

clear, limited diving may be permitted to a maximum depth of 18 metres without any free ascent practice.

References

- 1 Wachholz C. *Analysis of DAN member survey*. DAN Report, 1988
- 2 Edmonds C and Walker D. Scuba diving fatalities in Australia and New Zealand. Part 1. The Human Factor. *SPUMS J* 1989;19(3): 94-104.

Some other references relevant to asthma in diving

- Adolfson JA and Lindemerk C. Pulmonary and neurological complications in free escape. *Forsvarsmedicin* 1973; 9(3): 244-246
- Calder IM. Autopsy and experimental observations on factors leading to barotrauma in man. *Undersea Biomed Res* 1985; 12(1): 165-182
- Colebach HJH, Smith MM and NG Cky. Increased elastic recoil as a determinant of pulmonary barotrauma in divers. *Resp Physiol* 1976; 26: 55-64
- Elliott DH, Harrison JAB and Barnard EPP. Clinical and radiological features of 88 cases of decompression barotrauma. In Shilling CW and Beckett MW eds. *Proceedings of Sixth Underwater Physiology Symposium* Bethesda, Maryland; FASEB, 1978
- Hoff EC. *A Bibliographical Source Book of Compressed Air, Diving and Submarine Medicine. Vol 1* Washington, DC; Bu Med, Dept of Navy, 1948
- Macklin MT and Macklin CC. Malignant interstitial emphysema of the lungs and mediastinum. *Medicine* 1944; 23: 281-358
- McAniff JJ. *United States Underwater Diving Fatality Statistics/1970-79*. Washington DC; US Department of Commerce, NOAA Undersea Research Program, 1981
- McAniff JJ. *United States Underwater Diving Fatality Statistics/1986-87. Report number URI-SSR-89-20*, University of Rhode Island, National Underwater Accident Data Centre, 1988
- Malhotra MS and Wright HC. The effects of a raised intratracheal pressure on the lungs of fresh unchilled cadavers. *J Path.Bact* 1961; 82: 198-202
- Polak IB and Adams H. Traumatic air embolism in submarine escape training. *U.S. Navy Med Bull* 1932; 30:165
- Reed CE. Editorial. Changing Views of Asthma. *Sandoz Med Sci* 1988; 27(3): 61-66
- Shilling CW, Carlston CB and Mathias RA. editors *The Physicians Guide to Diving Medicine*. New York: Plenum Press, 1984
- Walker D. Reports on Australian and New Zealand diving fatalities. Serially presented in *SPUMS J* 1980-88

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A NEW CLASSIFICATION FOR THE DECOMPRESSION ILLNESSES.

Report on a workshop held at the Institute of Naval Medicine, Alverstoke, United Kingdom, October 1990

Des Gorman

Recently the Royal Navy Institute of Naval Medicine (INM) sponsored a 2 day workshop at the Institute to develop a new classification for the decompression illnesses. This was prompted by an attempt at organising a multi-centre trial of lignocaine in cerebral arterial gas embolism (CAGE), which foundered when diagnostic criteria for CAGE could not be agreed. Funding for the workshop was provided by INM and 35 delegates were invited and attended; including the author, Carl Edmonds, David Elliott, James Francis, Tom Shields, Ed Thalmann, Ed Flynn, Drew Dutka, Ramsay Pearson, Lindsay Symon, David Dennison, Richard Moon, Maurice Cross, Ian Calder, Hans Ornahagen and Yehuda Melamed. The proceedings will be published by the Undersea and Hyperbaric Medicine Society.

The existing classification.

Before the Workshop the decompression illnesses were conventionally divided into CAGE and decompression sickness (DCS). DCS was further divided in types I (mild) and II (serious) in a system proposed over 30 years ago for caisson work.¹ The workshop participants agreed that although sudden loss of consciousness in a scuba diving candidate on surfacing in a swimming pool was almost certainly CAGE and that left knee pain in a saturation diver developing 6 hours after reaching the surface was similarly certain to be DCS, between these two extremes differentiation was often impossible.

Furthermore it was agreed that:

- a CAGE can present before reaching the surface;
- b almost all cases of cerebral DCS have symptoms within 20 minutes of surfacing;
- c many cases of cerebral DCS were likely to be due to arterialisation of venous bubbles and hence that DCS often initiated CAGE;
- d arterial emboli could either precipitate DCS or occur concurrently with DCS (the so-called type III DCS²);
- e most cases of CAGE did not have any evidence of lung damage;
- f in submarine escapees de-novo formation of bubbles in arteries could not be completely excluded; and,
- g while most cases of CAGE showed some spontaneous recovery many were static or progressive.

Many delegates reported that attempts at retrospective analysis of case histories had resulted in very low concordance between observers in the diagnosis of either

CAGE or DCS. Also, the recent decision of the United States Navy to treat CAGE initially at 2.8 bar breathing 100% oxygen meant that most of those attending had no incentive to make the distinction as treatment regimens were essentially common. This is particularly true given the move away from a significant head-down posture for CAGE sufferers.

Similarly, the type I and type II DCS classification was considered unsatisfactory because:

- a if left untreated at least 30% of type I DCS cases developed overt evidence of neurological involvement;
- b the frequency of long-term personality, psychological and neurological sequelae was almost as high in divers with a history of type I DCS as in those with type II DCS;
- c type II DCS could indicate anything between a diver with paraesthesia in the left finger and either an unconscious diver or a diver with intractable hypotension and shock;
- d in diving operations, technicians and divers (and most doctors) are unable to identify subtle neurological signs and hence make a diagnosis of type II DCS; and,
- e much of the pain in DCS was likely to be referred from the nervous system.

It was accepted unanimously then that the existing classification needed to be changed and that for the reasons given above neither an aetiological nor an organ-system classification was achievable. The latter would be made even more difficult by the multi-focal nature of the decompression illnesses.

The consensus then was that:

- a no attempt should be made to distinguish CAGE from DCS; and,
- b a clinical descriptive classification should be developed.

A clinical descriptive classification of the decompression disorders

The term decompression illness was proposed and accepted to include both the previous DCS and CAGE categories and to demonstrate that no distinction was being made.

It was agreed that this term should be prefaced firstly by an evolutionary term (static, resolving, relapsing, progressive etc) and secondly by an organ system term (these being the symptomatic organ systems). For example: a diver who collapsed on surfacing, was found to be unconscious and then recovered would have "resolving neurological decompression illness"; a diver who had increasing shoulder pain would have "progressive musculoskeletal decompression illness"; and, a diver with unchanging shortness of breath and paraplegia would have "static pul-

monary and neurological decompression illness". The last case illustrates that no attempt is made at putting symptoms into a hierarchy.

The overall classification of decompression disorders would then be:

a Barotrauma

- ENT
- Pulmonary (radiologically or clinically apparent pneumothorax, mediastinal and sub-cutaneous emphysema)
- Others

b Decompression illness

The lignocaine trial.

It now becomes possible to perform the planned CAGE-Lignocaine trial by identifying the applicable clinical syndromes (e.g. only progressive neurological decompression illness cases).

Other conclusions

In addition to agreeing to a trial of this new classification, the participants conceded the need for a common database for diving accidents. The composition of this database will form the basis of a subsequent workshop.

References

- 1 Golding F, Griffiths P, Hempleman HV, Paton WDM and Walder DN. Decompression sickness during construction of the Dartford Tunnel. *Brit J Indust Med* 1960; 17: 167-180
- 2 Neuman TJ and Bove AA. Severe refractory Type II decompression sickness resulting from combined no-decompression dives and barotrauma. In: Bove AA, Bachrach AJ and Greenbaum LJ Jr. (eds) *Underwater and Hyperbaric Physiology IX*. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1987, 985-991.

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