13 Jarcho S. Alphonse Jaminet on Caisson Disease (1871). *Am J Cardiol* 1968; 21: 258- 260
14 Schrotter H von. *Der sauersstoff in der prophylaxe und therapie der luftdrucker- krankungen*. Berlin: Hirschwald, 1906
15 Smith A. *St Louis Med & Surg J* 1870 (3), quoted by Hill,[20] : 105
16 Moir EW. Tunnelling by compressed air. *J Soc Arts* 1896; XLIV: 567-585
17 Boycott GWM. Caisson-disease at the new high-level bridge, Newcastle-on-Tyne. *Trans Inst Civil Eng* 1906; CLXV: 231-237
18 Snell EH. *Compressed air illness or so-called caisson disease*. London: Lewis, 1896.
19 .Boycott AE, Damant GCC and Haldane JS. The prevention of compressed air illness. *J Hyg Camb* 1908; 8: 342-443
20 Hill L. *Caisson Sickness and the Physiology of Work in Compressed Air.* London: Arnold, 1912
21 Keays FL. Compressed air illness with a report of 3,692 cases. *Publ Cornell Univ Med College* 1909; 2: 1-55
22 Bassoe P. The late manifestations of compressed air disease. *Am J Med Sci* 1913; 145: 526-542
23 Bornstein A and Plate E. Über chronische Gelenkveränderungen, entstaden durch Pressluft-krankung. *Fortschr Geb Röntg Strahl* 1911-12; 18: 197-206
24 Twynham GE. A case of caisson disease. *Brit Med J* 1888; I: 190-191
25 Walder DN. Dysbaric osteonecrosis. In *Case histories of diving and hyperbaric accidents*. C L Waite. Ed. Bethesda, Maryland: Undersea Medical Society, 1988: 147-167
26 Davidson K. Dysbaric osteonecrosis. In *Aseptic necrosis of bone*. JK Davidson. Ed. Amsterdam: Excerpta Medica, 1976;147-212.
27 Elliott DH and Harrison JAB. Aseptic bone necrosis in Royal Navy divers. In *Underwater Physiology, Proceedings of the Fourth Symposium*. Lambertsen CJ. Ed. New York & London: Academic Press, 1971; 251-262

Dr David H Elliott was one of the guest speakers at the SPUMS 1996 Annual Scientific Meeting. He is Co-Editor of The Physiology and Medicine of Diving, which was first published in 1969, with the most recent edition in 1993 and is also the civilian consultant in diving medicine to the Royal Navy. His address is 40 Petworth Road, Haslemere, Surrey GU27 2HX, United Kingdom. Fax + 44-1428-658-678. E-mail 106101.1722@compuserve.com
.

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.[1]

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[2]

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

do not prevent passage."

3 Bubbles were trapped in the pulmonary arterioles and expired. "No air succeeds in traversing the pulmonary capillaries …….. gradually disappearing by excretion into the alveoli."

4 Although the cause of death in these dogs was not clearly identified, mortality was related to portal of entry (50 times more air needed if injected into the jugular vein or descending aorta as compared with the pulmonary vein) and posture (1-5 ml/kg into the pulmonary vein was lethal in head-up dogs, compared to about 15 and 30 ml/kg in horizontal and head-down dogs respectively).

5 Initial increase in systemic blood pressure with embolism. Together with air bleeding (bubbling from a stab) considered diagnostic of air embolism.

6 Spontaneous air embolism from fistula prevented by positive pressure ventilation and injection of epinephrine and ephedrine (as long as blood pressure was maintained).

These experiments were replicated in rabbits and the results reproduced almost 60 years later by Gorman and his naval research team at the University of Sydney.[3] Subsequent work by the same group at the University of Adelaide identified the following related phenomena.[4-6]

1 Infusion of air into a carotid artery causes ipsilateral embolism of the middle cerebral artery.

2 As bubbles traverse the cerebral arterioles, there is a loss of both blood flow and spontaneous and evoked brain electrical activity.

3 Most bubbles clear to the jugular vein. Clearance is usually accompanied by both a restoration of blood flow and function to normal levels. However, over the next 30 to 180 minutes, brain blood flow progressively fails and function deteriorates. These latter events are prevented by reducing the circulating number of white blood cells.

4 Bubbles that are trapped in the cerebral arterioles either do so in communicating vessels (anastamoses) or are of sufficient volume that the surface tension pressure acting on the advancing bubble interface exceeds that at the trailing interface by more than systemic blood pressure. Trapping is greatly enhanced by hypotension and is often lethal.

Polak B, Adams H. Traumatic air embolism in submarine escape training. *US Nav Med Bull* 1932; 30: 165-177[7]

Polak and Adams were US Navy medical officers involved in early US Navy submarine escape training. They drew attention to the difference between neurological decompression sickness and AGE secondary to a lung injury. Subsequently, Neuman and Bove have presented cases that show the 2 pathologies can co-exist[8] and Francis and Smith have suggested that the distinction between decompression and AGE may be somewhat artificial.[9]

Polak and Adams used a small series of dogs to measure the intratracheal pressure required to "rupture the stretched alveolar walls and drive air into the circulation." A critical pressure of about 80 mmHg was reported. The authors also showed that air embolism could be prevented by use of abdominal and thoracic binding.

These results were confirmed in 5 human cadavers 29 years later by two physicians, Malhotra and Wright, at the Royal Navy's Physiological Research Laboratories.[10] Like Polak and Adams, their interest was stimulated by submarine escape morbidity. They cite from their own experience of cases of "burst lung" in members of a submarine crew escaping from 40 fsw (12 m). Whereas intra-tracheal pressures of 73 and 80 mmHg resulted in lung trauma in 2 cadavers with either no or abdominal binding only, pressures of 190, 190 and 133 mmHg were necessary to cause such injury in 3 cadavers with both abdominal and thoracic binding.

About the same time, the critical nature of the intra-tracheal pressure was challenged by naval medical officers led by Schaefer at the US Naval Medical Research Laboratory in Connecticut.[11] The motivation for this group was common to those above and they cite low survival rates in genuine escapes from submarines. The authors also noted fatal air embolism in submarine escape trainees despite normal ascents and no indication of breath-holding. Their experiments employed dogs that were compressed (over 2.5 and 4 minutes) to 100 (30 m) and 200 fsw (60 m) in a recompression chamber (RCC), kept at pressure for 1 minute and then decompressed to the surface over I minute. Interstitial pulmonary emphysema and AGE were seen in dogs that had their trachea occluded by either a solenoid valve or a scissors clamp. These events were again encountered beyond intra-tracheal pressures of 80 mmHg. Both the lung injury and the AGE could be prevented by thoracic and abdominal binders despite increases in intra-tracheal pressures in excess of 180 mmHg. It was shown that, while these binders did not prevent this increase, they did maintain both trans-pulmonic (intra-tracheal minus intra-pleural pressure) and trans-atrial pressure (intra-tracheal minus intra-atrial pressure). Both the latter pressures are considered to be measures of lung distension and hence were argued to be the critical determinants of injury. A threshold of about 60 mmHg for both was measured. Twenty years later this concept was reinforced by Professor Colebatch at the University of New South Wales.[12] His findings are cited in more detail below.

The equivalent British experience with AGE in submarine escape training has been described many times, but the report by Elliott, Harrison and Barnard is still the definitive report.[13] This report showed that many of those trainees who suffered AGE did not have any real evidence

of lung injury and some AGE victims who appeared to recover after recompression subsequently relapsed. The cause of such relapses is still controversial. More recently, in a prospective study at the same facility, Brooks and his Royal Navy colleagues could not identify a good correlation between lung function as measured by spirometry and risk of AGE.[14]

Macklin M, Macklin CC. Malignant interstitial emphysema of the lungs and mediastinum as an important occult complication in many respiratory diseases and other conditions. An interpretation of the clinical literature in the light of laboratory experiment. *Medicine* 1944; 23: 281-358[15]

Madge and Charles Macklin wrote and described what is an exhaustive review and study of lung disease resulting in extrapulmonary release of gas. Their concern was based on patients who developed such a state, usually after some chest infection. They suggested that toxins of some infectious diseases (and especially influenza) predisposed to lung injury. Other patients were thought to have a constitutional weakness of their alveolar walls.

The Macklin's proposed 3 causes of pulmonary injury resulting in escape of air.

1 Atelectasis of some part of the lung followed by hyperinflation in adjoining regions of the same lung or in the opposite lung.
2 General over-inflation with or without increased intra-alveolar pressure.
3 Reduced blood supply to the pulmonary vessels either with increased intra-alveolar pressure or with hyperinflation.

Air was considered to become entrapped in lung tissue by escaping through ruptured alveolar bases into the sheaths of the pulmonary vessels. Such air was then though to have the following eventual destinations.

1 Retroperitoneum and peritoneum.
2 Mediastinum.
3 Subcutaneous tissues of the face, neck, chest, axillae and body.
4 Thoracic cavity.
5 Pericardium.

Although the Macklin's did not specifically address air escape into blood, their theories are still considered central in arguments about the likely pulmonary lesion in divers and submariners who suffer AGE.

Whereas the mechanism of lung injury causing AGE in chest surgery and after some chest infections can be obvious, the type of lung lesion that underlies AGE after decompression or as a consequence of mechanical ventilation remains controversial. Risk is not well predicted by simple lung function[14] and many victims of AGE after a decompression were seen to ascend normally and to not hold their breath.[11,13] One suggested mechanism for such lung injury, which is consistent with the Macklin's proposals, is that lung may tear as a result of shearing forces generated within tissues of heterogeneous compliance.[12] This is most likely in inspiration and is supported by a recent observation that apparently normal lungs can rupture during a sustained deep breath. [16]

de la Torre E, Meredith J, Netsky MG, Winston-Salem NC. Cerebral air embolism in the dog. *Arch Neurol* 1962; 6: 307-316 [17]

A neurosurgeon, two cardiac surgeons and a neurologist from the US Public Health Service were stimulated to study AGE because of the multiplicity of different surgical procedures that are complicated by such embolism. They noted the high tolerance for venous air embolism because of the filtering capacity of the pulmonary circulation and suggested an important role for any PFO. They also remarked on the importance of posture with respect to distribution of arterial bubbles.

Air and other gases were injected directly into the carotid artery of dogs. Their key results are as follows.

1 Whereas about 0.4 ml of air, helium or nitrogen gas infusate was lethal, 1 ml of oxygen and 2 ml of carbon dioxide were needed to kill the dogs.
2 No effect was noted for this type of infusion on the ECG.
3 Ischaemic infarcts were seen in the ipsilateral (to the infusion) distribution of the middle cerebral artery. "The physiologic and anatomic effects are generally less with air than solid emboli in part because air bubbles can enter the venous system by passing through the capillaries and because air is less damaging to blood vessels."
4 Morbidity and mortality increased with increased volume of air foam infusate.
5 The factor that correlated best with outcome was the degree of increase in the cisternal pressure. "Death occurs within 48 hours from increased intra-cranial pressure."

These relative risks for different gases were subsequently confirmed in studies on rabbits at the University of Sydney.[3]

Ischaemic infarcts at the junction of the grey and white matter of the brain, along with punctate haemorrhages, were also demonstrated in more recent but similar experiments in dogs at the United States Navy's Medical Research Institute (NaMRI).[18]

However, there is now considerable evidence from

both NaMRI and the University of New England (Australia) that bubbles have a significant deleterious effect on the endothelium and blood, and cause a temporary loss of the blood-brain barrier.[19,20] There are no data to show any correlation between a consequent increase in brain water content (brain oedema) and outcome in either animals or humans after AGE. However, there are data from both North American and Australian groups that show outcome (restoration of blood flow and evoked brain responses) is greatly enhanced if white blood cell behaviour is modified after AGE.[4,21]

Waite CL, Mazzone WF, Greenwood ME, Larsen RT. *Cerebral air embolism: 1. Basic studies*. USN SMRL Report 493, 1967[22]

These US Navy officers used a canine model to identify an effective treatment pressure for AGE. Their report is prefaced with a review of the risks of AGE after the different types of escape practised in the US Navy (see Table 1).

Other observations from the dogs that were compressed included the following.

"In every instance, there was evidence of a change in bubble size and partial restoration of the circulation just beyond 33 feet (10 m). In none of the experiments were intravascular bubbles seen to persist after pressure equivalent to four atmospheres (30 m) was reached. Equally important, in no instance was there a reappearance of bubbles during or after decompression….."

All these observations were subsequently confirmed by Gorman and his colleagues at the University of Sydney.[3,23] First, many trapped bubbles were seen to be cleared from brain arterioles by systolic pressure and especially during the period of systemic hypertension that usually follows AGE. Second, bubble clearance from the brain during recompression occurred with the first doubling of pressure and there was no real difference in efficacy in this context between compressions to 2 ATA, 2.8 ATA, 4 ATA, 6 ATA and even 11 ATA.

TABLE 1

Technique	1930-1953 SEA Momsen lung	1942-1957 Free ascent	1957-1965 Buoyant ascent	1963-1965 Steinke Hood
Number of ascents	193,000	17,583	130,679	32,679
Morbidity	0.004%	0.09%	0.009%	0.015%
Mortality	0.0005%	0.01%	0.0008%	0

The experimental model was based on anaesthetised dogs with surgically implanted cranial windows. The type of anaesthesia used was not described and no data were provided about the dogs' blood pressure. Air (1 to 7 ml) was injected into the carotid artery. Five dogs were kept at sea-level. Two died and 3 were left with sequelae. Three dogs were embolised at sea level and then compressed in a RCC to 165 fsw (50 m). The final 3 dogs were embolised at 2 atmospheres absolute (10 m) decompressed to sea-level and then compressed to 165 fsw (50 m). After 10 minutes at this pressure, the dogs were decompressed in accordance with a US Navy standard air diving table (170 fsw for 10 minutes). One of these dogs showed complete clearance of bubbles from the field of view before compression beyond 60 fsw (18 m), 3 by 80 fsw (24 m)and the remainder by 100 fsw (30 m). Five survived without sequelae. The sixth had a brain haemorrhage and suffered sequelae. Other observations from the dogs that were not compressed included the following.

1 Circulatory arrest occurred in small arterioles (30 to 60 μ).
2 "In other small arteries there was a slow pulsating progression of the bubble in response to the systolic pressure peaks."

Cerebral protection by lidocaine during cardiotomy. Mitchell SJ, Pellett O, Gorman DF. *Undersea and Hyperbaric Medicine* 1998; 25 (Suppl): 22.[24]

The preservation of neuroelectrical function in cats premedicated with the class Ib antiarrhythmic agent lignocaine (lidocaine in the USA) prior to experimental AGE was first described by Evans and his research group at the NaMRI in 1984.[25] The same group later demonstrated a similar benefit when lignocaine was administered "therapeutically".[26] This work was extended by others using in vivo models of AGE, focal, and global brain ischaemia. Lignocaine in standard antiarrhythmic doses was variously demonstrated to preserve neuroelectrical function and cerebral blood flow, and to reduce cerebral oedema and infarct size. Possible mechanisms for this cerebral protection by lignocaine were reviewed by Mitchell as a preface to the principal study described here and include: deceleration of ischaemic ion fluxes, reduction of cerebral metabolic rate and modulation of leucocyte activity.[27] Subsequent to that review, lignocaine has also been reported to reduce ischaemic excitotoxin release.[28] Several case reports have been published which claim benefit from lignocaine in decompression illness.

Patients undergoing cardiac surgery involving cardiopulmonary bypass often suffer post-operative neuropsychological (NP) impairment and several studies have implicated cerebral arterial emboli as the primary cause. We investigated the effect of lignocaine on brain function in patients undergoing left heart valve surgery; a group known to have a high incidence of post-operative NP impairment and where the elective nature of the surgery allows comprehensive pre-operative assessment. Consequently, patients were used as their own controls.

Fifty five patients completed 11 pre-operative NP tests, a self rating inventory for memory and inventories measuring depression and anxiety. These were repeated 10 days, 10 weeks and 6 months post-operatively. Patients received a 48-hour double-blinded infusion of either lignocaine in a standard anti-arrhythmic dose or placebo, beginning at induction of anaesthesia. The difference between each patient's pre and post-operative scores was calculated. A deficit in any test was defined as decline by ≥ 1 standard deviation of the pre-operative group mean for that test. Pre-operative scores were also normalised and sequential post-operative percentage change scores were also calculated for each patient in all tests and inventories.

Significantly more placebo patients had a deficit in at least one NP test at 10 days ($p < 0.025$) and 10 weeks ($p < 0.05$). The lignocaine group performance (sequential changes in normalised scores) was significantly "better" in 6 of the 11 NP tests ($p < 0.05$) and the memory inventory ($p < 0.025$). There were no group differences in the remaining NP tests or the depression and anxiety inventories. No important confounding differences between the groups were identified. In particular, there was no difference between the groups in terms of total operative emboli exposure or hypotension.

These data show persistent cerebral protection by lignocaine, which is unrelated to any effect on depression or anxiety, and is at a level that is noticed by the patients.

References

1 Shields TG. Gas embolism. In *Key documents of the biomedical aspects of deep-sea diving selected from the world's literature 1608-1982. Volume III.* Bethesda, Maryland: Undersea Medical Society, 1983; III-9-1 to III-9-184

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

3 Gorman DF, Browning DM, Parsons DW and Traugott FM. Distribution of arterial gas emboli in the pial circulation. *SPUMS J* 1987; 17: 101-115

4 Helps SC and Gorman DF. Air embolism of the brain in rabbits pre-treated with Mechlorethamine. *Stroke* 1991; 22: 351-354

5 Helps SC, Meyer Witting M, Reilly PL and Gorman DF. Increasing doses of intracarotid air and cerebral blood flow in rabbits. *Stroke* 1990; 21: 1340-1345

6 Helps SC, Parsons DW, Reilly PL and Gorman DF. The effect of gas emboli on rabbit cerebral blood flow. *Stroke* 1990; 21: 94-99

7 Polak B and Adams H. Traumatic air embolism in submarine escape training. *US Nav Med Bull* 1932; 30: 165-177

8 Neuman TS and Bove AA. Combined arterial gas embolism and decompression sickness following no stop dives. *Undersea Biomed Res* 1990; 17: 429-436

9 Francis TJR and Smith D. *Describing dysbarism*. Bethesda, Maryland: Undersea Medical Society, 1991

10 Malhotra MS and Wright HC. The effects of raised intrapulmonary pressure on the lungs of fresh unchilled cadavers. *J Path Bact* 1961; 82: 198-202

11 Schaefer KE, McNulty WP, Carey C and Liebow AA. Mechanisms in development of interstitial emphysema and air embolism on decompression from depth. *J Appl Physiol* 1958; 13(1): 15-29

12 Colebatch HJH, Smith MM and Ng CKY. Increased elastic recoil as a determinant of pulmonary barotrauma in divers. *Resp Physiol* 1976; 26: 55-64

13 Elliott DH, Harrison JAB and Barnard EEP. Clinical and radiological features of 88 cases of decompression barotrauma. In *Proceedings VIth Symposium on Underwater Physiology*. Bethesda, Maryland; Federation of American Societies for Experimental Biology, 1978; 527-536

14 Brooks GJ, Pethybridge RJ and Pearson RR. Lung function reference values for FEV_1, FEV_1/FVC ratio and FEF25-75 derived from the results of screening 3788 Royal Navy Submarine candidates by spirometry. In *XIVth Annual Meeting of the European Undersea Biomedical Society*. Aberdeen, 1988; paper number 13

15 Macklin M and Macklin CC. Malignant interstitial emphysema of the lungs and mediastinum as an important occult complication in many respiratory diseases and other conditions. An interpretation of the clinical literature in the light of laboratory experiment. *Medicine* 1944; 23: 281-358

16 Francis TJR and Gorman DF. Pathogenesis of the decompression disorders. In: *The physiology and medicine of diving. 4th edition.* Bennett PB and Elliott DH. Eds. London: Saunders, 1993; 454-480

17 de la Torre E, Meredith J, Netsky MG and Winston-Salem NC. Cerebral air embolism in the dog. *Arch Neurol* 1962; 6: 307-316

18 Dutka AJ, Kochanek PM and Hallenbeck JM. Air embolism may cause unrecognized ischaemia of the grey-white junction. *Undersea Biomed Res* 1988; 15: 99-106

19 Hallenbeck JM et al. Polymorphonuclear leukocyte accumulation in brain regions with low blood flow

during the early postischemic period. *Stroke* 1986; 17: 246-253
20 Hills BA. Microbubble damage to the blood-brain barrier: Relevance to decompression sickness. *Undersea Biomed Res* 1991; 18: 111-116
21 Dutka AJ, Kochanek PM and Hallenbeck JM. Influence of granulocytopenia on canine cerebral ischemia induced by air embolism. *Stroke* 1989; 20: 390-395
22 Waite CL, Mazzone WF, Greenwood ME and Larsen RT. *Cerebral air embolism: 1. Basic studies*. USN SMRL Report 493, 1967
23 Gorman DF, Browning DM and Parsons DW. The redistribution of cerebral arterial gas emboli: A comparison of treatment regimens. In *Proceedings IXth Symposium on Underwater and Hyperbaric Physiology*. Bethesda, Maryland: Undersea Medical Society, 1987; 1031-1054
24 Cerebral protection by Lignocaine during cardiotomy. Mitchell SJ, Pallett O and Gorman DF. *Annals of Thoracic Medicine* 1998 (in press)
25 Evans DE, Kobrine AI, LeGrys DC and Bradley ME. Protective effect of lignocaine in acute cerebral ischaemia induced by air embolism. *J Neurosurg* 1984;60:257-63
26 Evans DE, Catron PW, McDermott JJ, Thomas LB, Kobrine AI and Flynn ET. Effect of lignocaine after experimental cerebral ischaemia induced by air embolism. *J Neurosurg* 1989;70:97-102
27 Mitchell SJ. The role of lignocaine in the treatment of decompression illness: a review of the literature. *SPUMS J* 1995;25:182-194
28 Fujitani T, Adachi N, Miyazaki H et al. Lignocaine protects hippocampal neurons against ischemic damage by preventing increase of extracellular excitatory amino acids: a microdialysis study in Mongolian gerbils. *Neurosci Lett* 1994; 179: 91-4

Professor D F Gorman FAFOM, PhD, is Head, Occupational Medicine, Faculty of Medicine and Health Sciences, University of Auckland, New Zealand. He is the immediate Past-President of SPUMS. His address is 52 Albert Road, Devonport, Auckland 9, New Zealand. Telephone + 64-9-373-7599. Fax + 64-9-308-2379. E-mail d.gorman@auckland.ac.nz .

Dr Simon Mitchell, MB ChB, Dip DHM, was the Director of the Slark Hyperbaric Unit at the Royal New Zealand Navy Hospital, Naval Base, Auckland, when this paper was presented. His address now is Director, Wesley Centre for Hyperbaric Medicine, Wesley Hospital, Suite 53 Sandford Jackson Building, Auchenflower, Queensland 4066, Australia. Telephone +61-(0)7-3371-6033.

Correspondence should be addressed to Professor Gorman.

THE DIVING "LAW-ERS" A BRIEF RESUME OF THEIR LIVES.

Chris Acott

Key Words
History, physiology.

Introduction

Early diving and diving medicine researchers were not necessarily involved with diving, some were mathematicians, philosophers, scientists and astronomers while others were physiologists and medical clinicians. Hence the history of diving and diving medicine has involved many seemingly unrelated disciplines. However, many of the researchers lives and research work were intertwined. As an example, John Dalton, an astronomer, after whom Dalton's Law was named, first published on colour blindness (which is why it was originally called 'Daltonism'). The genetics of this disorder were first studied by J B S Haldane (the son of J S Haldane). JBS Haldane was involved with both the Royal Navy Admiralty's Deep Diving Units (the first with his father and the second with L Hill, R Davis and K Donald). J S Haldane designed the first "safe" decompression tables.

A menagerie of animals, divers, "volunteers" and self experimentation were used by the early diving medicine researchers. Boyle used a viper; Bert used birds, dogs, cats and many other animals; J S Haldane used goats; L Hill used frogs but was also involved in self experimentation; K Donald used naval divers and other "volunteers". Much of the actual original diving/diving medicine research demonstrated ingenuity, lateral thinking and unique observation skills.

Unfortunately, many of the original documents are impossible to find and reliance is therefore on second hand interpretation.

This paper is a brief outline of the lives of the originators of the physical laws of diving (Archimedes and Pascal's Principles; Poiseuille and Laplace's equations; the Laws of Boyle, Henry and Dalton).

Archimedes (287-212 BC)

Archimedes was the pre-eminent Greek mathematician and inventor, who wrote important works on plane and solid geometry, arithmetic and mechanics. He was born in Syracuse, Sicily (the exact date is not known, however, popular belief has it as 287 BC) and was educated in Alexandria, Egypt. In pure mathematics he anticipated many of the discoveries of modern science, such as the