently as 1907, although oxygen administration at the surface was recommended for the treatment of bends, and in-water recompression practiced, recompression chambers were not routinely available for diving work.[15] The US Navy (USN) Diving Handbook in 1905, containing a meagre 44 pages, did not even mention DCI or its treatment.[16] It was not until the 1924 edition of the USN Diving Manual that a standard therapy was proposed (Table 2).

The development of tables since the beginning of the 20th century has been concisely and accurately detailed in a recent *SPUMS Journal* article by Dr Chris Acott.[17] There are few reports in the medical literature pertaining to recompression treatment of DCS until the 1930s. Even at that time, treatment was largely empirical. Reporting on the experience during construction of the New York-Queens Midtown Tunnel in 1938 wrote that the recommended treatment pressure was equal to that to which the worker was originally exposed, although sometimes, in

TABLE 2

USN DIVING MANUAL (1924 EDITION) INSTRUCTIONS FOR TREATING DCS

Compress to 45 psi (101.5 fsw).
If substantial relief not achieved, compress
to 60 psi (135.3 fsw).
Stay at depth of relief until symptoms relieved.

Decompress at the following rates:

60-45 psi	1 psi/min
45-30 psi	1 psi/3 min
30-15 psi	1 psi/5 min
15-0 psi	1 psi/10 min

TABLE 3

EXPERIENCE WITH LONG AND SHORT AIR AND O_2 TABLES IN THE USN IN 1944

Table	Cases treated	Successful treatments	Recurrences
Short O_2 Table	6	6	0
Short Air Table	9	7	2
Long O_2 Table	10	5	5
Long Air Table	5	3	2

(Compiled from Van Der Aue[21])

order to achieve relief of symptoms, it was necessary to treat at 5, 10 or 15 psi higher.[18] After recompressing an injured man, stated Thorne, "The consensus favors waiting twenty to thirty minutes before starting the decompression process". One can infer that there was not unanimity of opinion on this issue. He went on to report that, despite the waiting period, symptoms were apt to recur, requiring one or more additional recompressions.

Oxygen administration, in part because it was not readily available in sufficient quantities until the mid 20th century, was not routinely used until much later. In the 1870s, Paul Bert, working in France, first noted that when 100% oxygen was administered to animals with DCS, some of the signs would resolve.[3] He suggested that oxygen administration caused resolution of gas within the veins and the right heart, but that recompression (with air) was necessary to resolve bubbles that had migrated into the central nervous system. Bert did not put the two modalities, oxygen and pressure, together, and it was Nathan Zuntz, a Professor of physiology in Berlin, who in 1897 suggested simultaneously using both to treat DCS, although he did not have facilities to try it.[19] The initial results of what would today be considered abbreviated oxygen therapy, were somewhat disappointing. Keays reported that oxygen had been given to several severe cases during decompression, but afforded no appreciable benefit.[6]

In 1939 Yarbrough and Behnke reported methods by which injured divers could be treated using oxygen under pressure, but these were not initially adopted.[20] Although the authors were USN investigators, 30 years would elapse before the USN introduced oxygen recompression as a routine. Instead, in the 1943 USN Diving Manual a new table was introduced, in which the diver was compressed using air to the depth of relief (up to 300 fsw), remained there for 30 minutes, then decompressed according to a schedule requiring up to 387 minutes of decompression time. In 1944 four tables were issued (long and short air tables, long and short O_2 tables).[17] The short tables had a maximum recompression depth of 100 fsw, while the long

tables had a maximum depth of 165 fsw. In the O_2 tables 100% O_2 was administered at depths of 60 fsw and shallower, but only for a total of 95 minutes. Only 30 minutes were spent breathing O_2 at 60 fsw (compared with the current standard of 60 minutes or more using USN Treatment Table 6), and a total of 95 minutes of O_2 in all. In 1947 Van Der Aue and colleagues reported the 1944 experience with these four tables, shown in Table 3.[21]

USN Tables 1 (Short O_2 Table), 1A (modified Short Air Table), 2 (modified Long O_2 Table), 2A (modified Long Air Table), 3 (modified Table 2A; not tested) and 4 (for difficult cases) were then promulgated, and used exclusively between 1945 and 1965. Several bends occurred in tenders during use of these therapeutic tables, and the failure rate for the injured divers was nearly 30%.[22]

USN Tables 5 and 6, in which divers were administered 100% O_2 at 18 msw and 9 msw continuously, except for short air breaks to reduce O_2 toxicity, were then developed, tested and adopted for field use. Workman's 1968 analysis revealed that these oxygen tables had a high degree of success.[23] Experience since that time has confirmed the initial observations (see Table 4), and these treatment tables remain in use today.[24]

It was believed that tables different from the ones used for DCS would be required for the treatment of arterial gas embolism (AGE). Studies to determine the appropriate treatment pressure for AGE were performed by investigators in the US Navy in the 1960s. Intracarotid air injection was performed in anaesthetised dogs. Using a skull window technique, Waite observed that all visible bubbles disappeared at a compression depth of 100 fsw, suggesting that this should be the appropriate compression depth for AGE in divers.[25] It was believed, however, that fleet diving officers would not accept a table with only 100 fsw as the maximum depth.

Thus USN Table 6A, specifically designed for treatment of arterial gas embolism, incorporated a 30 minute period of air breathing at 165 fsw, followed by O_2 administration according to USN Table 6.

TABLE 4

SINGLE RECOMPRESSION SUCCESS RATE OF USN OXYGEN TREATMENT TABLES

| Source	Number of cases	Complete relief %	Substantial relief %	Comments
Workman [23]	150	85	95.3%	after 2nd treatment
Erde and Edmonds [47]	106	81		
Davis [48]	145	98		Altitude DCS
Bayne [49]	50	98		
Pearson and Leitch [50]	28	67	83	
Kizer [51]	157	58	83	Long delays
Yap [52]	58	50	84	Mean delay 48 hours
Gray [53]	812	81	94	
Green [54]	208	96		All pain only, USN Table 5
Ball [55]	14	93 (mild cases)		
	11	36 (moderate cases)		Many cases with long delays
	24	8 (severe cases)		
TOTAL	**1763**	**81%**		

(Compiled from Thalmann[24])
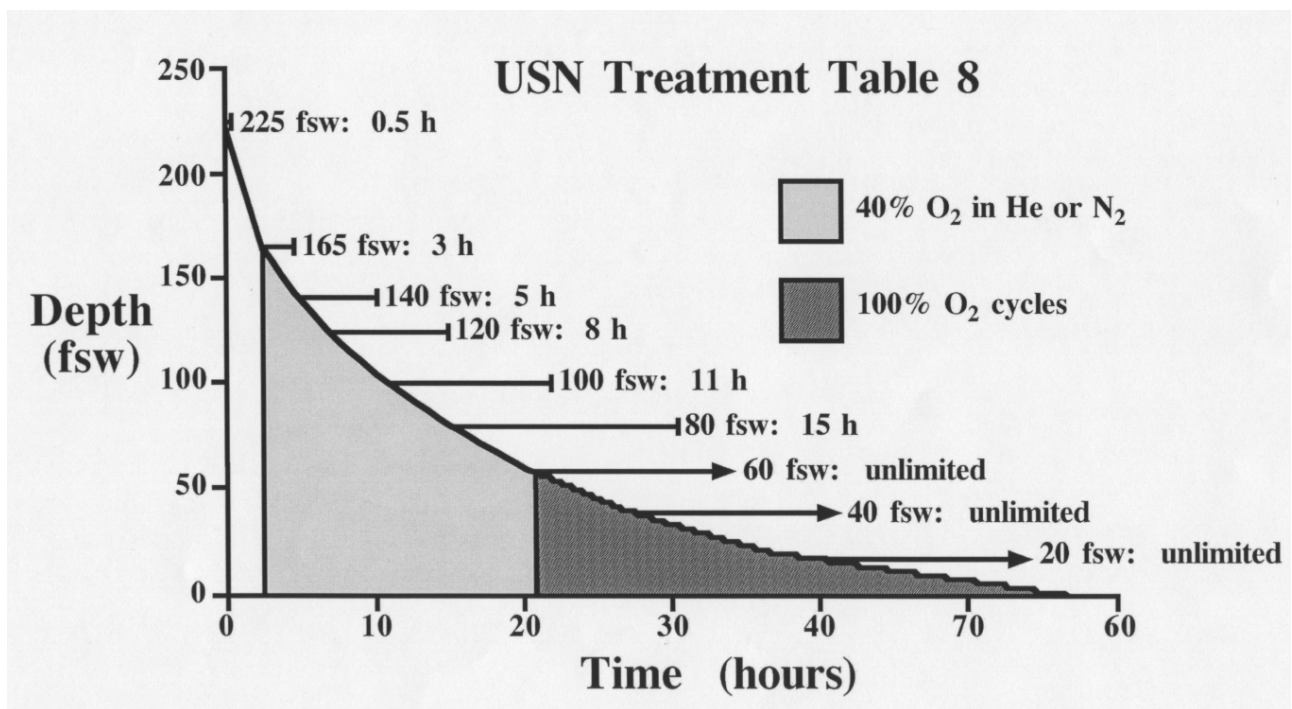
Figure 3. USN Treatment Table 8. Designed for treatment of deep "blowups", in which there has been more than 60 minutes of missed decompression stop time. It can be used in other situations, for example to compress to a higher pressure than 6 ATA (50 msw, 165 fsw), or to stop decompression between 18 m (60 ft) and 50 m (165 ft). Maximum times at each depth are shown; times at 18 m, 12 m and 6 m (60, 40 and 20 ft) are unlimited. Decompression occurs in increments of 0.6 m (2 ft). When deeper than 50 m (165 ft, 6 ATA), to reduce narcosis, a 16-21% O_2 in helium can be administered. Four treatment cycles, each consisting of 25 minutes of "treatment gas" followed by 5 minutes of chamber air can be administered deeper than 18 m (60 ft). Treatment gas used deeper than 18 m (60 ft) is 40% O_2 in either He or N_2; at 18 m (60 ft) or shallower treatment gas is 100% O_2. USN Treatment Table 7 guidelines are used for O_2 administration at 18 m (60 ft) or shallower. Further details can be found in the US Navy Diving Manual [27]. Figure reproduced from Moon et al [29], with permission.

Developments since the 1960s have included "specialty" tables designed to treat bends under exceptional circumstances, such as saturation treatment (e.g. Miller saturation table,[26] USN Treatment Table 7[27]) after deep dives or after rapid ascent to the surface with omitted decompression ("blowups"). USN Treatment Table 8 is a relatively new table designed for the latter purpose (see Fig 3).[27] Other developments have included modification of standard USN treatment tables to allow more intensive treatment. For example, the Catalina modification of USN Table 6 allows up to eight 20 minute periods of 100% oxygen breathing at 18 msw.[28,29] Others have modified USN Table 6A to allow longer than the usual 30 minutes at 50 msw, and breathing enriched oxygen mixtures.[30] Shorter tables have been designed to permit treatment of DCI while continuously breathing 100% oxygen in a monoplace chamber without an excessive risk of oxygen toxicity.[31-34]

Altitude Decompression Illness

In France in 1875 three men, Crocé-Spinelli, Sivel and Tissandier, attempted to reach an altitude record in the hydrogen filled balloon *Zenith*. The balloon ascended to an altitude of about 8,000 m and then began descending. Desiring to go higher, the balloonists dumped all of the available ballast, and the balloon resumed its ascent. After landing, Crocé-Spinelli and Sivel were dead. In Tissandier's written account (which is reproduced by Bert)[3] related that at one point in the flight he was unable to move his arms. One might speculate that this effect might have been due to neurological DCS rather than the hypoxia to which it has traditionally been ascribed.

The possibility of altitude DCS was first considered by Henderson in 1917.[35] He speculated that the disease would not occur until an altitude of 20,000 feet had been reached, an impossible feat for aircraft of that era. Juxta-articular pain, which was probably due to bends, was described in a paper by Barcroft and colleagues in 1931.[36] In a study at 30,000 feet in an altitude chamber, one of the authors (Margaria) developed knee and muscle pain.[36] Paralysis was first described, at an altitude of 35,000 feet, in 1938.[37] Altitude bends were experienced during operational flights in World War II, since few aircraft were pressurised, and limited or no oxygen pre-breathing was performed. Of 215 B-17 bomber aircrew during World War II who answered a questionnaire, 39 reported 90 instances of bends[38]. When pressurised high altitude reconnaissance aircraft were first introduced, two of three Spitfire pilots who engaged and shot them down at 42,000 ft suffered severe DCS.[39]

Treatment Tables for Altitude Decompression Illness

Bends were systematically studied in flight using a B-24 bomber, using pressure bags in which air crew with
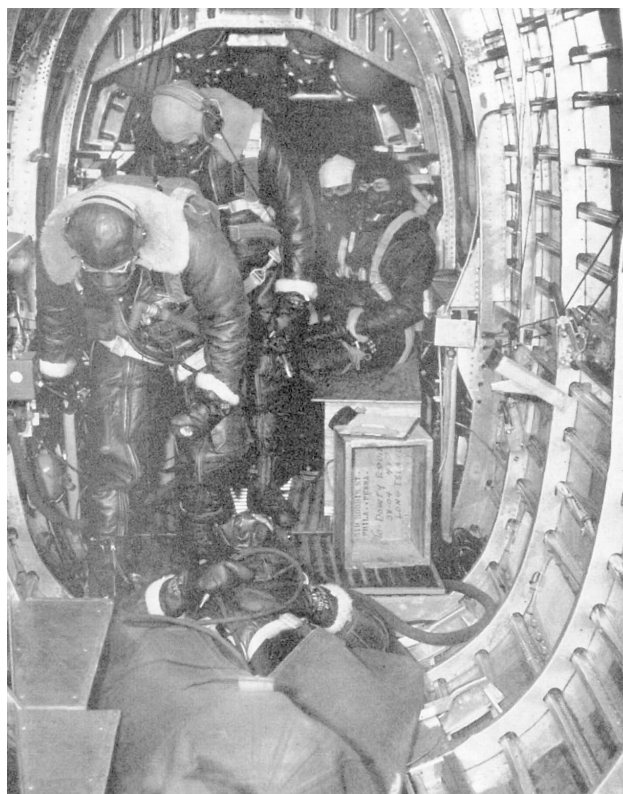

Figure 4: Recompression bag developed in World War II to treat altitude DCS in air crew. Inflatable to a pressure of 3.5 psi, it was a forerunner of the bags currently used to treat altitude sickness (photograph from Lawrence and colleagues [40]).

bends were placed. Symptoms were treated by pressurising the bags to 3.5 psig (see Fig 4).[40] The resulting "descent" from 35,000 feet to around 21,000 feet was successful at resolving even severe symptoms such as "chokes". Despite oxygen pre-breathing altitude DCS still exists today. In an anonymous survey, three quarters of 416 active duty and retired U-2 pilots admitted to having experienced in-flight bends at some time during their career.[41] Manifestations of altitude DCI are somewhat different from bends associated with diving.[42] Of 447 cases observed during altitude exposure in a hypobaric chamber, 83.2% had musculoskeletal involvement (70% knee pain, which is less common in diving than arm pain), 2.7% had chokes, 2.2% skin manifestations, 10.8% paraesthesias, and 0.5% more severe neurological features.[43] The low prevalence of neurological features may be due to the use of oxygen pre-breathing prior to ascent and early recompression in this setting. It is also possible that individuals in the training and operational environments are more likely to report frank neurological involvement than other forms of DCS.

Minimal recompression was observed in the World War II era to be effective in most cases of altitude bends, with only a minority of victims reporting symptoms after

return to ground level.[39] However, in some cases the symptoms and signs persisted, as exemplified by the following case, initially reported in 1943, and published by Ferris:[44]

> "This man developed vasodepressor syncope during descent, recovered consciousness briefly on reaching ground level, and then lost consciousness again. When examined in the hospital, he was unconscious and the arterial pressure was 108/76 mmHg and the pulse rate was 80 per minute. The patient remained stuporous for two days, during which time he developed several convulsive seizures beginning in the right arm and becoming generalised, and examination revealed a right hemiparesis. As consciousness returned, the patient complained of impaired vision, which then cleared in the next few days. Lumbar puncture on the third day revealed a cerebrospinal fluid pressure of 190 mm H_2O".

The modern, more meticulous approach to treatment of altitude bends includes a period of 100% oxygen breathing at ground level, followed by recompression with oxygen if symptoms persist.[45,46]

As the Space Shuttle cabin pressure is 1 ATA and space suit pressure is only one third ATA, DCS could also occur in space during extravehicular activity. Although it has been rarely reported during space operations, ground based chamber simulations of the decompression profiles in use by Space Shuttle astronauts has produced both pain only and neurological bends. There is also the possibility of catastrophic decompression in the event of suit rupture. A recompression chamber was initially eliminated from the Space Station currently under construction because of space and weight considerations. If recompression is required for an injured astronaut it will be by breathing 100% oxygen in a pressurised space suit, although installation of a pressure chamber has received reconsideration.

Summary

The initial observations in the 19th century of DCS in compressed air workers and divers revealed a disease that could cause severe neurological injury and death, but would often surprisingly resolve spontaneously. The standard of care for this illness today includes recompression with oxygen. For diving injuries occurring in scuba divers or air crew there are presently no convincing data indicating the superiority of any table compared with USN Table 6 or its equivalent. Recompression is a highly effective treatment in most cases if applied shortly after the onset of symptoms.

Therefore there is (fortunately) little opportunity to monitor the natural course of this illness. However, in keeping with the 19th century experience, it is frequently observed that, when severe instances of DCI do not respond to recompression therapy, the prognosis several weeks or months later is often good. Unlike traumatic spinal cord injury, in which improvements are usually small, the cord is often tolerant to injury of similar severity due to gas bubbles.

The obvious combinations of treatment pressure, time and breathing gas composition have been examined, at least anecdotally. Further advances in the treatment of decompression illness are unlikely to occur through new table development, but rather mechanisms by which more prompt recompression can be administered and new adjunctive resuscitative measures.

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TESTING
JS HALDANE'S DECOMPRESSION MODEL

Chris Acott

Key Words

Diving theory, history, physiology, research, tables.

Last year I presented a paper on J S Haldane summarising his work.[1] As the majority of dive computers are based on the decompression algorithm that J S Haldane designed, I present here the testing procedures, for goats and humans, used to support his conclusions. Quotations from Haldane's report[2] are printed in *italics* in this paper.

Haldane's assumptions

1   That for bubble formation, the tissue pressure must be greater than the environmental pressure.

2   That tissues can hold gas in supersaturation, and only if the decompression rate is correct this supersaturation can be tolerated without risk.

3   That there were no symptoms of decompression sickness (DCI) without bubbles. We now know that is not correct.

4   From Sir Leonard Hill's work Haldane assumed that carbon dioxide had no influence on decompression risk. We now know this is probably incorrect.

5   That tissue perfusion is the limiting factor in gas uptake. Haldane adopted a mathematical simplification, in the form of an exponential, to describe uptake of inert gas. Hill considered that the limiting factor was gas diffusion.

6   That gas elimination is the mirror image of gas uptake. We now know that is incorrect.

Half times

Using these assumptions and the published work of other people Haldane divided the body into arbitrary half time tissues. A half time is half the time it takes to saturate a tissue with a specified gas. A short half time denotes a tissue with a good blood supply and rapid saturation. A long, or slow, half time is a tissue which takes up the gas slower because of a lesser blood supply. Haldane calculated these half lives to represent what happened in the body. Moir's data gave him his 20 minute half time. Haldane used Hill and Greenwood's[3] data of nitrogen excretion to provide his 5 minute half time. That is based on the nitrogen content of urine. Hill and Greenwood considered that as the kidneys are very well perfused in life they represented what we now call a fast tissue. They experimented on themselves in a chamber under pressure where they passed urine every 5 minutes or so. The urine was passed out of the chamber and the nitrogen content measured. JS Haldane's experiments on goats gave a 75 minute half life. From his mathematical calculations he showed that, if the body had an equal perfusion, then this would represent a 10 minute half time. I have not been able to find the origin of his 40 minute half time.

Funding for Haldane's research was both private and from the Admiralty. At first Haldane started by using small animals. Later he used a large chamber, donated by Ludwig Mond, based at the Lister Institute in London. Haldane worked with Boycott, who was Professor of Physiology at Oxford University, and Damant, who was a Lieutenant in the Royal Navy (RN) and Inspector of Diving. When the tables were developed, Damant and Mr Catto, Gunner, RN, were the divers who tested the tables in London and in the water in Scotland. Damant was also involved with the second RN Deep Diving Unit in the 1930s. In the 1930s Damant modified the Haldane tables so that RN divers could dive to 300 feet on air.