Letters to the Editor

Inner-ear decompression sickness: 'hubble-bubble' without brain trouble?

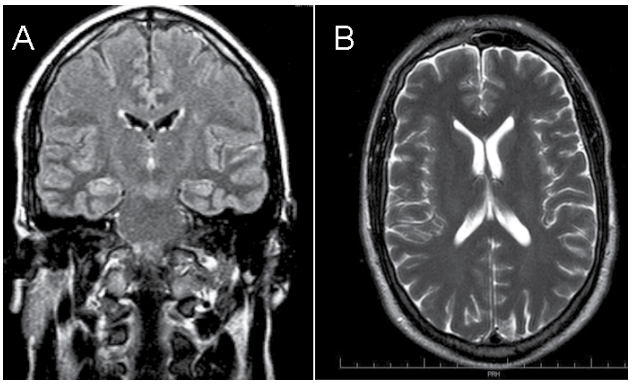
Inner-ear decompression sickness (DCS) is an incompletely understood and increasingly recognized condition in compressed-air divers. Previous reports show a high association of inner-ear DCS with persistent foramen ovale (PFO),[1,2] suggesting that a moderate-to-severe right-to-left shunt might represent a major predisposing factor, and more properly defining it as an event from arterial gas embolism (AGE). However, other conditions characterized by bubbles entering the arterial circulation, such as open-chamber cardiac surgery, do not produce inner-ear involvement, while sometimes damaging the brain extensively.[3] Moreover, in other sites, such as the spinal cord, the prevailing mechanism for DCS is not AGE, but more likely local bubble formation with subsequent compression of venules and capillaries. Thus, AGE might be, more properly, a predisposing condition, neither sufficient, nor possibly even strictly necessary for inner-ear DCS.[2]

A 'two-hit hypothesis' has been proposed, implying a locally selective vulnerability of the inner ear to AGE.[3] Modelled kinetics for gas removal are slower in the inner ear compared to the brain, leading to a supersaturated environment which allows bubbles to grow until they eventually obstruct the labyrinthine artery.[3] Since this artery is relatively small, there is a low probability for a bubble to enter it; this might explain the disproportion between the high prevalence of PFO in the general population (25–30%) and the very low incidence of inner-ear DCS in compressed-air diving (approximately 0.005%).[1,4]

Furthermore, given that the labyrinthine artery usually originates either from the anterior inferior cerebellar artery, or directly from the basilar artery, shunting bubbles will more frequently swarm through the entire brain.[3] In this case, however, the brain's much faster gas removal kinetics might allow for them to be reabsorbed without damaging brain tissue. In line with this scenario is the low probability (approx. 15%) of inner-ear DCS presenting with concomitant symptoms suggestive of brain involvement.[1] Interestingly, PFO is a putative risk factor not only for DCS but also for ischaemic stroke, and it has been hypothesized that a predominantly silent ischaemic cerebral burden might represent a meaningful surrogate of end-organ damage in divers with PFO, with implications for stroke or cognitive decline.[5,6]

Here we report the case of a 44-year-old diving instructor (> 350 dives) who suffered from inner-ear DCS about 10 min after a routine dive (5 min/40 metres' fresh water (mfw), ascent 7.5 mfw·min^{-1}, stop 10 min/5 mfw), resulting in severe left cochlear/vestibular impairment (complete deafness and marked vertigo, only the latter slowly receding after a few hours). The patient was not recompressed. A few months later, transcranial Doppler ultrasonography demonstrated a moderate-to-severe shunt (> 30 bubbles), presumably due to a PFO (he refused confirmatory echocardiography), while a brain MRI (1.5 T) was reported as negative for both recent and remote lacunar infarcts (Figure 1).

Figure 1
(A) Coronal FLAIR and (B) axial T2W brain MR images



We believe this may be evidence that inner-ear DCS could occur while the brain is completely spared, not only clinically, but also at neuroimaging. This would support either of two hypotheses: (a) that the brain is indeed relatively protected from arterial bubbles that preferentially harm the inner ear where, however, they only rarely infiltrate, or (b) that direct bubble formation within the inner ear cannot be completely discarded, and that the elevated PFO/inner-ear DCS association might be, in this latter case, merely circumstantial. We favour the hypothesis that inner-earDCS might be related to AGE in an anatomically vulnerable region. More precise data regarding the exact incidence of inner-ear involvement, isolating those subjects with moderate-to-severe shunt should be obtained before exploring the risk-to-benefit ratio given by transcatheter occlusion of a PFO for prevention of inner-ear DCS; odds that could end up to be sensibly different with respect to other types of DCS presentation.[7]

References

1 Klingmann C, Benton PJ, Ringleb PA, Knauth M. Embolic inner ear decompression illness: correlation with a right-to-left shunt. *Laryngoscope.* 2003;113:1356-61.

2 Gempp E, Louge P. Inner ear decompression sickness in scuba divers: a review of 115 cases. *Eur Arch Otorhinolaryngol.* 2013;270:1831-7.

3 Mitchell SJ, Doolette DJ. Selective vulnerability of the inner ear to decompression sickness in divers with right-to-left shunt: the role of tissue gas supersaturation. *J Appl Physiol.* 2009;106:298-301.

4 Klingmann C, Gonnermann A, Dreyhaupt J, Vent J, Praetorius M, Plinkert PK. Decompression illness reported in a survey of 429 recreational divers. *Aviat Space Environ Med.*

2008;79:123-8.
5 Ammirati E, Comi G, Camici PG. Patent foramen ovale closure and brain ischaemic lesions. *Heart.* 2013;99:1543.
6 Schwerzmann M, Seiler C, Lipp E, Guzman R, Lövblad KO, Kraus M, Kucher N. Relation between directly detected patent foramen ovale and ischemic brain lesions in sport divers. *Ann Intern Med.* 2001;134:21-4.
7 Moon RE, Bove AA. Transcatheter occlusion of patent foramen ovale: a prevention for decompression illness? *Undersea Hyperb Med.* 2004;31:271-4.

Acknowledgments

We thank Mirko Patassini and Emanuela Laura Susani, San Gerardo Hospital, Monza, and our patient for permission to publish his medical details and MRI.

Lucio Tremolizzo,[1] Massimo Malpieri,[2] Carlo Ferrarese,[1] Ildebrando Appollonio[1]

[1] Dept.artment of Neurology, San Gerardo Hospital and University of Milano-Bicocca, Italy
[2] Emergency Medical Service, Ventotene Island, Italy

E-mail: *<lucio.tremolizzo@unimib.it*

Key words
Inner ear; decompression illness; persistent foramen ovale; case reports; letter (to the Editor)