Last Word on Viewpoint: Why predominantly neurological decompression sickness in breath-hold divers?

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To the Editor: We appreciate the insightful comments provided by all authors (see Ref. 3) who commented on our Viewpoint (4) on the hypothesis that the hypoxia of breath-hold diving may recruit pulmonary shunts and lead to the arterIALIZation of venous gas emboli (VGE). Because of the large number of comments we cannot respond to each author; however, we would like to address a couple of points that warrant further discussion:

Dr. Foster and colleagues (see Ref. 3) and Drs. Boussuges and Gavarry (see Ref. 3) point out that there are very few reports confirming the presence of VGE in breath-hold divers. We believe this is because of low awareness by researchers that breath-hold diving actually can be associated with VGE and probably also because of the lack of opportunity to measure VGE as there is a limited population engaged in extreme breath-hold diving. In fact, Dr. Balestra in his comment (see Ref. 3) is reporting on significant VGE detection in a subject after repeated shallow water breath-hold dives, and there are more reports on decompression stress as evidenced by bubble detection after breath-hold diving in the literature (5). Importantly, arterialized VGE in a diver may not necessarily cause symptoms (1).

Dr. Lemaître (see Ref. 3) and Dr. Muth (see Ref. 3) consider the possibility that nitrogen bubbles may form in arterial but not venous blood. We believe this to be an unlikely mechanism because of the fact that small bubbles would dissolve rapidly in the arterial system because of the effect of surface tension when remote from supersaturated tissue (4). However, arterialized VGE may reach a critical size to survive travel time to the brain and become hyperinflated when lodging in supersaturated tissue, i.e., the brain.

The hypothesis that a possible relation between intermittent hypoxia and cerebrovascular dysfunction, as observed in obstructive sleep apnea (OSA), is an interesting twist made by Dr. Foster and colleagues (see Ref. 3). However, sympathetic responses have been found to markedly differ between (healthy) breath-hold divers and patients with OSA, with central chemoreflex control of respiration and sympathetic activity being maintained in divers, whereas OSA is characterized by prolonged sympathetic overactivity and dysregulated cerebral circulation (2).

We agree with Dr. Madden and colleagues (see Ref. 3) and Dr. Fitz-Clarke (see Ref. 3) who indicate the role of exercise possibly augmenting nitrogen bubble formation outside the nervous system and being an alternative or supportive factor in the opening of IPAVA. Although this may apply to commercial repetitive shallow water breath-hold diving, exercise is less likely to play a role in assisted no-limits deep diving where spectacular cases of neurological insult have been reported even after single breath-hold dives.

In conclusion, we concur with the view of our colleagues that the pathophysiology of DCS-like phenomena in breath-hold divers may be complex, and more studies are needed to further elucidate the causes of neurological insult in extreme human breath-hold diving.

Disclosures

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Author Contributions

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References