Mitchell: I think they were using swimming aids, and were diver trainees like Dr Walker's diver.

Davis: It would be worth reviewing the old long-distance swimming physiology work to see whether it is documented anywhere. It is the use of swimming aids in one form or another that perhaps creates a different respiratory and cardiovascular workload which someone swimming without aids in cold water for long periods does not experience. Another interesting aspect is that many of these swimmers were not youngsters; Gerry Forsberg broke the two-way English Channel record in his late forties, for instance.

von Neullen, Holland: Dr Mitchell, could a contaminated air source be a cause of pulmonary oedema, and have you ever seen such cases?

Mitchell: You are absolutely right. If you inhale irritants or noxious substances you can definitely suffer pulmonary oedema. The air in this patient's cylinder was tested and no contaminants were found. So it was not the problem in her case. I personally have not seen any diver suffer pulmonary oedema because of contaminants in the breathing mix but I believe such cases have been reported in the literature.

Thomas: Do you know if they routinely test for oxides of nitrogen in the air? (**Mitchell:** I do not think so.) These are described as a cause of pulmonary oedema. Her air was probably tested for carbon monoxide and hydrocarbons as it was a diving situation. Presumably though, you would expect a large number of people to be affected if the system was contaminated.

Deborah Yates, Sydney: It is highly unlikely that they would have been able to test for oxides of nitrogen because it is difficult to get hold of the equipment. We have recently seen some cases in blast furnace workers, but we had considerable difficulty measuring levels. Quite variable levels were seen (paper in preparation).

Thomas: If you measure the levels of nitric oxide in hospital compressed air they vary with the urban pollution. We have recorded up to about 300 parts per billion in our hospital air. This is probably not important, but you do not know what else is being compressed out of the atmosphere to give to the patients. Oxides of nitrogen have been described as being present in ventilator circuits in intensive care units.

References

1 Pearn JH. Secondary drowning involving children. *BMJ* 1980; 281: 1103-1105

2 Modell JH, Graves SA, Ketover A. Clinical course of 91 consecutive near-drowning victims. *Chest* 1976; 70: 231-238

3 Davis FM, Warner M, Ward B. Snorkelling and scuba diving drownings in New Zealand, 1980–2000. *SPUMS J* 2002, 32: 70-80

4 Shupak A, Weiler-Ravell D, Goldenberg I, Halpern P, Shoshani O, Margolis A. Strenuous swimming-induced hemoptysis and pulmonary oedema. *Undersea Hyperb Med* 1995; 22(Suppl): A50

5 Pugh LGCE, Edholm OG, Fox RH, et al. A physiological study of channel swimming. *Clin Sci* 1960; 19: 257-273

Salt water aspiration syndrome

Simon Mitchell

Key words
Salt water aspiration syndrome, near drowning, diving, scuba diving

Abstract
A "salt water aspiration syndrome" has been described in scuba divers. It is characterised by early cough, a latent period of one to 15 hours, followed by a productive cough, retrosternal chest pain, dyspnoea, shivering, nausea, vomiting and constitutional symptoms such as malaise and fever. It could be a mild form of near drowning, a reaction to the inhalation of irritants or micro-organisms, or due to occult nebulisation of salt water.

Salt water aspiration syndrome (SWAS) was first described by Carl Edmonds.[1] He described 30 cases in military divers characteriaed by an early post-dive cough and then, after a latency which might be one to 15 hours, a productive cough, dyspnoea, shivering, nausea, vomiting and various constitutional symptoms such as malaise and fever. One of the defining symptoms was retrosternal pain, which he reported as being present in 90% of divers. Typical physical findings were lung crepitations and patchy consolidation on the chest X-ray in 50% of patients. The cases reported all recovered over 24 hours with treatment on 100% oxygen and little else. Since then, there have been various anecdotal

case reports, including from South Australian abalone divers (Acott C, personal communication) who believe they suffer SWAS from time to time.

The pathophysiology of SWAS is probably similar to that of near drowning, but at a lower level of severity. For example, the presence of salt water in the alveoli might dilute or destroy pulmonary surfactant, thus causing atelectasis. This, in turn, would cause a cough and possibly dyspnoea, and the patchy changes that are seen on chest X-ray. These radiological changes differ from the typical pulmonary oedema pattern most commonly seen in near-drowning cases. Hypertonic saline (sea water) and the various irritants, particulate matter or marine organisms contained in it, may elicit an inflammatory response. This might further contribute to alveolar collapse and also might explain the fever and malaise. The delay in developing such a response could account for the latent period described by Edmonds. Hypertonic salt water may also exhibit an osmotic attraction of fluid into the alveolar space as described in this issue by North.[2]

Given the obvious similarities with near drowning, what then are the differences between SWAS and near drowning? It may simply be a matter of nebulised water volume. Near drowning is considered to involve the aspiration of moderate amounts of water into the lungs and does not typically involve the systemic symptoms with a long latency described by Edmonds. Salt water aspiration syndrome probably involves more subtle or even occult aspiration of which the diver might not be aware. Such aspiration might occur in those situations typically associated with near drowning, such as panic at the surface, out of air situations, loss of the regulator and any unconsciousness when immersed.

Subtle or occult aspiration might occur via nebulisation of salt water with a leaky regulator diaphragm, a leaky exhaust valve or mouthpiece tears. Such equipment problems do arise, and most experienced divers have noticed the slightly wetter feeling of breathing air under those circumstances. Whether or not this results in an aspiration syndrome is not clear. The nebulisation of droplets to traverse the airways down into the respiratory tissue is an exacting science. Nevertheless, it seems plausible that a regulator nebulising water particles across a wide range of sizes will produce some of the right size to pass into the distal airways.

Another issue that arises is whether pathogenic organisms might be involved. Dr Jones alluded to this in the discussion with respect to Key West scuba divers disease (page 204, this issue). An alternative infectious theory has been proposed by Bradley and others.[3] They described a similar syndrome, although typically with a longer latency and less shortness of breath, that was attributed to contamination with various organisms, most commonly *Pseudomonas spp.*, and their endotoxins. These were isolated from the breathing circuits of diving equipment. One of the cases reported died due to *Pseudomonas* infection.

Salt water aspiration syndrome may be a heterogeneous collection of problems with different causes but similar manifestations. It could be a mild form of near drowning, a reaction to the inhalation of irritants or, indeed, be due to occult nebulisation of salt water. It is conceivable that this was true even for the cohort of divers described originally by Edmonds.[1] This author has only very rarely seen cases where the classic pattern of SWAS was followed. This may be because of spontaneous recovery and under-reporting; in other words, these divers might often not come to the attention of diving physicians.

References

1 Edmonds C. Salt water aspiration syndrome. *Mil Med* 1970; 135: 779-785
2 North R. The pathophysiology of drowning. *SPUMS J* 2002; 32: NNN
3 Bradley ME, Bornmann RC. SCUBA disease revisited. In: Bachrach AJ, Matzen MM (eds). *Underwater physiology VIII. Proceedings of the Eighth Symposium on Underwater Physiology*. Undersea and Hyperbaric Medical Society, Bethesda: 1984: 173-180

Dr Simon Mitchell, MB, ChB, DipDHM, DipOccMed, PhD, is a consultant in diving and hyperbaric medicine at the Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital,
Randwick, New South Wales 2031, Australia.
Phone: +61-2-9382-3883
Fax: +61-2-9382-3882
E-mail: <dr.m@bigpond.com>

Audience participation:

Knight, Melbourne: At the time Carl Edmonds described this syndrome, the Royal Australian Navy was using the Porpoise Regulator. During the divers' training they were expected to disassemble and reassemble it while holding their breath. I have had salt water aspiration twice. Once was on the diving medical officers course. After buddy breathing in a salt water swimming pool, three of us developed symptoms overnight. We all improved on oxygen. The next time was in Thailand. There was a lot of particulate matter in the sea and my regulator had a faulty exhaust flap valve. My own view of the aetiology is that the cause is primarily particulate matter in the water, especially plankton, combined with a wet regulator.

Thomas, Sydney: In subjects who have inhaled 4.5% saline in challenge tests, we have demonstrated an airway neutrophilic infiltrate at 18-24 hours. This can be associated with an increase in airway reactivity. In addition, salt water would induce a mild peripheral inflammation and restrictive lung pattern. Thus, this challenge would be similar to the process experienced by those with salt water aspiration, who probably also have a neutrophilic inflammatory lung infiltrate. It can be very mild and disappear within 24 hours.