Reply: Diving-related disorders in breath-hold divers could be explained with the distal arterial bubble hypothesis

Thank you for the opportunity to respond to Dr Arieli. First of all, no clear mechanism of decompression sickness (DCS) in breath-hold (BH) diving has been confirmed or established, and we are all only guessing at various possibilities and hypotheses for the diving disorders.[1] Repetitive BH diving mainly causes stroke-like neurological events consistent with ischaemic brain lesions, and moreover the clinical characteristics of DCS in BH divers are different from those in scuba diving.

Dr. Arieli's hypothesis that cerebral DCS is caused by distal arterial bubbles developed from nanobubbles is interesting;[2] it seems to explain the formation of venous bubbles. However, we consider that gas micronuclei underlying decompression bubbles are circulating in all blood vessels, and that they may expand as a result of nitrogen transfer from the blood and surrounding tissues in repetitive BH dives. Microbubbles (less than 21 micron) formed from gas micronuclei can pass through capillaries of all tissues; they may grow mainly in venous capillaries. Hence, venous bubbles have been detected in some BH divers,[1,3] similar to the presentation in scuba diving.

We have presented some cases of stroke-like neurological disorders in Japanese Ama divers whose magnetic resonance imaging (MRI) of the brain showed signal changes in the 'external' watershed areas and the territories of the perforating arteries.[3] Watershed infarcts of the brain are grouped into two main categories based on their location in either internal or external regions.[4] The former are located at the junctions of the cortical arterial territories with deep perforating arteries, showing the rosary-like pattern in the centrum semiovale; they are mainly affected by hypoperfusion due to arterial stenosis or haemodynamic impairment. In contrast, the latter 'external' infarcts occur at the junctions of the distal fields of cortical arteries and are usually wedge-shaped, and their cause may be embolic rather than haemodynamic in nature.[4] Other ischaemic lesions involve the territories of the perforating arteries in the basal ganglia and brainstem.[3] While lacunar infarcts resulting from occlusion of a single perforating artery are considered to be due to atheromatous changes, one-third may include emboli from cardiac or carotid sources.[5] The MRI findings of Ama divers with stroke-like neurological disorders support the hypothesis that an embolic mechanism plays a crucial role in the pathogenesis of infarcts in the external watershed areas and the territories of perforating arteries; that is, the most plausible aetiology is occlusion of the cerebral arteries by emboli.[3]

Venous nitrogen bubbles formed following BH dives are trapped or retained in the pulmonary arteries. The 'trapped' bubbles are compressed during each dive and may therefore arterialise.[1] Circulating bubbles in arteries will increase in size during ascent; some of the bubbles flow into the arteries of the brain, especially at the junctions of the cortical arteries or in the perforating arteries. Moreover, even small bubbles may cause endothelial damage,[1] and may provoke thrombus formation and affect arterial occlusion of the brain. The bubble seeds are the first step in neurological DCS for BH divers, and this may be followed by expansion of bubbles is due to nitrogen influx from the end of occluded arteries and surrounding brain tissue. At present, the above hypothesis seems to best explain the mechanisms of neurological DCS in Ama divers.

In conclusion, based on MRI findings of the brain in Ama divers, repetitive BH dives cause stroke-like neurological disorders compatible with cerebral arterial embolism. We consider that nitrogen bubbles arterialised across the pulmonary circulation play a key role in brain damage in BH diving, although the mechanisms are not clear.

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Kiyotaka Kohshi

Division of Neurosurgery, Nishinihon Hospital, Kumamoto, Japan

Address for correspondence: *Dr Kiyotaka Kohshi, 3-20-1, Hattanda, Kumamoto-higashi, Kumamoto 861-8034, Japan*
kohshi33@gmail.com

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