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New Directions in Decompression Sickness Prevention – A Narrative Review

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Abstract

Background: Decompression sickness (DCS) is a serious medical condition that can develop in divers during ascent. It is associated with the formation of inert gas bubbles in blood vessels and tissues. Prevention is executed by performance of decompression stops to release nitrogen in a controlled manner, but as researchers highlight the role of complex inflammatory mechanisms and endothelial dysfunction, novel directions of prophylaxis in DCS are being explored.

Objective: This narrative review synthesizes evidence on potential supplementary measures in DCS prevention, evaluating physical activity, biochemical and pharmacological interventions, as well as the impact of breathing mixtures usage.

Methods: Evidence from clinical trials and animal studies was analyzed. Outcomes included biomarkers and parameters linked with DCS development, such as bubble grades, platelet depletion, microparticle (MP) accumulation and reduction of endothelial flow-mediated dilation (FMD).

Results: Pre-dive aerobic exercise and usage of oxygen-enriched breathing mixtures consistently decrease venous gas emboli (VGE) formation. Ascorbic acid supplementation attenuates post-dive microparticle accumulation and mitigates reductions in FMD. In animal models, anti-thrombotic pretreatment with clopidogrel significantly decreases DCS mortality and platelet consumption, whereas aspirin and heparin show no protective effects.

Conclusion: Pre-dive physical conditioning, specific antioxidant and anti-thrombotic pharmacological interventions, alongside enriched-oxygen breathing mixtures offer promising, complementary prophylactic measures to standard decompression protocols. Given the varying

individual susceptibility to DCS, these alternative interventions provide a foundation for personalized diver safety protocols. Further large-scale clinical studies are necessary to establish optimal scopes of interventions and concrete guidelines for their implementation.

Keywords: decompression sickness**;** scuba diving; prevention; exercise; premedication; anticoagulants; nitrox; ascorbic acid

Introduction

Decompression sickness (DCS) is a serious health concern for a diver. During ascent, inert gases absorbed under high ambient pressure are released into the blood as bubbles. The venous gas emboli (VGE) form in or travel to all parts of the body, causing obstruction of blood vessels, inflammation, and tissue damage (Cooper JS, Hanson KC, 2023). Milder symptoms include musculoskeletal pain and skin rash, but it may also lead to serious pulmonological and neurological complications and can be fatal (Mitchel SJ, 2024; Cooper JS, Hanson KC, 2023).

VGE traditionally serve as an indicator of decompression stress, but are not the only factor in the pathophysiology of decompression sickness (Mitchel SJ, 2024). It has been proven that gas bubbles damage the vascular wall as well as directly cause platelet activation and inflammation, further compounding the effects of mechanical obstruction (Pontier et al. 2011; Pontier et al., 2012). Researchers are investigating blood platelet counts, platelet aggregation, cell-derived microparticles (MPs), and endothelial flow-mediated dilatation (FMD) as critical markers and mediators of DCS.

Adherence to decompression protocols, which are a series of stops during surfacing designed to manage the release of inert gas in controlled manner, remains the main method of prevention (Mitchel SJ, 2024). Interventions targeting bubble formation, inflammation, and platelet aggregation are explored as alternative approaches.

Objective of the article

The primary aim of this review is to synthesize the available evidence on alternative DCS prevention methods. Our objective was to evaluate the pathophysiological mechanisms of DCS in order to identify available points of intervention, and examine the efficacy of pre-dive exercise, biochemical interventions and the usage of enriched gas mixtures usage as potential measures supplementary to standard decompression protocols.

Methods

The present study was conducted as a narrative review of available studies regarding alternative prevention methods for decompression sickness. The purpose of the review was to provide a broad summary of existing scientific data on the topic rather than quantitative synthesis or statistical meta-analysis.

The literature search was performed using the PubMed biomedical database. Various combinations of search phrases were used, including the terms: Decompression Sickness, DCS, caisson disease, prevention, pharmacology, prophylaxis, drug, premedication, scuba, diver, hyperbaric. Only articles published in English were considered.

First, the abstracts of studies were reviewed to identify potentially relevant publications. Articles were excluded if they were incomplete, not focused on preventive measures or unrelated to decompression sickness in divers. Subsequently, full publications were reviewed to assess their suitability for inclusion. Types of publications included original research articles, clinical trials, case reports and pre-clinical studies.

Considering the experimental nature of some of the interventions, both animal and human studies were reviewed to provide a suitable scope of research. However, clinical studies were given priority. Articles were selected for final inclusion based on their relevance to the topic of DCS prevention, clarity of methodological reporting and contribution to improvement of mechanisms underlying DCS and possible improvement of safety in scuba diving.

Results

1. Pathophysiology of DCS

The pathogenesis of Decompression Sickness (DCS) is a complex cascade of mechanical, ischemic, and inflammatory events (Cooper JS, Hanson KC, 2023). The foundational

trigger for these events is the formation and distribution of gas bubbles within the body's tissues and vasculature (Mitchel SJ, 2024).

1.1. The Mechanisms of Bubble Formation

During a compressed gas dive, pressure of inspired air is in direct proportion to the ambient underwater pressure (Mitchel SJ, 2024). This in turn leads to an increase in partial pressure of gas dissolved in tissue (Vann et al, 2011). When surfacing, if total gas partial pressure exceeds the ambient pressure (a condition referred to as ‘supersaturation’), bubbles will form in tissues or the blood passing through them (Cooper JS, Hanson KC, 2023; Vann et al, 2011). Because the de novo formation of a bubble in a pure fluid requires immense pressure differentials not seen in recreational diving, current physiological models dictate that bubbles must grow from pre-existing "gas micronuclei" - microscopic gas pockets trapped in hydrophobic crevices on the endothelial walls of blood vessels, or circulating within the blood itself (Mitchel SJ, 2024).

Capillaries and venules draining supersaturated tissues are the primary sites for intravascular bubble growth (Mitchel SJ, 2024). These venous gas emboli (VGE) are readily detectable using modern Doppler ultrasound and echocardiography, allowing researchers to track bubble grades as a surrogate for decompression stress (Eftedal et al., 2007). Bubbles also form directly within the extravascular spaces of supersaturated tissues.

Most VGE are effectively filtered by the pulmonary capillary bed and exhaled harmlessly (Vann et al, 2011, Mitchel SJ, 2024). However, if the bubble load overwhelms the lungs, or if the diver possesses a right-to-left shunt like a patent foramen ovale (PFO), venous bubbles can bypass the lungs and enter the arterial circulation (Vann et al, 2011). Arterialized bubbles lodging in tissues can lead to localized ischemia, secondary inflammatory swelling, and mechanical distortion of nerve pathways, presenting clinically as headaches, motor weakness, paraplegia, loss of proprioception, or severe vertigo (Mitchel SJ, 2024; Cooper JS, Hanson KC, 2023). More benign manifestations of DCS include deep, boring musculoskeletal joint pain, profound fatigue, and cutaneous rashes (Cooper JS, Hanson KC, 2023).

1.2. Microparticles and the Inflammatory Cascade

DCS is not merely a mechanical obstruction problem. The presence of VGE in the bloodstream acts as a foreign surface, serving as a catalyst for a systemic inflammatory response (Mitchel SJ, 2024; Pontier et al., 2011).

Decompression stress triggers the shedding of microparticles (MPs)—sub-micron cellular fragments carrying pro-inflammatory cytokines—from the membranes of erythrocytes, leukocytes, platelets, and endothelial cells (Cialoni et. al 2022; Pontier et al. 2012). Notably, MP generation can be stimulated by elevated partial pressures of inert gases and hyperoxia-induced oxidative stress even before bubbles fully form (Mitchel SJ, 2024). Some researchers theorize that gas-containing MPs may actually serve as the micronuclei that seed bubble growth (Mitchel SJ, 2024).

Bubbles directly damage the vascular endothelium and strip its protective surfactant layer (Ribičić et al., 2019; Mitchel SJ, 2024). This mechanical damage, combined with circulating MPs, activates leukocytes and triggers the coagulation cascade - blood platelets recognize bubbles as foreign bodies, leading to adhesion and bubble-induced platelet aggregation (Mitchel SJ, 2024; Pontier et al., 2011).

2. Types of preventive interventions

2.1. Pre-dive Exercise

Pre-dive aerobic exercise generally demonstrates a protective effect against decompression sickness (DCS) by modulating bubble formation and systemic inflammatory responses, although the specific mechanisms involved are not yet clear and remain to be evaluated. Exercise may alter the population of gaseous nuclei from which bubbles form and improve endothelial resistance to damage caused by VGE (Blatteau et al., 2005).

Dujic et al., (2004) conducted a randomized crossover trial in healthy male divers examining the effects of a single bout of strenuous interval exercise performed 24 hours before a simulated hyperbaric chamber dive to 280 kPa. Participants performed 8 intervals of exercise for a total of 40 minutes, each consisting of treadmill running for 3 minutes at a high intensity (90% of their predetermined maximum heart rate), immediately followed by a 2-minute active recovery period at a lower intensity. Following the intervention, participants demonstrated a significant reduction in both the maximum bubble grade and the average number of venous gas bubbles in the pulmonary artery compared to dives

without preceding exercise. Although the sample size was small, the results suggest that strenuous aerobic exercise 24 hours before a dive can effectively reduce bubble formation, potentially by reducing the number of pre-existing gas micronuclei from which bubbles grow.

A study in trained military divers by Blatteau et al., (2005) examined the effects of a 45-minute submaximal running session performed 2 hours before a simulated dry chamber dive to 30 msw (meters of sea water, approximately 294.2 kPa). Following the intervention, participants demonstrated significantly lower mean VGE grades 60 minutes after surfacing, and no diver showed an increase in bubble grade after the exercise. Although the exact threshold of necessary exercise intensity remains unknown, the results suggest that aerobic exercise 2 hours before a dive decreases bubble formation, likely through biochemical protections such as nitric oxide pathways or heat shock protein responses.

Madden et al., (2014) examined the effects of 60 minutes of high-intensity treadmill interval exercise performed 2 hours before an open-water scuba dive to 18 msw (180.7 kPa) in experienced divers. Following the intervention, participants demonstrated significantly lower counts of circulating microparticles and reduced surface markers for platelet and neutrophil activation, despite no significant reduction in median VGE scores. Although exercise did not alter the bubble grades in this specific open-water profile, the results suggest that pre-dive exercise mitigated the inflammatory and microparticle responses associated with decompression stress.

In summary, studies performed in hyperbaric chambers show significantly lower VGE grades after the exercise, while no such effect was reported in open-water dive experiments, although other pathomechanisms considered in the development of DCS were affected (Dujic et al., 2004; Blatteau et al., 2005; Madden et al., 2014). Further research is required to determine optimal intensity and timeframe of exercise before performing a dive.

2.2. Vitamin C supplementation

Supplementation of ascorbic acid reduces the levels of microparticles, intensity of inflammatory reaction and alleviates FMD reduction in cerebral arteries post-dive, with no significant influence on bubble formation.

Yang et al., (2015) in a randomized cross-over study in male divers examining the effects of ingesting 2 g of ascorbic acid (Vitamin C) daily for 6 days prior to an 18 msw scuba dive,

demonstrated no significant post-dive elevations in total microparticles, specific microparticle subgroups (such as those bearing CD66b, CD41, or nitrotyrosine), or neutrophil activation, unlike the placebo group which experienced approximately twofold increases. Although ascorbic acid had no significant effect on post-dive intravascular bubble production quantified by echocardiography, the results suggest that dietary ascorbic acid supplementation can effectively abrogate the inflammatory and microparticle-associated changes triggered by diving.

Glavas et al., (2009) examined the effects of an acute oral administration of placebo, vitamin C, or a combination of tetrahydrobiopterin (BH4) and vitamin C approximately three hours prior to an open-water field dive to 30 msw in healthy male divers. Following the intervention, participants demonstrated no significant differences in post-dive venous gas bubble grades, mean pulmonary artery pressure (mPAP), or left ventricle ejection fraction (LV-EF) across the three conditions. Although the study had a small sample size and involved relatively young divers with preserved baseline endothelial function, the results suggest that the acute co-administration of BH4 and vitamin C does not reduce venous bubble formation or attenuate diving-induced alterations in cardiovascular function.

Barak et al., (2018) conducted a randomized, double-blind, crossover trial in 14 divers examining the effects of vitamin C supplementation (2 g/day for 6 days) compared to a placebo on peripheral and cerebral circulation following an 18 msw scuba dive and a separate laboratory exposure to 60% oxygen. Following the intervention, participants demonstrated that vitamin C effectively abrogated the reduction in peripheral flow-mediated dilation (FMD) in middle cerebral artery (MCA) and posterior cerebral artery (PCA) following the 60% oxygen exposure, and it significantly reduced the transient elevations in intracranial blood velocities observed 30 minutes after the scuba dive. Although vitamin C did not completely abolish the post-dive decrease in FMD, the results suggest that antioxidant supplementation can mitigate diving-induced cerebral hyperemia, highlighting that post-dive vascular dysfunctions are only partially mediated by hyperoxia and involve other diving-related stress factors.

The oxidative stress is not believed to be the primary cause of microparticle accumulation after decompression, but ascorbic acid supplementation prior to diving reduces the number of MPs to a significant degree (Yang et al., 2015; Glavas et al., 2009). Such prophylactic intervention also alleviates emergent vascular dysfunctions and inflammation (Barak et al.,

2018). Therefore, supplementation of vitamin C in addition to decompression protocols may improve safety in divers.

2.3. Anti-thrombotic pretreatment

Pontier et al., (2011) conducted a controlled experimental study in a rat model of DCS examining the effects of anti-thrombotic pretreatment with acetylsalicylic acid (ASA), heparin, or clopidogrel prior to a 45-minute hyperbaric exposure to 1,000 kPa. Following the intervention, the subjects demonstrated that clopidogrel significantly reduced DCS mortality risk, neurological DCS incidence, and the post-decompression fall in platelet count in comparison to the control group, whereas ASA and heparin offered no protective effect on DCS incidence.

Although this was an animal model study and the results cannot definitively differentiate between bubble-induced vessel wall injury and bubble-blood component interactions, the findings suggest that clopidogrel, a specific ADP-receptor antagonist, successfully reduces post-decompression platelet consumption and bubble-induced platelet aggregation. Further studies with human divers are necessary to confirm the usefulness of clopidogrel as an adequate prophylactic measure.

2.4. Oxygen-Enriched Breathing Mixtures

Breathing oxygen-enriched gas mixtures (EAN/Nitrox) during the bottom phase or decompression phase of a dive reduces venous gas bubble formation and may influence endothelial function compared to breathing standard compressed air.

Souday et al., (2016) conducted a double-blind, randomized, cross-over trial in human volunteers susceptible to bubble formation examining the effects of breathing enriched air nitrox (EAN 36% oxygen) versus compressed air during a simulated hyperbaric chamber dive to 28 msw. Following the intervention, participants demonstrated a significant reduction in intravascular bubble scores at all time points when breathing EAN, with zero decompression incidents occurring compared to three in the air group. Although the study was conducted in a dry hyperbaric chamber rather than in open water, the results suggest that EAN breathing markedly reduces venous gas bubble emboli and increases diving safety when oxygen toxicity limits are respected.

A trial was conducted by Brebeck et al., (2017) measuring the effects of breathing Nitrox28 versus compressed air during a standardized open-water dive to 24 msw. The study was performed in 108 advanced recreational divers. Following the intervention, participants demonstrated fewer venous gas bubbles overall in the Nitrox28 group compared to the air group across multiple venous sampling sites. Although only a small percentage of divers in either group were completely bubble-free after the dive (11% for Nitrox28 vs. 7% for air), the results suggest that breathing Nitrox28 helps to reduce venous gas emboli in recreational divers.

Ribičić et al., (2019) examined the bubble grades as well as FMD in brachial artery post-dive on air, nitrox 50, and nitrox 99 (where the percentage represents the fraction of oxygen in the mixture) specifically during the decompression phase following an open-sea dive to 45 msw. Following the intervention, all ten of participants demonstrated significantly reduced bubble formation after a cough when using nitrox 99 compared to air and nitrox 50. Furthermore, nitrox 99 preserved post-dive flow-mediated dilatation (FMD), whereas nitrox 50 significantly decreased it. Although the sample size was limited, the results suggest that utilizing highly oxygen-enriched mixtures like nitrox 99 during decompression significantly reduces bubble formation. Endothelial function was highly impaired in the nitrox 50 dive in comparison to air and nitrox 99 dive protocols.

The main benefit of oxygen-enriched gas mixtures is reducing the nitrogen uptake, therefore allowing for extended dives with shorter decompression protocols in the aftermath. This positive effect on VGE formation is indeed confirmed by findings of reviewed studies - bubble grades in participants who used EAN were significantly lower than on pressurized air and showed to be lower with increasing oxygen concentration in comparative study. Additionally, Ribičić et al. postulated effects of higher oxygen concentration on FMD. It was expected for higher oxygen concentrations to impair FMD to a more significant degree than air, as hyperoxia causes vasoconstriction (Brubbak et. al, 2005; Attaye et al. 2018). Although the evidence is inconclusive, the disparity between nitrox 50 and 99 users may be explained by higher bubble grade in the first group, as VGE concentration is considered another factor impacting FMD negatively (Ribičić et al. 2019).

Discussion

This narrative review synthesizes current evidence evaluating methods of prophylaxis for decompression sickness (DCS) that could provide an expansion of standard decompression protocols and further improve safety in divers. Pre-dive aerobic exercise appears to target both the precursors and consequences of accumulating bubbles; exercise performed 24 hours prior may reduce the population of pre-existing gas micronuclei (Dujic et al. 2004), while exercise performed closer to the dive mitigates the secondary inflammatory reaction, reducing microparticle shedding and neutrophil activation despite the presence of bubbles (Madden et al. 2014; Dujic et al. 2004; Blatteau et al. 2005). Biochemical and pharmacological approaches are explored as countermeasures to decompression stress, targeting microparticle shedding and platelet aggregation (Yang et al. 2015; Pontier et al. 2011). Ascorbic acid directly scavenges reactive oxygen species, preventing hyperoxia-induced oxidative stress and the subsequent release of pro-inflammatory MP (Yang et al. 2015). Clopidogrel acts on the ADP-receptor pathway and was proven to inhibit bubble-induced platelet aggregation in animal studies, halting the coagulation cascades that lead to microthrombi and vessel occlusion (Pontier et al. 2011). Furthermore, usage of oxygen-enriched breathing mixtures (Nitrox) increases arterial oxygen pressure, accelerating the diffusion and elimination of nitrogen from tissues. This directly limits the supersaturation required for bubble formation. Highly enriched mixtures have been found to preserve endothelial function (Souday et al., 2016; Brebeck et al., 2017; Ribičić et al. 2019).

In comparison to control groups in respective experiments, all of these preventive measures have a positive influence on various indicators linked with onset of DCS through distinct but complementary mechanisms. Despite these encouraging findings, several research gaps remain. There is a necessity of larger population studies regarding the covered interventions to solidify evidence and identify exact measures needed for prophylactic results. For example, discussed studies regarding aerobic exercise vary both in regard to intensity of physical exertion performed by participants and the final findings, despite confirming the protective effect. Thorough comparison of training varying in intensity and time relation to diving could lead to formulation of optimal training parameters for pre-dive exercise regimes and precisely quantify their impact.

The individual susceptibility for DCS is not yet well understood. While standard decompression algorithms attempt to limit tissue supersaturation through time and depth limits, recent data

reveal marked within-diver variability in VGE formation even when performing identical dive profiles under controlled conditions (Mitchel SJ, 2024). This underscores the need for individualized prevention strategies guided by personal risk factors—such as the presence of a patent foramen ovale (PFO), age, and body composition—rather than relying solely on rigid, algorithm-based recommendations (Mitchel SJ, 2024). Thus, integrating individualized preconditioning, appropriate gas mixtures, and targeted physiological therapies offers the greatest potential for advancing holistic DCS prophylaxis. Further research on these supplementary measures is needed to clear out the existing uncertainties and to prepare adequate guidelines.

Conclusion

There are numerous promising directions being explored in regard to DCS prevention. Pre-dive aerobic exercise, ascorbic acid supplementation, oxygen-enriched gas mixtures usage, and clopidogrel administration have been proven to positively affect levels of biomarkers linked with DCS development. With evidence on variable susceptibility to developing the condition, these interventions provide a wide range of possible prophylaxis measures that can be tailored for needs of individual divers. Studies on more numerous diver populations are necessary to further research the protective effects of mentioned interventions. This in turn will allow formulation of concrete clinical recommendations for their supplementary inclusion in eligible divers.

Disclosure

Author Contributions

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The authors declare no conflict of interest.

Declaration of AI Use

Artificial intelligence (AI) was used only for language enhancement purposes, such as grammar correction and stylistic refinement.

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