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exposure, it is often referred to as “dysbaric osteonecrosis” (DON). Ohta and Matsunga6 and Wade et al7 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.8–10 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.11 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.12 There are reports of other “diving” related causes, including theories about the compression/decompression cycles. There is, however, little direct supporting evidence.13 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.

Wilmshurst14 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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