Letters to the Editor

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

In the June 2021 issue of Diving and Hyperbaric Medicine,[1] Dr Kohshi and colleagues, expressed a degree of skepticism that distal arterial bubble formation[2,3] can be the main explanation for cerebral decompression sickness (DCS) in breath-hold divers. We have previously suggested that in the bifurcating tree of a distal artery, the vessel's wall becomes thinner and wall surface to volume ratio of the vessel increases; both of these circumstances enhance nitrogen diffusion from the surrounding tissue into the blood. A local reduction of blood flow, also leaves enough time to enhance nitrogen diffusion from tissue into the blood. If an active hydrophobic spot (AHS) is located in the distal artery, a nanobubble at the AHS could develop into a larger decompression bubble, blocking perfusion. In repeated breath-hold diving, a local bubble would remain almost stable because of a very small oxygen window in the arterial blood, and would continue to expand in further dives.

Kohshi and colleagues claimed: "*However, this hypothesis cannot explain why one or more large ischaemic lesions are not invariably accompanied by multiple small ones in the subcortical areas*".[1] There are two prerequisites for the expansion of distal arterial bubbles: enhanced diffusion of nitrogen with respect to the volumetric blood flow; and existence of an AHS. The distribution of AHS is highly variable between individual sheep, and within their blood vessels. I assume a similar variability exists in divers. Thus, if the diffusional conditions are appropriate for gas loading but there are no AHS, bubbles would not appear. If, however the diffusional conditions are appropriate for gas loading in a somewhat large artery, all arteries of the next generations having AHS would have decompression bubbles. The authors stress: "*The ischaemic lesions…were situated in the terminal zone…and border zone. They are so-called...low flow*".[1] Local low perfusion, which allow enough time for nitrogen loading, is exactly a major cause for distal arterial bubble formation.[2] The authors also conclude: "*The dramatic and rapid response suggests the presence of bubbles in the cerebral arteries. Nitrogen accumulation in fat tissues increases throughout repetitive breath-hold diving*".[1] These are exactly the appropriate parameters in distal arterial bubble formation. As is remarked in the review: "*Why lesions in breath-hold diving mainly involve the brain but not spinal cord is an unresolved question*".[1] I believe the distal arterial bubble is the answer.

Alessandro Marroni (personal communication) reported one case of clear clinical neurological symptoms in breath-hold diver without any detectable bubbles on echocardiography. A viewpoint on neurological decompression sickness in breath-hold diving claimed: "*Most studies reported no or rare circulating bubbles after repeated breath-hold dives. Consequently, other mechanisms should be considered*",[4] and other authors suggested: "*However, comparing the incidence of DCS in breath-hold diving with the low prevalence of venous gas emboli with scuba divers who often produce venous gas emboli in higher quantities with relatively low rate of injury, clearly the pathogenesis is more complex*".[5] These viewpoints and Marroni's observation contradict the preference expressed by Kohshi and colleagues for veno – arterial shunts as the main cause for breath-hold DCS: "*The prevalent theory of brain involvement is that arterialised venous gas bubbles passing through right to left shunts may be the plausible mechanism*".[1]

In summary, I am confident that the distal arterial bubble formation is the best explanation for the mechanism of cerebral DCS in breath-hold diving, rather than bubbles shunting from the venous to the arterial circulation and directed mainly to the brain.

References

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