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### EARLY DECOMPRESSION EXPERIENCE: COMPRESSED AIR WORK

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#### Key Words

Decompression illness, history, osteonecrosis, working in compressed air.

#### Introduction

Although there must be more than 100 divers at this meeting, I doubt if many have been exposed to compressed air in a civil engineering project. One of the last major such projects in the Southern hemisphere that I am aware of was the construction of the Auckland Harbour Bridge some 40 years ago but of course since then there have probably been many smaller contracts for sewage outfalls, bridges and tunnels. So what, if anything, does the history of compressed air work have to do with recreational diving in the South Pacific today?

The answer lies in studying these pioneering exposures to pressure in order to find observations which might improve our understanding of today's pressure-related illnesses and their prevention. The development of these early engineering procedures was associated with a gradual realisation that these achievements also led to some adverse medical consequences for those who were exposed to raised environmental pressure. Methods for decompressing the workforce did not exist at the beginning of this industry, and written procedures of around 100 years ago are scarce. The history of this era deserves more study than this summary can provide. Clinical examination in those days could be detailed but some of the terms are difficult to interpret and many of the therapeutic approaches are, with hindsight, totally inappropriate. Over the same period of some 50-60 years there were parallel developments in diving with similar lessons to be learned from diving case histories, but the total number of persons in diving were many fewer. The exposures in compressed air workings are generally very much longer and this difference is associated with some differences in clinical presentations and long-term effects from which useful conclusions can also be made.

#### Recognition of a new medical problem

Professor Trouessart in his report to the Industrial Society of Angers in 1845 is quoted by Paul Bert<sup>1</sup> as attributing the concept of the caisson to Denis Papin who said in 1691 that if fresh air were pumped constantly into a

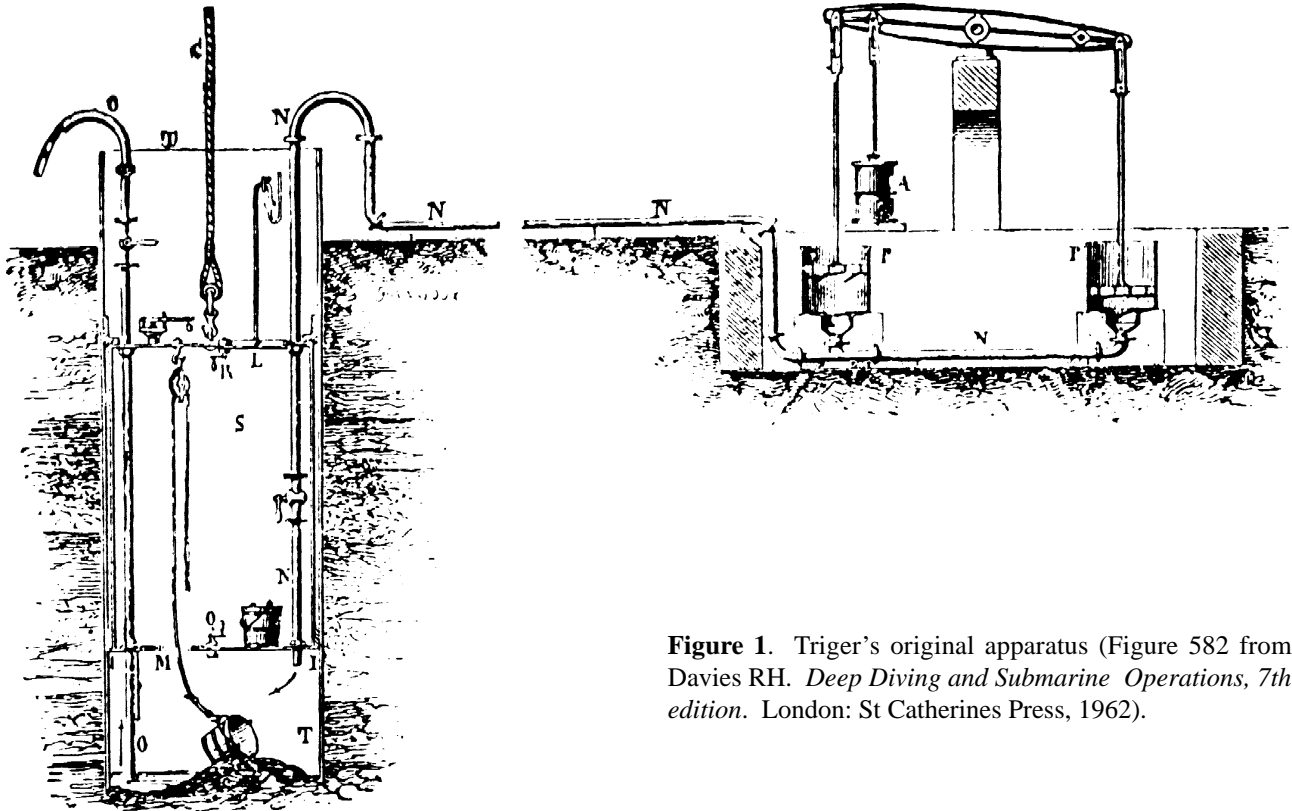
bell on the sea-bed by strong leather bellows, the bottom of it could be kept almost entirely dry and one could work on the sea or river bed just as effectively as at the surface. There was no industrial need to develop Papin's practical idea for working in compressed air until 1839 when coal deposits were found 20 metres under the quicksands and alluvial deposits of the river Loire in France. Although Sir Thomas Cochrane, had some 10 years previously patented the concept of using compressed air in water-laden ground for constructing shafts and tunnels, the credit for making such ideas work in caissons goes to M. Triger. A complete account of the caisson he developed for opening up this coalfield was presented to the French Academy of Sciences in 1841 and published in 1845.<sup>2</sup>

The technical aspects of these developments do not concern us here but, as they provided some of the very first observations of the effects of raised environmental pressure upon man, it is worth mentioning what was reported at the time. Triger noted the increasingly nasal quality of speech with increasing depth and, on reaching around 3 atmospheres, the loss of the ability to whistle. Though not the first to do so, he also described pains in the ears associated with compression and decompression and further said of the workmen that ... *they become much more tired when working in compressed air than in open air. We think that this is the result of the very great humidity .....*, *which hampers the insensible perspiration and promotes the more rapid secretion of sweat in those who have to exert their physical powers ... . Perhaps this humidity would also explain the somewhat severe pains in the articulations experienced by some workmen a few hours after leaving the shaft ... .*

He also describes the case of two workmen who, after .... *seven consecutive hours in compressed air, experienced rather keen pains in the articulations, half an hour after leaving the shaft. ... Rubbing with spirits of wine soon relieved this pain in both men; they kept on working the following days.*

As Paul Bert commented some years later, Triger gave no details about the duration of the decompression, only that they opened the cock gradually.

The method developed by M. Triger was then used to open coal mines in Northern France where, as reported by M. Blavier,<sup>3</sup> the engineering problem of penetrating permeable limestone was much greater than that of penetrating soft quicksand. After his own exposure to around 2.6 to 3 atmospheres on 5 December, he reported that the next day .... *keen pains appeared in the left side, and we felt a rather severe painful discomfort for several days afterwards. ... After we were quite free from these pains, 28 December, we were anxious to try the experiment again*



**Figure 1.** Triger's original apparatus (Figure 582 from Davies RH. *Deep Diving and Submarine Operations*, 7th edition. London: St Catherines Press, 1962).

... at the same hour, that is, 20 hours after our exit from compressed air, we felt in the right side pains just like the former ones, which kept us numb for four or five days.

The maximum pressure there was 4.25 atmospheres (32 m) with shifts twice daily of around 4 hours, each with a 45 minute compression phase, and each followed by a 30 minute decompression. The Superintendent of the mine in Douchy had assured Blavier that the various symptoms of the workmen, *heaviness in the head or pains in the legs*, almost always coincided with some excess committed by them between shifts. *Only one of them experienced complete paralysis of arms and legs for 12 hours*. Of the 64 workmen there, 2 died.

Dr Pol and Dr Watelle went to these mines in order to examine these phenomena but their study, upon their own admission, was not designed for publication. It was a collection of *observations made without plan*.<sup>4</sup> Nevertheless from their observations they deduced that "*The danger does not lie in entering a shaft containing compressed air; nor in remaining there a longer or shorter time; decompression alone is dangerous; pay only when leaving*".

As one reads through their case descriptions from among the men employed there, one is struck by decompression experiences which, hopefully, are now only rarely encountered and, if they do occur, would be managed differently. For example, Case 1-V:

*One day, an hour after leaving the shaft,... he complained of distress; when placed in bed he lost*

*consciousness. Pulse full and rapid, face congested, respiration short and stertorous; obscure sound everywhere ... bled, purged, plastered. After 4 hours, return to consciousness. In 3 days, cured.*

From among the 10 or so serious cases, i.e., those with more than just musculo-skeletal pains, one notes a curious absence of what might be termed "spinal" manifestations. The observed symptoms were attributed by them not to bubbles but to "*superoxygenation and congestion*". Nevertheless, they said that one is justified in hoping that a sure and prompt means of relief would be to recompress immediately, then decompress very carefully. Indeed,<sup>1</sup> at another mine in Belgium, some workers with severe attacks of articular pain were completely relieved by returning to work, but then found that the *pains returned after leaving the apparatus*. To put these developments into chronological perspective, the paper by Pol and Watelle<sup>4</sup> was published in 1854, the same year that Florence Nightingale went to the Crimean War.

With the subsequent use of caissons in bridge building around Europe and then around the world, illness among the workforce was reported time and time again. For example, symptoms<sup>5</sup>... *were much worse when the change was made from caisson to the open air: serious, even fatal, symptoms appeared then.*

And another case .... *completely prostrated ... unable to walk, hands and feet cold and without sensation. Was seated with his feet in the fire, so that several of his*

toes were burned without feeling the heat... Two days later he was cured except for his burns.

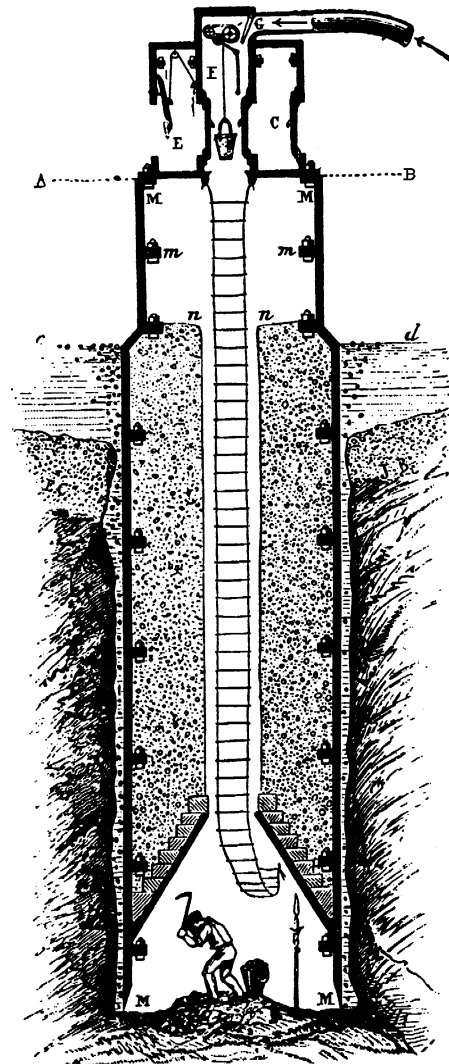
During the building of a bridge in Bayonne,<sup>1</sup> a 20-year old engineer decompressed in 4 or 5 minutes after 60 minutes at 4 atmospheres. Within a few minutes he had dizziness followed by unconsciousness. When he regained consciousness after 3 hours he was paraplegic with also loss of sensation in the arms. After bedsores and other complications, function began to return after seven weeks. Twelve years later he could climb one flight of stairs with difficulty. Bert<sup>1</sup> also quotes a mining student's own description of his decompression manifestations. *During the decompression I felt a discomfort which I attributed to the cold. After I had come out, when I wished to raise my right arm, I could not make it reach a definite point. My sight was affected, and I saw ... much as one perceives objects after whirling around several times. The paralysis grew worse .. I could not walk.. I was dazzled and my eyes refused to serve me at all. My eyes were dull and glassy, they told me, and perceived only a white vaporous light. I recovered first the use of my leg, then of my arm; ... and I saw distinctly for longer periods. Finally ... my headache disappeared in the open air and I went home, having nothing but fatigue to remind me of my former experiences.*

Many similar clinical observations continued to be made and were accompanied by various explanations of the underlying patho-physiology many of which can now be dismissed as fanciful. In the absence of any facts, these were vehemently argued and used by some to justify the apparent need for a slow decompression but with a totally incorrect logic.

At the same time, there were others<sup>1</sup> who did not consider the rate of decompression to be important but said, for example, that the major factor was chilling during decompression...*if the thick and icy mist, which is sure to appear, seems to be penetrating you, make haste!*

But a few curious clues did emerge. For example, M. Bucquoy<sup>6</sup> in 1862 watched the use of dry cupping-glasses placed around a workman's painful knee. The cups had been applied properly by a skilful orderly but, repeatedly, after a certain time they fell off. He concluded that the elimination of free gases explained the prompt disappearance of the knee pain through repeated application. He went on to conclude that the gases of the blood are increased in quantity, and *...at the time of decompression, these gases tend to be liberated again, just as the carbonic acid escapes from charged water when the stopper is removed from the bottle containing it.*

Another comment, possibly based on Hoppe-Selyer's observation in 1857 that decompression to altitude had caused bubbles in the heart and blood vessels of a rat,<sup>7</sup> was made by a Danish physiologist, Panum<sup>8</sup> who, after studying dogs' blood pressure at depth in order to disprove



**Figure 2.** One of the first caissons used in France by Foley (Fig.6 from Paul Bert<sup>1</sup>).

some other fanciful hypothesis, said that the symptoms of decompression *...result from the fact that the air which has suddenly been liberated in the blood vessels ... forms embolic obstructions in different vascular regions.*

An engineering report,<sup>9</sup> based on some 10 locations around France, stated that the diseases caused by these accidents could be prevented by the use of the means which they specified...

- woollen garments in the lock chamber
- not to open the cock too quickly... M. Triger requires that the decompression last 7 minutes, and states that then the symptoms disappear completely. It seems to us that this time should vary with the constitution of the workman.

No thoughts about depth and duration of exposure yet, though in Belgium around that time M. Barella

suggested a decompression rate of 10 minutes per atmosphere.<sup>10</sup>

One of the first bridges to be constructed in the United States using work in compressed air was over the Mississippi at St Louis where a large number of workmen was exposed to pressure (34 m at the East pier; 4.4 bar). With decompression in 3 to 4 minutes, 30 of the 352 men were seriously affected and 12 died. The clinical report on some of these cases is interesting but not relevant to the prevention of decompression injury.<sup>11</sup> The report by the engineer, Captain Eads<sup>12</sup> after whom the bridge is named, describes the use of 'galvanic rings' of zinc and silver which were worn by the majority of the workmen to stop attacks of paralysis or pains. He also mentions that the physician at the site was severely affected after 2 hours at 90 feet (27m) and, in Dr Jaminet's own words,<sup>13</sup> *...We were only three minutes and a half in the air-lock to return to the shaft or normal atmosphere. ... epigastric pain ... dizzy ... I reached home ... three quarters of an hour after leaving the caisson. The last effort brought me to my office where in a few minutes I became paralysed...* in both legs and left arm and also had transient aphasia. Within 10 hours or so his paralysis had gone though he felt weak for another week or so.

Some physicians, Eads says, *..... maintained that a slower return to normal pressure would have been less dangerous; others blamed too rapid compression ... .. we believe that the real cause lies in the long duration of the stay in this air ... at pressure, and not the rapid changes to which it is exposed.*

This belief appears to be based not on nitrogen uptake but on a 30 to 50% reduction of respiratory rates at pressure, and thus is a reaction *...against the introduction of oxygen in a proportion 2 to 3 times greater than in normal atmosphere.*

As Eads stated elsewhere in his report *...The duration of stay in the air chamber was gradually shortened from 4 hours, to 3, to 2, and finally to 1 hour after which there were no more fatalities.*

Many of these are among the observations and reports which formed the background to Paul Bert's experimental work,<sup>1</sup> the real importance of which is that he reported evidence in 1878 to suggest that on decompression *... all the symptoms, from the slightest to those that bring on sudden death, are the consequences of the liberation of bubbles of nitrogen in the blood, and even in the tissues when compression has lasted long enough.*

He made no hypotheses concerning the dynamics of the dissolved gases but concluded: *"The great protection is slowness in decompression; ... from 3 to 4 atmospheres, one hour ... .. The longer the workmen remain in the caissons, the more slowly they should undergo decompression, for*

*they must not only allow time for the nitrogen to escape from the blood, but also allow the nitrogen of the tissues time to pass into the blood".*

These rates are between 0.5 m and 1 m per minute, similar to those proposed later by von Schrotter,<sup>14</sup> 20 minutes per atmosphere. Bert also proposed that if a workman becomes stricken one should return him to a pressure greater than that from which he came, then make decompression very slowly.

### Frustrated beginnings of decompression theory

Paul Bert's clear conclusions in 1878, together with his pioneering work on oxygen toxicity, mark the start of the scientific approach to hyperbaric physiology. One might expect such logical recommendations to be adopted by the doctors who had responsibilities for compressed air workers, but not so. Dissemination of knowledge and the subsequent conversion of non-believers took time and in the meanwhile much preventable morbidity continued.

The name "bends" for the musculo-skeletal variety of decompression illness was adopted from the name of a then fashionable ladies' posture, the Grecian Bend, by the men who were working on the Mississippi bridge at St Louis and those working on Brooklyn Bridge in New York. In 1870 the Brooklyn workings were at 20 m (79 ft) and, although Dr Smith<sup>15</sup> at that location also recognised that re-exposure to compressed air would alleviate the symptoms, recompression was not made available for the 110 cases of serious decompression injury that did occur. Dr Smith rejected the intravascular bubble theory because Bert's experimental work had been from a greater pressure than that experienced by tunnel workers and because he attributed the illness to the long-standing hypothesis of vascular congestion due to vasomotor paralysis

When the construction of a tunnel under the Hudson was resumed in 1879, it reached 30 pounds [per square inch] (about 20 m depth) and at the beginning provided some of the worst recorded decompression risks ever encountered in industry. In 1882 there was a mortality of 25% of the workforce there in one year but, fortunately for the tunnellers, insufficient funds caused work there to cease.<sup>16</sup>

When work on the Hudson tunnel restarted in 1890, *an air compartment like a boiler was made in which the men could be treated homeopathically, or re-immersed in compressed air. ... The medical lock should be used at once, as it does not appear to have much effect after some time has elapsed.*

By introducing the lock,<sup>16</sup> the deaths were reduced to only two in fifteen months, 1.5%.<sup>17</sup>

In the year that Marconi patented radio, 1896,

tunnelling was being carried out extensively in London and other great cities in response to the increasing demands of urban development, Sir Ernest Moir<sup>16</sup> made his address on *Tunnelling by Compressed Air* to the Society of Arts and Snell<sup>18</sup> published his book on *Compressed Air Illness: Blackwall Tunnel*.

It may have been an era of major civil engineering throughout Europe and North America, but not everyone involved necessarily understood the physics of pressure. In the early 1890s when the Blackwall Tunnel had completed a significant phase of its construction, the builders threw a champagne party for dignitaries at pressure but, because of the raised ambient pressure, they could not get the corks out! When at last they succeeded, by drilling holes through the champagne corks, they found that the champagne was flat. After all that effort to keep the party going, they drank it anyway. There were some reports about their subsequent decompression experiences in the press but, alas, no detailed account by any of the party-goers.

In 1904, just a year after the Wright brothers had flown the first aeroplane, the Hudson river tunnel was completed after more than 30 years work and many deaths. In decompression theory, this brings us to the time when Professor Haldane,<sup>19</sup> in addition to working on decompression tables for the Admiralty, was preparing tables for compressed air work. The principle was to decompress rapidly to half the absolute pressure in 3 min and then continue at a specified linear rate to the surface. For example, after working in compressed air at 40 psig (27 m) for 6 or more hours exposure, ascend from 13 psig at 9 minutes per pound (about 2 hours decompression). The response of employers in the construction industry<sup>20</sup> could have been anticipated ...

*... are these times practical?  
... out of the question ...  
... it quite appals one to think of taking so long.*

Between 1906 and 1908 more tunnels were built under the East River in New York with twice daily exposures to pressures up to 42 psig (28 metres) and Dr Keays, the doctor there, reported<sup>21</sup> more than 3,600 bends, including 20 fatalities. He described the association of abdominal girdle pain with serious spinal cord injury and many other clinical features. Also, from a total of more than half a million decompressions, he analysed the bends percentages in the first shift (0.35%) and the second (0.72%) of the two 3-hour shifts in one day (i.e., cumulatively 1.07%), and also in more than 10,000 single eight-hour shifts (0.62%) clearly suggesting that one decompression might be better than two. However the "split shift" continued for many more years.

It then became necessary to raise the pressure in the East River tunnels to 40 psig. The shifts were 3 hours, twice daily with a 3-hour interval between them (though it is not

clear if this was a true surface interval or if it included the previous decompression time). Because prolonged decompression stops were not welcomed by the workers, the response of the contractor<sup>20</sup> was to install an intermediary lock at 28 psig, in which the workmen had to spend 5 min, and another chamber at 12 psig in which they had to spend 8 min and then take 15 min for the final decompression to surface. The intermediate stop time which was necessary was introduced without it being noticed because the two chambers were some 1,000 ft (300 m) apart in the length of the tunnel and, by the time the stragglers walking from one chamber to the next had been rounded up, the total decompression time became 48 min. They used 330 men for this deep phase, none new but only experienced workers. At first the workmen rebelled against the final 15 minute decompression, but then agreed and no fatal or serious cases resulted thereafter.

In 1912, Sir Leonard Hill published his book on *Caisson Sickness and the Physiology of Work in Compressed Air*<sup>20</sup> which was another landmark in the application of good physiological science to a complex environment. He despaired of the many *astounding hypotheses* that still abounded more than 30 years after Bert's work. He methodically demolished each of the hypotheses that he reviewed and presented experimental evidence in disproof of some. He particularly deplored the slowness of many doctors around the world to accept *the magnificent work of the French school*. Hill also reviewed the solution of the respiratory gases at pressure into the blood and their uptake into the tissues and argued the importance of duration under pressure as a factor. He also emphasised the close concordance between the results of his own experimental work and those of Haldane but was concerned that the advantages of stage over continuous decompression might not be as great as Haldane had suggested.

Hill had hoped to recoup some of the expenses of his own team's decompression work with pigs by *a sale of the victims* but found that bubbles in the fatty tissues prevented bleeding the pigs to ensure the whiteness of the meat and that, because of the pink fat, *no butcher would take a second pig from us at any price*. Hill then used two experimental subjects, Major Greenwood and himself, to achieve pressures up to 7 atmospheres (62 m, more than 200 feet) but the decompression profiles are not detailed in his book. His experiments led Hill to believe in the value of moderate exercise during decompression and, on his suggestion at the construction of the Greenwich tunnel, the men were made to climb the ladder out of the shaft immediately after decompression to atmospheric pressure.

The development of safer tables for compressed air work has continued since then in parallel with, but independently from that for diving tables, the nature of the workmen's prolonged exposures creating some distinct problems. So, for a final illustration of the relevance of compressed air workers' decompression safety to that of

diving, I will turn to an aspect of decompression injury that was first reported at the same time that the Sir Leonard Hill published his book on compressed air work.

### An occupational health hazard

Decompression sickness was now recognised as a hazard of exposure to raised environmental pressure because the illness was acute and it could be attributed to a recent decompression. But, in 1911, two papers were published, each reporting a new and more subtle threat to health.

Bassoe,<sup>22</sup> under the auspices of the Illinois State Commission on Occupational Diseases, reported 11 caisson workers with chronic joint pain and stiffness and showed an x-ray typical of arthritis deformans. Bornstein and Plate<sup>23</sup> reported 3 cases of necrosis in men who had worked in compressed air during the construction of the Elbe tunnel in Hamburg and x-rays had been taken of the symptomatic joints. (There had been one earlier report of necrosis in compressed air worker by Twynam,<sup>24</sup> but in retrospect this is considered to have been osteomyelitis not osteonecrosis.<sup>25</sup>) In the subsequent years there were many more similar papers each reporting cases of articular collapse and pain in compressed air workers who had been investigated because of their crippling joint symptoms.

The significance of this tale is that, some 40 years after these first reports, the Medical Research Council began to screen compressed air workers on major contracts in the UK in order to try and diagnose necrosis before the joint surface collapsed and so be able to remove susceptible workers from further exposure to hazard. To do this, they defined standardised radiographic procedures and used internationally agreed diagnostic criteria.

They also used the results of these surveys to monitor expected improvements in morbidity when, from time to time, the decompression schedules were changed.

The importance to divers of these extensive studies on compressed air workers, which are discussed in detail elsewhere,<sup>26</sup> is to demonstrate the application of the basic principles of occupational health to the hyperbaric environment. First, it is necessary to recognise that an injury is probably related to some occupational hazard (in this case pain due to joint collapse and a history of exposure to compressed air). Then it is necessary to assess the risk of this condition to the apparently healthy exposed population, to control the hazard perhaps by improving the decompression procedures and finally to monitor the outcome by health surveillance. For bone necrosis in compressed air workers this became a successful epidemiological investigation and the study was continued later with divers.<sup>27</sup> Although for the individual who develops dysbaric osteonecrosis it may affect the quality of life, the risk to the individual workman or diver of

acquiring necrosis was assessed by these studies as relatively small.

The significance of the original reports<sup>22,23</sup> to today's diver is that what started with a clinical problem of chronic pain in compressed air workers was followed by good epidemiology and led to measures aimed at control.

In contrast, there are other more recent studies which appear to have been started not because of a known clinical illness but maybe as interesting or speculative research using procedures accepted for clinical investigations in hospitals. The results, such as of some brain scans of divers, may indeed show some abnormalities (assuming, of course, that the limit of normality has been defined by appropriate controls) but these abnormalities are not correlated with recognised clinical manifestations. These findings, which have caused much concern among divers, are perhaps "dubious deficits in search of an unknown disease".

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## **A HISTORY OF CEREBRAL ARTERIAL GAS EMBOLISM RESEARCH: KEY PUBLICATIONS.**

Des Gorman and Simon Mitchell

### **Key Words**

Air embolism, history.

### **Introduction**

In 1982, Dr Tom Shields was asked to assemble the key references in cerebral arterial gas embolism research for the Undersea Medical Society. He did so and it is remarkable how many of these remain the key references for this subject.<sup>1</sup>

Only six key publications are chosen here. Each is discussed in the context of precedent and consequent research. One, our own, is chosen because it is the only prospective, controlled, blinded clinical study.

### **Van Allen CM, Hrdina LS, Clark J. Air embolism from the pulmonary vein. *Arch Surg* 1929; 19: 567-599<sup>2</sup>**

Van Allen, Hrdina and Clark were surgeons, at the University of Chicago and Iowa respectively, who were interested in air embolism of the pulmonary vein as a complication of lung surgery. They recognised 2 mechanisms for arterial gas embolism (AGE): direct infusion into the pulmonary vein and arterialisation of venous bubbles via a patent *foramen ovale* (PFO). The authors cited 2 key earlier references. Bichat (1808) caused pulmonary venous air embolism by blowing air into the lungs of a "living animal" at a sustained pressure greater than maximal respiratory effort. Ewald and Kobert (1883) claimed that such embolism arose through distended normal stoma and not through ruptured alveolar septa. This was thought to explain the findings of air in the left heart chambers of people who had died from drowning and hanging and in infants who had died after unsuccessful resuscitation. It was also considered a possible explanation of the brain injury seen occasionally after whooping cough.

The paper is prefaced by a series of clinical cases of iatrogenic air embolism. A series of experiments in dogs are described. Air was introduced into the pulmonary vein by way of a surgical broncho-venous fistula. The major findings are as follows.

- 1 The distribution of bubbles was determined by posture (buoyancy).
- 2 Air traps were used to show that bubbles passed from carotid arteries via the brain capillaries to the jugular veins and to the right heart. "The capillaries hinder but