

Question 3. LTHE of diving as defined at the Consensus Conference in Norway 1993 are:

- A. Chronic dysbaric disorders.
- B. Sequelae of acute DCI.
- C. Have insidious onset.
- D. Not found in professional divers but typically in recreational deep divers.
- E. Include neurosensory impairment due to noise and welding gas exposures.

Question 4. With regard to DON lesions:

- A. Diagnostic investigations for possible DON after major bends DCI may consist of X-ray or MRI in the acute phase (as baseline only), and follow-up examinations not earlier than 6 months thereafter. Early MRI lesions may spontaneously disappear as they represent functional states while X-Ray lesions may appear much later.
- B. Will need early surgical intervention in order to avoid progression of the disease.
- C. X-ray changes represent zones of bone infarction by occlusion of small vessels. The theory says that these vessels are damaged directly or indirectly by microbubbles from inadequate decompression after hyperbaric exposure.
- D. Morphologically two types are distinguished: juxta articular and shaft lesions. Shaft lesions tend to collapse and produce pain symptoms, while the juxta articular ones usually remain asymptomatic.
- E. Divers with more than 20 h per week under pressure and going deeper than 30 msw should periodically be screened for DON. Traditionally, this means X-rays of the shoulders, hips and knees. A modern approach would be to perform MRI instead, as this avoids radiation exposure and is more sensitive.

Question 5. The medical examiner of divers should:

- A. Perform a SPECT-tomography when suspecting a DON. With this technique, DON can best be distinguished from similar lesions caused by other aetiologies.
- B. Propose a bone screening programme to the employer for divers being exposed to decompression stress known to be related to higher rates of DON.
- C. Declare a diver with a DON type B (shaft lesion) unfit for diving.
- D. Establish longitudinal neurological screening for those exposed to unusual decompression stress and environmental hazards (oil, gas, contaminants, dusts, etc).
- E. Search for alcoholism and hyperlipidaemia when faced with an X-ray suggesting a DON.

Letters to the Editor

Ultrasound under pressure

Dear Editor,

I enjoyed the review article “*Ultrasound in diving and hyperbaric medicine*”.¹ Gawthrope correctly asserts that ultrasound (US) is an excellent method to detect pneumothorax, pleural effusion, and adequacy of vascular filling, and that these skills are easily learnt. He also mentions more advanced uses of US in the hyperbaric chamber – intravascular bubble detection and cardiac function. The hyperbaric chamber is a potentially hostile environment in the sense that some of the usual clinical aids are often unavailable, and deterioration of the patient may produce a quandary as to whether the treatment session requires early cessation. I would like to mention other potential uses suitable for the hyperbaric environment.

US accurately confirms safe placement of endotracheal tubes – the tube can be visualized within the trachea and bilateral lung sliding shows both main bronchi are being ventilated.²⁻⁴

If there is clinical concern for raised intracranial pressure, US measurement of optic nerve sheath diameter may provide good guidance as to the need for urgent CT and/or neurosurgical intervention. A measurement greater than 5 mm is considered positive for intracranial hypertension.^{5,6}

US can be very useful to assist in obtaining both peripheral and central venous access in the unwell patient. If central venous access has been obtained and the patient deteriorates subsequently, US can be used to exclude pneumothorax, haemothorax and cardiac tamponade.

I believe US is an important tool for use both prior to hyperbaric treatment and also within the course of hyperbaric treatment for critically ill patients. Many machines that are relatively inexpensive, easy to use and robust are available, but not all may be suitable or safe in a hyperbaric environment, and so each model must be carefully assessed prior to use.

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Key words

Ultrasound, hyperbaric medicine, equipment, letters (to the Editor)

Control groups in hyperbaric trials

Dear Editor,

I read with interest Dr Bennett's excellent recent appraisal of the study by Londahl and colleagues.¹⁻⁴ However, there are some concerns with respect to the trial design that I would like to highlight. Londahl et al's study on the addition of hyperbaric oxygen to specialised wound care for chronic diabetic foot ulcers uses a questionable "sham" treatment method, which has been employed by the same research team previously.⁵ The paper by Londahl et al was also included in the recently updated Cochrane review of hyperbaric oxygen therapy for chronic wounds and appraised as having a low risk of bias, exclusively owing to the inclusion of a control group.⁶

What has not been commented on is whether their choice of control (sham) was appropriate. Londahl et al compared the effect of hyperbaric oxygen at 254 kPa in patients with diabetic foot ulcers with a sham group where patients breathed air at 254 kPa. In real terms, therefore, sham was equivalent to breathing 50% O₂ under normobaric conditions, which is not a true control. It could be argued that breathing 100% O₂ at normobaric pressure may have produced the same differences between the two groups. To better discern the effects of hyperbaric oxygen at 254 kPa a better control group would have been air at 1.0 ATA. Such an approach would confirm beyond doubt that the wound-healing effects are entirely attributable to hyperbaric oxygen.

There is also lack of discussion regarding the possible risk of decompression illness (DCI) in the control group since they are exposed to 90 mins of air at 254 kPa. This also raises ethical issues as the 'control' group is being exposed to a risk that the experimental group is not subject to. There were no reports of any adverse effects in the control arm, but the study only analysed 90 patients and the relative risk may be low, but still real. Conducting research in hyperbaric medicine is very difficult because of the problems of delivering sham

treatments and Londahl and colleagues have improved substantially on previous published studies. For instance, the study by Annane et al gave hypoxic gas mixtures under pressure to their control group to ensure they received the same oxygen dose equivalent to a patient breathing air at normobaric pressure.⁷ This was confirmed by blood gas analysis and the control group was therefore not only exposed to a potentially lethal gas mixture if pressurisation failed, but also the dual risks of arterial puncture and decompression sickness.

In order to undertake well-designed RCTs in hyperbaric medicine there has to be careful thought given to the appropriate control treatment group/sham, which should carry with it a negligible risk. Hyperbaric research needs to be promoted internationally and intervention trials should be designed with high methodological rigour. I disagree with Dr Bennett's assertion that this trial satisfied that principle.

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