

25. Kizer, K.W. Dysbaric cerebral air embolism in Hawaii. *Annals. Emerg. Med.* 1987; 16(5): 535-541.
26. Melamed, Y., Sherman, D., Wiler-Ravell, D., Kerem, D. The transportable recompression chamber as an alternative to delayed treatment in serious diving accidents. *Aviat Space Environ. Med.* 1981; 52(8): 480-484.
27. Murphy, B.P., Cramer, F.S. Results of hyperbaric oxygen therapy in 43 cases of cerebral air embolism. In: (Programs and abstracts) *Aerospace Med. Assoc. Scientific Program*; San Diego, California; 1984.
28. Francis, T.J.R., Dutka, A.J. The influence of latency on the outcome of spinal cord decompression sickness. *Undersea Biomed. Res.* 1988; 15 (Suppl.): 77.
29. Ward, C.A., Yee, D., McCullough, D., Stanga, D., Fraser, W.D. Complement proteins mediate decompression sickness in rabbits. *Undersea Biomed. Res.* 1987; 14(2) Suppl.: 16.
30. Lauckner, G.R., Nishi, R.Y. Canadian forces air decompression tables. *Defence and Civil Institute of Environmental Medicine Report No. 85-R-03*. Downsview, Ontario, Canada: DCIEM, 1985.
31. Weathersby, P.K., Survanshi, S.S., Hays, J.R., MacCallum, M.E. Statistically based decompression tables III: Comparative risk using US Navy, British, and Canadian standard air schedules. *US Navy Medical Research Institute Report NMRI 86-50*. Bethesda, Maryland: NMRI, 1986.
32. Reid, M.A., Runciman, W.B., Illsley, A.H., et. al. Circulatory and respiratory kinetics of nitrous oxide in the sheep. *Clin. and Exp. Pharmacol. and Physiol.* 1988 (in press).
33. Hempleman, H.V. British decompression theory and practice. In: Bennett, P.B., Elliott, D.H., eds. *Physiology and Medicine of Diving and Compressed Air Work (First Edition)*. Balliere Tindall and Cassell, London, 1969.
34. Gorman, D.F., Parsons, D.W. Decompression meters; philosophical and other objections. *SPUMS J* 1987; 17(3): 119.
35. Catron, P.W., Hallenbeck, J.M., Flynn, E.T., Bradley, M.E., Evans, D.E. Pathogenesis and treatment of cerebral air embolism and associated disorders. *Naval Medical Research Institute Report 84-20*. Bethesda, Maryland 1984.
36. Dutka, A. A review of the pathophysiology and potential application of experimental therapies for cerebral ischaemia to the treatment of cerebral arterial gas embolism. *Undersea Biomed. Res.* 1985; 12: 404-423.
37. de la Torre, E., Mitchell, O.C., Netsky, M.G. The seat of respiratory and cardio-vascular responses to cerebral air emboli. *Neurol.* 1962; 12: 140-147.
38. Evans, D.E., Kobrine, A.I., Weathersby, P.K., Bradley, M.E. Cardio-vascular effects of cerebral air embolism. *Stroke* 1981; 112: 338-344.
39. Greene, K.M. Causes of sudden death in submarine escape training casualties. In: Hallenbeck, J.M., Greenbaum, L.J. Jr., eds. *Workshop on arterial air embolism and acute stroke*. Bethesda, M.D.: Undersea Medical Society, 1977: 8-13.
40. Van Allen, C.M., Hrdina, L.S., Clark, J. Air embolism from the pulmonary vein - a clinical and experimental study. *Arch. Surg.* 1929; 19: 567-599.

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DIVING ACCIDENTS WHAT IS THE MAGNITUDE OF THE PROBLEM IN NEW ZEALAND ?

Allan F.N. Sutherland

Diving is one of the fastest growing sports with the New Zealand Underwater Association (PADI Franchise) now claiming that 5% of New Zealand's adult population has been either trained, or is undertaking scuba diving from training numbers and tank inspections. This figure is twice the percentage quoted from Australia of 2.5% and higher than that of other countries. In New Zealand, the numbers of both deaths and accidents requiring hyperbaric oxygen, recompression therapy, are on the increase. Last year there were 13 deaths and 35 recompression therapies (30 in Auckland) with no accurate count of the number of serious

ear problems or other major scuba diving injuries. But from experience, there must have been many times more with decompression therapies being but the tip of the iceberg. Consequently diving medicals, accident prevention and treatment are areas of medical attention by doctors interested in Sports Medicine. So far in 1988 (May) there have been 7 deaths (scuba) and 15 recompression.

At a recent course at the Philomel Naval Hospital conducted by Dr Des Gorman, a Hyperbaric Medicine Specialist from the Royal Adelaide Hospital in South Australia, the physics, physiology and mechanisms of air embolus and decompression sickness were clearly presented, as were other diving medical problems related to lung and ear.

Air embolus is an intravascular collection of air or other respired gas resulting from barotrauma to lungs usually after a rapid ascent from depth. The exact site of this intravascular gas entry in the lungs is only rarely accurately located. The resultant gas can pass to vital areas such as the cerebral circulation if the person is head up and the coronary circulation if prone. Cerebral arterial gas embolism, CAGE, causes loss of consciousness and other neurological symptoms. The natural history of CAGE is that some cases spontaneously resolve, regaining consciousness if unconscious, as the gas embolus passes through the cerebral circulation.

Decompression sickness (DCS) is now thought to be a tissue disease rather than a vascular disease with nitrogen dissolving more slowly out of some tissues, especially fatty tissue, neural tissue and myelin sheaths at a rate slower than it can be cleared and thus bubbles are formed. It is these bubbles which cause local tissue effects of local compression, evoking chemical effects and rupturing into blood vessels. The single and multi tissue models of nitrogen off-gassing used by decompression tables and decompression meters have little relevance except as an empirical model when one considers the multiplicity of tissues which are off-gassing and at varying rates. Doppler studies show intravascular bubble formation in most divers who have dived below 30 feet. These venous bubbles usually clear in the circulation at the lung unless there is an arterial-venous connection and momentary back flow, e.g. Atrial septal defects are potentially patent in 20% of the population.

Intravascular bubbles, be they air embolus or decompression sickness in origin, not only can cause immediate intravascular effects, but can become lined by surfactant produced in the lungs making these bubbles stable. This may explain why delayed signs and symptoms, especially with DCS, present many days after exposure.

Any patient presenting with unusual signs or symptoms following a scuba dive should have a careful history and examination with the physician considering a scuba diving cause. The first aid management is as follows for acute dive accidents:

1. A, B, C, resuscitation.
2. Head down 30° left lateral.
3. Give fluids, preferably intravenous and carefully record fluid balance.
4. Give oxygen at maximal rate, carefully recorded.
5. Obtain diving medical advice re resuscitation, diagnosis and retrieval to an appropriate treatment site.

THE DIVER EMERGENCY SERVICE

This toll-free, New Zealand-wide, telephone number, paid for by the New Zealand Underwater Association, located at the Philomel Naval Hospital is (09) 458-454. South Island cases being referred to Christchurch (03) 792-900.

This is a summary of a paper presented to the International Sports Medicine Meeting held in New Zealand 12th to 15th May 1988

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HOW DO AMERICAN DIVERS DIE?

A Review of the Scuba Diving Fatalities in the USA in 1985

John Lippmann

This review is a summary of an extensive report titled "U.S. Underwater Diving Fatality Statistics, 1985" issued by the National Underwater Accident Data Centre (NUADC) at the University of Rhode Island.

The full report addresses two distinct types of underwater fatality. The first is the Non-occupational fatality, which includes all fatalities of a sport or recreational nature which occurred while using scuba (or in a few cases, some other type of underwater breathing system). The second type of underwater diving fatality is titled Occupational, and addresses fatalities associated with professional, commercial and military diving.

This review will only consider the Non-occupational fatalities since these are far more relevant to the sport diver.

NUADC defines an active diver as one who dives at least three times per year, and estimates that in 1985 there