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Taravana, vestibular decompression illness, and autochthonous distal arterial bubbles

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ABSTRACT

Decompression bubbles can develop only from pre-existing gas micronuclei. These are the nanobubbles which appear on active hydrophobic spots (AHS) found on the luminal aspect of all blood vessels. Following decompression, with the propagation of blood along the arterial tree, diffusion parameters cause increased transfer of nitrogen from the tissue into the artery, and more so if perfusion is low. Taravana is a neurological form of decompression illness (DCI) prevalent in repeated breath-hold diving. A nanobubble on an AHS in a distal artery of the brain may receive an influx of nitrogen after each dive until it occludes the arterial blood flow. The vestibular organ has very low perfusion compared with the brain and the cochlea of the inner ear. We suggest that a nanobbubble on an AHS in the distal artery of the vestibular organ will receive a high influx of nitrogen from the surrounding tissue after decompression due to the low nitrogen clearance, thus expanding to cause vestibular DCI.

1. Introduction

1.1. Gas micronuclei – nucleation and stability

Decompression bubbles can expand and develop only from pre-existing gas micronuclei. The search for these gas micronuclei is almost a century old. It is known that nanobubbles form spontaneously when a smooth hydrophobic surface is submerged in water containing dissolved gas. We have shown that these nanobubbles are the gas micronuclei underlying decompression bubbles and decompression illness (DCI) (Arieli, 2017). Hills (1992) suggested that the hydrophobic multilayers of phospholipids he observed on the luminal aspect of blood vessels, which we have termed active hydrophobic spots (AHS), were derived from lung surfactant. The main component of lung surfactant, dipalmitoylphosphatidylcholine, was found in the plasma of sheep and at the AHS (Arieli et al., 2018). These AHS are to be found in almost all blood vessels (Arieli, 2017; Hills, 1992): in the arteries, veins, pulmonary and systemic circulation, and all the way along the arterial tree to the capillaries in the brain. Nanobubbles formed on the surface of these lamellar layers of phospholipids in divers will expand into bubbles on decompression (Arieli, 2017).

1.2. Evolution of bubbles in distal arteries

After decompression and the release of superfluous nitrogen from the venous blood into the lung, perfusion continues along the arterial tree. As blood advances toward the distal arteries the arterial wall becomes thinner, resulting in decreased resistance to the diffusion of nitrogen from the surrounding tissue into the blood. The ratio of the surface area of the vessel wall to its volume increases, further enhancing the diffusion of nitrogen. Finally, there will be an additional accumulation of nitrogen within the artery due to the reduction in blood flow. In this way, nitrogen from the surrounding tissue can cross over to the luminal side of the distal artery and increase its nitrogen tension. We calculated the increase in nitrogen tension for the distal arteries, demonstrating that the reduction in arterial blood flow will lead to increased nitrogen loading (Arieli and Marmur, 2017). An autochthonous nanobubble at an AHS in this distal artery may then develop into a bubble which will occlude perfusion. This proposed mechanism may provide an explanation for a number of hitherto enigmatic phenomena of DCI.

2. Taravana

Tuamoto divers, who engage in repeated breath-hold diving to collect pearls from the sea (40–60 dives a day), suffer from a form of

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DCI called taravana or madness in Polynesia (Cross, 1965). A nanobubble situated at an AHS in a distal artery within the brain may receive an influx of nitrogen in the course of the ascent and during the surface interval between dives. Because there is no "oxygen window" within the artery, this bubble will continue to adhere to the AHS during the next dive; as we showed (Arieli, 2017), bubble detachment occurs only after expansion to a diameter of about 1 mm. On a subsequent dive, because the brain and blood are fast tissues, continued nitrogen loading will lead to further growth of the bubble during the next ascent and surface interval. Repeated diving may cause the bubble to expand to the point where the artery is occluded. Nitrogen tension in the tissue was calculated to increase during repeated breath-hold diving (Paulev, 1967), and increased nitrogen tension was measured in Japanese Ama breath-hold divers following 3 h of diving (Radermacher et al., 1992). This elevation of nitrogen tension may result in enhanced transfer of nitrogen to the growing bubble in the distal artery.

Explanations available for the phenomenon of neurological DCI in breath-hold diving would appear to be rather flimsy and untenable. Schipke and Tetzlaff (2016) in the VIEWPOINT symposium (2016, J. Appl. Physiol. 120, 1474–1483) raised the questionable possibility of venous-arterial shunts, while their correspondents stressed that the mechanism of bubble formation within the arteries is unlikely. Loading of nitrogen in these two fast tissues (blood and brain), may indeed explain why there are mainly neurological disorders after repetitive breath-hold diving (Gempp and Blatteau, 2006; Tamaki et al., 2010b). None of the Japanese breath-hold divers who suffered acute stroke (neurological DCI) had a patent foramen ovale (Schipke and Tetzlaff, 2016). In compressed air diving, cerebral insults are less common than spinal cord disorders. Therefore, no support may be found for the notion that bubbles shunting from the venous side to the arterial circulation are the cause of neurological insults in breath-hold diving. Profuse venous bubbles would cause other symptoms of DCI (Tamaki et al., 2010a).

A search for bubbles in the right heart cavities following repeated breath-hold diving in one study of 15 subjects was without findings (Boussuges et al., 1997). However, bubbles were demonstrated in a study of a single spearfishing diver (Cialoni et al., 2016), and in other studies of breath-hold divers and the Ama of Japan (Lemaître et al., 2014; Spencer and Okino, 1972). The risk factor for taravana is the short surface interval between dives (Tamaki et al., 2010a), which would not affect shunting of bubbles from the right heart (the previously suggested mechanism), but would indeed have an effect on nitrogen unloading and autochthonous bubble growth.

Further support for autochthonous bubble formation in distal arteries may be gleaned from the study by Accurso et al. (2018) of a competitive spearfishing diver, who twice suffered taravana with evidence of a chronic brain blood flow deficit. As stated earlier, reduced perfusion increases nitrogen tension within the blood vessel (Arieli and Marmur, 2017), and is thus a risk factor for bubble formation in distal arteries. In summary, the risk of taravana can only be related to inert gas loading and unloading in the brain.

3. Decompression illness of the vestibular organ

There is a high incidence of DCI of the vestibular organ in the inner ear, resulting in dizziness, nausea, vomiting, vertigo and ataxia. About one quarter of recreational divers with neurological DCI have vestibular-cochlear symptoms (Nachum et al., 2001). Of 70 recorded cases of DCI in sport divers (Dick and Massey, 1985) 25 had vestibular involvement, 15 of these also having cerebral air embolism. In a cohort of 206 patients with inner ear DCI (Mitchell and Doolette, 2015), 136 of the 195 patients with vestibular involvement had vestibular DCI alone. Vestibular DCI may occur in the water very early on in the ascent, placing the diver at serious risk (Ignatescu et al., 2012). In most cases, symptoms appeared within 30 min of decompression (Mitchell and Doolette, 2015).

Right-to-left shunts were reported in 25% of divers who had never had DCI, and in 81% of divers who had suffered inner ear DCI. This led some authors (Mitchell and Doolette, 2015; Verrecchia et al., 2012) to attribute the vulnerability of the vestibular organ to bubbles reaching the arteries of the inner ear as the result of a venous-arterial shunt. Due to the low perfusion of the vestibular organ, nitrogen washout is decelerated, with subsequent expansion of the bubbles in the vestibular arteries.

A clear indication of the low blood flow in the vestibular organ may be obtained by calculating the half-time for nitrogen washout, which in the brain is 1.2 min and in the inner ear 8.8 min. The cochlea receives four times the blood flow of the vestibular organ. The terminal arterioles serving the vestibular subunits are longitudinal without collateral supply, and very few arteriovenous shunts that could bypass the capillary net. The perfusion of the vestibular organ is thus especially low and vulnerable. Low perfusion may be the mechanism underlying the expansion of an autochthonous bubble in a distal artery. Low perfusion allows enough time for a large amount of nitrogen to diffuse from the tissue into the artery and promote the growth of a local bubble. Inner ear DCI is more common on the right side (Gempp and Louge, 2013; Nachum et al., 2001), which the authors related to the narrower diameter of the right vertebral artery. However, considerations of arterial bubble load should place the left inner ear at greater risk (Solano-Altamirano and Goldman, 2014). As suggested here, the low perfusion on the right side should have an effect on the growth of a local bubble adhering to an AHS in the distal artery, and is therefore in agreement with the findings.

The appearance of vestibular DCI specifically in isobaric counterdiffusion (Lambertsen and Idicula, 1975) also supports the notion of local bubble expansion in a distal artery, rather than right-to-left shunting of bubbles or isobaric counterdiffusion alone within the inner ear. In the dry hyperbaric chamber, vestibular DCI appeared on breathing mixtures with neon or nitrogen as the inert gas in ambient helium at 37.4 ATA. Helium diffused through the round window from the middle ear, and was able to diffuse faster into the local nanobubble in the distal artery compared with the exit rate of the resident (inspired) gas. The high solubility of the resident gas in tissue and the low perfusion reduced the tension gradient between nanobubble, tissue and blood, inhibiting the clearance of the resident gas and causing bubble expansion in the distal artery. A switch during decompression from helium to air breathing at a depth greater than 33 m resulted in vestibular DCI (Hamilton, 1976). The greater solubility of nitrogen compared with helium resulted in a high influx of nitrogen via the blood and low outflux of helium, which caused the bubble in the distal artery to expand.

Our proposed mechanism of distal arterial bubble expansion cannot exclude the possibility of bubbles shunted from the venous circulation, because there is nevertheless a high incidence of patent foramen ovale in divers suffering from vestibular DCI. However, a right-to-left shunt is not proof that bubbles were shunted from the venous side to the arterial circulation. It may well be that there is shunting of venous blood rich in nitrogen, which will also promote bubble expansion in a distal artery. The proposed mechanism may explain the prevalence of vestibular DCI overall, its occurrence in the absence of venous-arterial shunts, and its predominance in isobaric counterdiffusion. When there is a right-to-left shunt, both mechanisms may be brought into effect, bringing about an increase in vestibular symptoms with respect to other forms of DCI.

4. Other forms of DCI

Reduced perfusion and the involvement of bubbles in distal arteries may be related to additional manifestations of DCI. A diver who dove to 20 m for 30 min suffered total loss of vision in the left eye, and was found to have left anterior cerebral artery hypoplasia (Omar et al., 2018). Thus reduced local perfusion could be the cause of local bubble expansion. Microvascular insult and the focal, highly localised punctate

lesions seen in the white matter of experienced divers, as well as other cerebral deficits such as deterioration of working memory and visual spatial performance in recreational divers unrelated to a PFO, and spinal cord DCI, have been discussed previously as a possible outcome of reduced perfusion (Arieli, 2017).

5. Conclusion

1 Nanobubbles on the surface of active hydrophobic spots are to be found in distal arteries of the brain, the vestibular organ and other tissues.
2 On decompression, diffusion parameters enable the transfer of nitrogen from the tissue to the blood within a distal artery especially when perfusion is low, resulting in growth of the nanobubble.
3 Each ascent and surface interval in repetitive breath-hold diving may add nitrogen to the growing bubble in a distal artery. This may lead to taravana.
4 Low clearance of nitrogen in the vestibular organ may enable a nanobubble in a distal artery to gain nitrogen during and after decompression, causing vestibular DCI.

Conflict of interest

The author declares that he has no conflict of interest.

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