Letters to the Editor

Mannitol bronchial challenge testing and scuba diving

Dear Editor,

I was interested at your decision to publish Dr Anderson's letter about the mannitol bronchial provocation test.[1] I remain puzzled as to its relevance to scuba diving. As I have observed previously, bronchial smooth muscle evolved in order to contract and narrow the airways and can be made to do so in anyone if sufficient stimulus is applied. The level at which this bronchial responsiveness is labelled hyperresponsiveness and thus identified as a disease state seems to be arbitrary.

The mannitol test has been used along with eucapnic voluntary hyperventilation to try to document abnormalities in elite athletes who wish to use bronchodilators to improve their performance. This has been accepted by major sporting bodies to try to limit the very high use of bronchodilators by athletes. However, the problem is that mannitol and other tests of exercise-induced bronchospasm correlate very poorly with either reported symptoms or diminished performance in elite athletes such that some athletes with positive tests have no symptoms and others with symptoms and diminished performance have no bronchospasm on testing.[2] It is not clear, therefore, which group actually has a significant clinical problem.

So far as scuba diving goes, there is no evidence that either asthma or bronchospasm induced by testing with either pharmacological or non-pharmacological agents has any adverse outcomes in relation to barotrauma, decompression illness or mortality. Dr Anderson refers to individuals who are relieved at having an excuse to avoid scuba diving in the form of a positive bronchial provocation test.[3] I think that most doctors doing diving medicals would find this an unusual situation, and the vast majority of prospective divers who fail their medical are actually deeply disappointed. As the positive predictive value of bronchial provocation testing for adverse events in scuba diving must be so low as to approach zero, it would seem that introduction of a new test at this time is not sensible.

References

1 Anderson SD. Bronchial hyperresponsiveness, spirometry and diving. *Diving and Hyperbaric Medicine*. 2006; 36: 109-10.

2 Holtzer K,Douglass JA. Exercise induced bronchoconstriction in elite athletes: measuring the fall. *Thorax*. 2006; 61: 94-6.

3 Anderson SD,Wong R, Bennett M, Beckert L. Summary of knowledge and thinking about asthma and diving since 1993. *Diving and Hyperbaric Medicine*. 2006; 36: 12-22.

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Conflict of interest

I am currently an investigator in a multicentre trial looking at the use of mannitol manufactured by Pharmaxis in the treatment of bronchiectasis. I am not personally in receipt of any financial benefit from Pharmaxis in relation to this study nor have I any financial interest in Pharmaxis or other pharmaceutical companies.

Key words
Bronchial provocation testing, asthma, fitness to dive, letters (to the Editor)

Editor's note: This is the last submission that the journal will take on the specific issue of mannitol for bronchial provocation testing. However, letters regarding the SPUMS policy on asthma and diving, and related to Dr Walker's paper in this issue, are welcome.

Decompression sickness following breath-hold diving

Dear Editor,

Gemp and Blatteau point out in a recent case report that decompression sickness (DCS) is a possibility in breath-hold (BH) divers and advised that anyone who experiences unusual symptoms after BH diving should seek medical attention.[1] They describe a fit, young sailor in the French Navy who performed repetitive dives to 10–18 metres' sea water over 60–90 minutes, made 10–12 unassisted dives, each dive lasting 1–2 minutes with surface intervals of 5–6 minutes. Ascent times were 15–20 seconds.

The dive profiles should theoretically preclude such a person from developing DCS. However, due to forceful Valsalva manoeuvres, he suffered dizziness, visual disturbance, tightness in the chest with dyspnoea, flushed face and numbness of all limbs and the right side of the face. These symptoms appeared two hours after surfacing and lasted about one hour. He was discovered to have a patent foramen ovale (PFO) on subsequent investigation.