Effects of Diving on the Brain (Part 1)

The question of whether diving can have deleterious, long-term health effects emerges from time to time but appears unanswered so far. Possible neurological complications from acute dive injuries are undisputed, but some studies show evidence of lesions in the central nervous system of divers with no history of decompression sickness (DCS). These subclinical lesions or “white spots” in the brain are detected with magnetic resonance imaging (MRI), a method very sensitive to changes in the brain. It is not clear whether they are more common in divers than in nondivers, nor is it certain that their presence has any importance.

In some studies, measurements of neurological function also indicated abnormal results in divers. These measurements included neuropsychological assessments such as memory and concentration tests, electroencephalograms (EEGs), which detect electrical activity in the brain, and single-photon emission computed tomography (SPECT) scans, which measure cerebral blood flow. In the Geneva “Memory Dive” study (Slosman DO et al., 2004), reduction in cerebral blood flow and neuropsychological performance was associated with a history of high dive frequency (more than 100 dives per year), dive depth (deeper than 40 m) and dive environment (cold water).

Establishing a causal relationship to diving and determining the pathological mechanisms of these brain lesions is difficult. Factors such as age, history of head injury, alcohol consumption, migraines, smoking, hypertension, high blood cholesterol, infection and presence of a patent foramen ovale (PFO) appear associated with these findings. Often, bubbles traveling through cardiac chambers and visualized using ultrasound do not cause any symptoms; these “silent bubbles” could cause the subclinical lesions.

A few studies have focused on the influence of a PFO, an opening between the right and left atria that can vary in size and is found in about 25 percent of the population. Bubbles formed as a result of decompression stress could theoretically travel from systemic circulation to the heart, cross from the right side to the left through the PFO and enter arterial circulation and, potentially, the brain. This mechanism mimics paradoxical embolism, in which a clot from a deep vein crosses through a PFO and ends up in the brain, causing a stroke. Although the presence of a PFO is considered a risk factor for brain lesions, so far there is no unequivocal evidence of a causal relationship between PFOs and silent injuries.

Additional evidence shows that breath-hold divers exhibit central nervous system effects as well. Acute strokelike injuries in breath-hold divers are well documented. A Swedish study showed that prolonged voluntary apnea can transiently increase levels of a brain-damage marker protein, even in the absence of symptoms of acute injury (Andersson JP et al., 2009). The researchers proposed that exposure to severe hypoxia could cause neurological damage over time. The risk of asymptomatic neurologic events and their possible long-term effect in divers remains unresolved. We ask the experts.

What, if any, evidence is there for brain lesions in divers without any history of DCS?

Richard Moon: Some studies, using MRI, observed a greater number of brain lesions in divers compared to nondivers. So far no relationship between the number of lesions and the number of dives has been established, which suggests that the lesions are not related to diving itself.

Günalp Uzun: Studies conducted in the last 20 years aimed at illuminating the presumed correlation between diving and brain lesions revealed conflicting results. Due to methodological differences among the studies, it is not possible to pool the data and reach a clear conclusion. Consistent with some earlier reports, we found a higher incidence of white-matter lesions in asymptomatic military divers compared to

nondiving controls (Erdem et al., 2009). A positive correlation, however, does not always imply causation. Most of these studies (including ours) did not establish any significant relationship between white-matter lesions and diving indices. Even if divers had increased numbers of white-matter lesions, their clinical relevance and association with neuropsychological symptoms has not yet been clearly defined.

Kay Tetzlaff: There is an abundance of studies that investigated MRI in a variety of diving cohorts, and many of these reported associations between parameters of diving exposure and presence of brain lesions on MRI. However, none could actually prove a causal relationship. A fundamental flaw in study design has been the possibility of a selection bias, in that the lesions in the selected divers could have been pre-existing. In fact, the studies could not disprove a hypothesis that the decision to start diving may be the first sign of brain damage. One way to reduce bias would be a longitudinal follow-up of a cohort of divers from the beginning of their diving career compared to a cohort of nondivers while controlling for confounding risk factors such as alcohol intake, smoking, hypertension and others. Such a study has yet to be reported.

What is the relationship between a PFO and brain lesions?

Moon: There is a weak relationship between the presence of a PFO and the presence of these lesions. But again, there is no evidence that these lesions indicate brain damage.

Uzun: So-called “silent gas bubbles,” which may be detected even after dives in shallow water, do not produce clinical symptoms and are generally filtered through the pulmonary vasculature. A PFO, an opening between the right and left atria, may serve as an entry point for silent gas bubbles into the arterial circulation. It is hypothesized that these bubbles can interrupt small vessels in the brain and cause white-matter brain lesions. Indeed, a number of studies demonstrated that divers with a PFO had an increased risk for white-matter lesions compared to divers without a PFO. There is no general recommendation that asymptomatic scuba divers should undergo examinations for PFO detection. However, a diver with a known PFO should use a conservative dive profile to reduce the risk of DCS.

Tetzlaff: A PFO increases the risk of decompression illness (DCI) and thereby may also enhance brain lesions on MRI. It has been estimated from a clinical study that divers with a PFO have a 4.5-fold increase in DCI events and twice the incidence of ischemic brain lesions compared to divers without a PFO (Schwerzmann M et al., 2001). However, it should be noted that diving even with a PFO is considered safe when dives are performed according to guidelines. Note that it is not the PFO that causes injury but the presence of gas bubbles during or after the dive. The bubble load can be minimized by avoiding risk factors such as deep dives, cold dives and decompression dives.

What are other possible mechanisms of formation of the brain lesions known as white spots?

Moon: They could be related to normal aging processes such as changes in blood vessels.

Uzun: White spots of the brain observed on MRI are actually common in elderly people and may be associated with head injuries, alcohol consumption, migraines, smoking, hypertension and/or high blood cholesterol. It is generally accepted that white-matter lesions represent parenchymal damage due to cerebrovascular disorders or cerebral ischemia.

Tetzlaff: White-matter hyperintensities are regarded as typical MRI expressions of cerebral small-vessel disease. Pathological correlates are varied with most pointing toward white-matter hyperintensities as a reflection of small-vessel ischemic burden. The predominant clinical associations are with stroke, cognitive impairment and dementia. The prevalence of white-matter hyperintensities increases with age.

The discussion continues in the second part of the article which will be published in the September issue.

Meet the Experts

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