Comentarios sobre la visión: ¿Por qué predominantemente neurología DCS en divers de apnea?

IPAVA RECRUTEMENT CAN’T BE RESPONSIBLE FOR DECOMPRESSION SICKNESS WITHOUT EVIDENCE OF ARTERIALIZED MICROBUBBLES!

TO THE EDITOR: Schipke and Tetzlaff (5) suggest breath-hold diving may recruit intrapulmonary arteriovenous anastomoses (IPAVA), providing a pathway for venous gas emboli to become arterialized leading to transient neurological injury consistent with transient ischemic attacks. To be a valid hypothesis there must be evidence of microbubbles in the right ventricle, left ventricle, carotid, or cerebral arteries of breath-hold divers after a typical dive profile with common dive times and surface intervals. Only one study could find evidence of microbubbles in the pulmonary infundibulum in a single subject (2) who had never even had symptoms of decompression sickness! With no evidence of arterialized microbubbles, the suggestion of IPAVA recruitment is premature. The recruitment of IPAVA by hypoxia is equally controversial, owing in part to an invalidated measurement technique that lacks a quantifiable assessment of IPAVA blood flow and involves a contrast agent that is susceptible to changes in transit time, blood gases, barometric pressure, and blood viscosity (1). Alternatively, we wondered if the dive profile of commercial breath-hold divers could be a sufficient intermittent hypoxic stimulus predisposing divers to the same cerebrovascular dysfunction observed in obstructive sleep apnea patients with similar presentations on neuroimaging studies (4). Progressive cerebrovascular dysfunction may underlie acute neurological injury, particularly if breath-hold diving acutely increases cerebrovascular transmural wall stress (through increased cerebral blood volume). The presence of vasogenic edema in breath-hold divers with neurological injury (3) supports a role for the breakdown of the blood-brain barrier, rather than gas emboli from IPAVA recruitment.

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COMENT ON VIEWPOINT: WHY PREDOMINANTLY NEUROLOGICAL DCS IN BREATH-HOLD DIVERS?

TO THE EDITOR: Hypoxia-induced pulmonary shunting was recently proposed to explain the predominantly neurological decompression sickness observed in breath-hold diving (4). This mechanism was hypothesized for both repetitive breath-hold dives with short surface intervals and long breath-holding sessions (1). However, despite the high exposure of these divers, few circulating intravascular bubbles have been observed (3). We therefore suggested that the brain lesions in breath-hold divers are not caused by disturbed venous circulation and are not characterized by increased S100B but instead may have several other possible causes (2). For example, arterial blood gas tensions very quickly reflect inspired partial pressures, whereas other tissues equilibrate more slowly; therefore, bubbles may form in arterial but not venous blood, which would have lower PAO2. The effects of depth and the induced hemodynamic changes (blood shift) are also possible. During a rapid ascent, the blood that has shifted into the thoracic cavity can reverse. However, this reversal is probably not as rapid as the pulmonary expansion, resulting in the entrapment of bubbles and their passage into the arterial circulation. Last, distinguishing between concussed breath-hold divers and healthy subjects based only on the current S100B cutoff value of 0.1 μg/l is not precise. Values below 0.1 μg/l are associated with a low risk of obvious neuroradiologic changes, and S100B should therefore not be used as the only diagnostic tool for concussion management (5). To conclude, the pathogenesis of predominantly neurological decompression sickness in breath-hold divers is probably multifactorial, indicating the need for broad safety procedures.

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NEUROLOGICAL DCS IN BREATH-HOLD DIVERS IS NOT JUST VGE; POSSIBLE MECHANISMS AND IMPLICATIONS!

TO THE EDITOR: Schipke and Tetzlaff (4) describe potential mechanisms of decompression sickness (DCS) in breath-hold divers (BHD) related to the arterIALIZATION of emboli citing magnetic resonance imaging and case reports that are of interest. In SCUBA diving, other areas have been investigated with links to DCS including microparticles, impairments of endothelial function, and platelet activation, suggesting complex mechanisms (5). If the explanation was as simple as the arterIALIZATION of venous gas emboli (VGE), the cases of DCS would be quite alarming as we regularly see frequent arterial-
izations in asymptomatic SCUBA divers with a grade of 4a or higher on the Spencer scale (2).

AGE cannot be ignored, and intrapulmonary arteriovenous anastomoses (IPAVA) remain a potential pathway for their introduction. Related to mechanisms, VGE are often found after surfacing where hypoxia in BHD is normalized within seconds. Additionally, exercise should not be discounted because light activity postdiving, such as treading water, could be enough to open IPAVAs when VGE are present (3). We concluded that this postdive period is as critical as the dive itself and have observed emboli in the cerebral circulation (1).

We agree that one critically placed bubble could cause severe injury. However, comparing the incidence of DCS in BHD with the low prevalence of VGE with SCUBA divers who often produce VGE in higher quantities with a relatively low rate of injury, clearly the pathogenesis is more complex. Still, BHD experience symptoms of DCS and it remains a risky sport; further studies with these subjects are needed to examine additional mechanisms.

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BRAIN DAMAGE INDUCED BY REPEATED BREATH-HOLD DIVING: WHAT PATHOGENESIS?

TO THE EDITOR: In their Viewpoint, Schipke and Tetzlaff (5) discussed the pathogenesis of the neurological injury observed in breath-hold divers (BHD). They suggested that paradoxical air embolism, through intrapulmonary shunt, played a key role in brain damage. The contribution of intravascular circulating bubbles is a factor commonly cited in the literature to explain the neurological troubles observed in BHD. However, this hypothesis presents some weaknesses. Indeed, although the creation of circulating bubbles secondary to nitrogen supersaturation is a well-known phenomenon after SCUBA diving, T, most studies reported no or rare circulating bubbles after repeated breath-hold dives. Consequently, other mechanisms should be considered.

During abrupt changes in blood pressure, cerebral blood flow (CBF) is maintained constant thanks to dynamic cerebral autoregulation (dCA). In real diving conditions, several stressors can alter cardiovascular function. First, apnea induces changes in heart rhythm such as bradycardia or arrhythmia (3). Hemodynamic changes are also induced by swimming and by alterations in thoracic pressure secondary to the Valsalva maneuver, to changes in lung gas volume at depth, or to glossoharyngeal insufflation maneuvers (1).

Breath-hold diving induces acute changes in arterial blood-gas tensions, including hypoxia and hypercapnia. It has been demonstrated that dCA was impaired by exposure to hypoxia (4) and by hypercapnia during maximal apnea (2). Consequently, the regulation of CBF might be disturbed during breath-hold diving.

Although the improvement in performance and the use of underwater scooters support the contribution of decompression sickness in the brain lesions observed in BHD, other potential mechanisms should not be forgotten.

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NEUROLOGICAL DECOMPRESSION SICKNESS IN BREATH-HOLD DIVING: A CASE OF HYPOXEMIA-INDUCED FLOATING BUBBLES?

TO THE EDITOR: Schipke and Tetzlaff (3) suggest several explanations for why breath-hold diving (BHD) causes neurological decompression sickness (DCS), such as hypoxemia, body position, and increased cardiac output. Increasing cardiac output while resting supine and during upright exercise is known to increase blood flow through intrapulmonary arteriovenous anastomoses (IPAVA), which would allow for bubble arterIALIZATION (2). Blood flow through IPAVA is significantly increased when breathing hypoxic gas at rest while supine, but is minimal or undetectable at the same FIO2 while resting upright demonstrating a body positioning effect on hypoxemia-induced bubble arterIALIZATION (2). Additionally, upright exercise breathing hypoxic gas increases bubble arterIALIZATION beyond that observed during exercise breathing air (2). What remains unknown is whether arterialized bubbles flow to the cerebral vasculature to a greater extent after BHD compared with SCUBA diving and therefore cause a greater incidence of cerebral bubble arterIALIZATION and neurological DCS. Both SCUBA diving and BHD would increase cardiac output and have similar body positioning during ascent, but only BHD...
results in hypoxemia (5). Thus, nitrogen bubble arterIALIZED from hypoxemia-induced blood flow through IPAVA is likely greater in BHD compared with SCUBA diving (1). Nitrogen bubble emboli in dogs distribute according to their size/buoyancy, preferentially floating upward at vertical bifurcations (4). Although every bubble does not likely float upward, the hypoxemia-induced increase in the number of bubbles, combined with hypercapnia- and hypoxia-induced increases in cerebral blood flow in BHD, increases the likelihood that bubbles may reach the cerebral vasculature.

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COMMENT ON VIEWPOINT: WHY PREDOMINANTLY NEUROLOGICAL DCS IN BREATH-HOLD DIVERS?

TO THE EDITOR: Decompression sickness (DCS) is a regular occurrence among SCUBA divers, whereas confirmed reports in apeine divers are still rare. Schipke and Tetzlaff (4) discuss pulmonary shunts as a reason for venous gas emboli (VGE) in the case of stroke-like phenomena similar to gas embolism in scuba divers. Even short hyperbaric episodes lead to saturation of nitrogen and could favor a cumulative bubble formation in the venous part of the body. The last 10 years have seen an increase in the number of famous apeine divers with stroke-like symptoms, even in absence of official scientific data. Their hypothesis that VGE-like symptoms could be caused by hypoxic recruitment of intrapulmonary arteriovenous (IPAV) anastomoses seems well considered; the line of argument convincing.

Nevertheless, it should be mentioned that hemoptysis after breath-hold diving (1, 3) may indicate pulmonary barotrauma. Involuntary breathing movement in the end of apeine (2), u-turns after reaching the maximal depth, and upper limb pull-ups in the discipline “free immersion” when the lung is already maximally squeezed could also foster the emergence of VGEs. Therefore, barotrauma should be investigated as a potential trigger of gas embolism in apeine deep divers.

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TO THE EDITOR: Decompression sickness (DCS) is of significant concern for breath-hold divers, and the chances of suffering from it increase with increase in depth of diving, bottom time, rate of ascent, and duration of surface interval (4). In breath-hold divers who experienced DCS the average diving pattern was characterized by dives to 31 m for 2 min, separated by 2.5 min surface intervals over 5 h with 16 dives/h (3). DCS symptoms ranged from minor joint pain, skin rash/itching to more serious respiratory, circulatory, and neurological complications (1). Of all the symptoms, the most prevalent (56%) is neurological in breath-hold divers (2). Authors of the Viewpoint (5) suggest hypoxia induced pulmonary shunts and breathing of pulmonary filters by venous gas emboli during decompression and subsequent arterIALIZATION of gas emboli to result in cerebral DCS in breath-hold diving. These bubbles are reported to cause vascular endothelium damage, blood-brain-barrier disruption, complement cascade initiation, elicitation of inflammatory cascade (1, 2), and activation of platelets. They could lead to focal ischemia in neurological tissue. The TREK-1 potassium channels are reported to mediate this effect in a neuroprotective manner. Resolution of most of the DCS symptoms with hyperbaric oxygen treatment proves the role of hypoxia in neurological DCS. Before treating DCS it is vital to differentiate the signs and symptoms caused by extreme changes in blood pressure, middle/inner ear barotrauma, mask barotrauma (in scuba diving), microvascular disease, hypercapnia, increased release of erythrocytes from spleen leading to symptoms having semblance to neurological DCS.

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COMMENT ON VIEWPOINT: WHY PREDOMINANTLY NEUROLOGICAL DCS IN BREATH-HOLD DIVERS?

TO THE EDITOR: The possibility of decompression sickness (DCS) in apnea divers after repeated dives is well documented. It has been shown that with repetitive dives, especially with short surface intervals and diving depths beyond 15 to 20 msw (50–66 ft), nitrogen accumulates progressively in the tissues. The level of nitrogen tissue loading not only depends on the diving depth but also is determined by the ratio of dive duration to recovery period at the surface (2). However, a few cases of DCS-like syndromes have also been reported after single apnea dives, and their complete response to recompression treatment was considered indirect evidence that bubble formation was the cause of this incident (1).

In contrast to the repetitive diving where nitrogen loading can be calculated and correlated to symptoms, the mechanism for DCS after a single dive is not clear. Schipke and Tetzlaff (3) present an interesting hypothesis that DCS after single apnea dives may be possible through shunting of venous
nitrogen bubbles to the arterial system. An alternative explanation would be that supersaturation and bubble formation in arterial blood may be possible due to very rapid ascents from deep breath-hold dives.

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