A case is reported that provides further evidence of an old occupational hazard, dysbaric osteonecrosis, presenting in a new population (sports scuba divers) who also appear to be at risk. It highlights the need for an accurate diagnosis of diving related illness.

**CASE REPORT**

A previously fit and healthy 35 year old scuba diver presented to his local accident and emergency department. He had been diving the previous day and was now complaining of his legs “feeling woolly”, pins and needles in his toes, and poor hand-eye coordination the previous evening. Given the classic symptoms of decompression illness (DCI) and a recent history of diving, a provisional diagnosis of DCI was made. He was placed in 100% oxygen and transferred to our hospital’s recompression facility for further care.

On arrival, his symptoms were unchanged, but close questioning revealed that he had been complaining of pain and restricted movement in his left shoulder for several months. Detailed neurological examination was normal. Heel to toe walking and sharpened Romberg time were within normal limits. A diagnosis of acute DCI was made and a decision taken to treat with therapeutic recompression.

In view of the patient’s long standing shoulder complaints, a radiograph of the shoulder joint was taken before recompression treatment. The radiograph (fig 1) showed ABN as an advanced necrotic humeral head (A4b MRC classification). Treatment was started using the United States Navy recompression treatment table 6. The recompression treatment was uneventful, and the patient made a full recovery from acute DCI.

During follow up, the patient was offered a radiographic long bone survey, and magnetic resonance imaging (MRI) of any lesions found. Radiographs revealed subtle “snow capping” of the right humeral head with increased subchondral density typical of early ABN (fig 2). MRI detailed the extent of the damage underlying the bony changes seen on plain radiographs (figs 3 and 4).

**DISCUSSION**

Radiographic examination is not routine in assessing diving illness. However, when making a diagnosis one needs to exclude other causes, such as trauma, so as not to treat symptoms that do not origin from acute diving illness, thereby avoiding the expense and inconvenience of protracted recompression treatment. We identified evidence of ABN that accounted for the shoulder pain. The extent of the damage was striking in the light of the patient’s continued use of the limb, his occupation as a fireman, and pursuit of arduous physical sport.

The patient has no history or known medical causation of ABN other than hyperbaric exposure—for example, diabetes, steroid use, hyperlipidaemia, or trauma. He is a non-smoker, and consumes less than 15 units of alcohol a week. As an experienced diver, he was conducting sports dives to the limits of technology—that is, relatively deep dives using multiple gas mixtures rather than depth limiting air as the breathing medium. Although such deep diving practice is currently restricted to the more adventurous, advancements in technology and equipment are making such dives more commonplace.

ABN is a well documented hazard of pressurised tunnel work and to a lesser extent professional diving. It was first reported in 1911 by Bassoe. When associated with pressure
exposure, it is often referred to as “dysbaric osteonecrosis” (DON). Ohta and Matsunga and Wade et al reported an incidence of DON greater than 50% in discrete populations of professional shell fishermen in Japan and Hawaii who conduct deep scuba dives. When it affects the articular surfaces of the joints, it can lead to disablement of the affected limb. Its initial presentation can be painless necrosis of the joint detectable only by MRI, radiography, or similar diagnostic tool. It can occur in divers and tunnel workers who have not reported experiencing acute DCI. There are only three previous case reports in the world literature of DON in amateur scuba divers. In one of the reports, the diver had a history of insulin dependent diabetes and joint trauma, thus detracting from hyperbaric exposure as the sole causation. DON is believed to result from inadequate decompression. There is convincing evidence that pathologically DON is the result of nitrogen and lipid emboli initiating a coagulation cascade in both tissue and the microcirculation of bone. There are reports of other “diving” related causes, including theories about the compression/decompression cycles. There is, however, little direct supporting evidence. DON can occur months or years after exposure to increased atmospheric pressure. It appears that only one pressure exposure is needed to trigger the condition.

Wilmshurst reported that advancement of technology does not always advance the safety of sports divers. With increasingly modern equipment and changing life styles, amateur divers go deeper for longer, more often. Perhaps more worrying, amateur divers have a no (or low) risk perception of DON, a potentially disabling condition.

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