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LETTER TO THE EDITOR

Distal arterial bubble: an alternative mechanism underlying vestibular decompression illness

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TO THE EDITOR: In their recent paper, Doolette and Mitchell (1) suggested that the extended lifetime of bubbles contributes to inner ear decompression illness (DCI). They attributed this to right-left shunt (PFO or IPAVA) of bubbles, which travel to the inner ear and expand while perfusion is low in the inner ear. They calculated bubble expansion by diffusion through a shell barrier surrounding the bubble for the assurance that the bubble survived the transit from heart/lungs to the inner ear. They also related the high incidence of excursions in saturation diving to the high pressure.

We suggested that in a "distal arterial bubble" formation, in the bifurcating arterial tree, the vessel's wall becomes thinner and wall surface to the volume of the vessel increases, both of these circumstances enhance inert gas diffusion from the surrounding tissue into the blood. A local reduction of blood flow, also leaves enough time to enhance inert gas diffusion from tissue into the blood. If an active hydrophobic spot (AHS) is located in the distal artery, the formed nanobubble at the AHS would develop into a decompression bubble. In repeated decompressions during a dive, a local bubble would remain almost stable because of a very small oxygen window in the arterial blood, and would continue to expand in following decompressions to a size that block the artery (2). We also suggested that vestibular DCI is related to distal arterial bubbles (3). Our suggested mechanism was supported by the occurrence of the following: very low vestibular perfusion; lower perfusion and more vestibular DCI in the right side as compared with the left side; occurrence of vestibular DCI in isobaric counter diffusion; and terminal arterioles that are longitudinal without collateral supply. In saturation dives, where excursions are common, the multiple decompressions should enhance the expansion of distal arterial bubble.

The increased vestibular DCI with right-left shunt is not proof of bubble transfer. Shunted venous blood with a high load of inert gas may also enhance inert gas transfer into a sedentary distal arterial bubble. Diffusional growth of a stationary bubble adhering to an AHS is more likely than a bubble that flowed in the blood stream. The association of high venous bubbles (VGE) and vestibular DCI is not proof of cause and effect. The distribution of bubblers/nonbubblers in divers and in blood vessels of sheep is similar (4). Bubbling in sheep blood vessels is related to the amount of dipalmitoylphosphatidylcholine (DPPC) in the AHS (5). Therefore, a diver with high AHS is prone to both high VGE and high distal arterial bubble formation.

It is not surprising that pain in joints and vestibular DCI are common in excursions in saturation dives. Both joints and vestibular organs are slow compartments, which become gas loaded in saturation dives. However, the mechanisms of DCI are different. Joint pain is most probably due to local expansion of nanobubbles at one of the four hydrophobic layers in the joint (6), and vestibular DCI was suggested to be related to distal arterial bubble formation.

For their calculations, the authors used a shell surrounding the bubble. This shell was introduced to match the discrepancy between fast diffusional growth of a bubble to the delayed DCI. In some reports, diffusion coefficient was markedly reduced to adjust the timing of bubble expansion with the delayed DCI. The presence of such a shell has never been experimentally established. We demonstrated in blood vessels of sheep that the initiation of AHS (the formation of the first optically observed bubble (diameter 0.1 mm) is the function that governs the appearance of bubbles. This is a slow process, peaking 45 min after decompression (4, 7). Expansion of a bubble from 0.1 mm to 1 mm obeys simple diffusion and lasts ~12 min. Therefore, I believe that the artificial induction of the shell is obsolete.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

R.A. conceived and designed research; performed experiments; drafted manuscript; edited and revised manuscript; approved final version of manuscript.

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