

Editorials

Aerobic demand and scuba diving: concerns about medical evaluation

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Scuba diving has become a popular recreational sport throughout the world. Although it is not a competitive sport, a certain level of physical fitness is recommended because of the physical characteristics of the underwater environment.¹ Immersion alone will increase cardiac preload leading to a rise in both cardiac output and blood pressure, counteracted by increased diuresis. Increased oxygen partial pressure and cold exposure during scuba diving will additionally increase afterload by vasoconstrictive effects and may induce bradyarrhythmias in combination with breath-holds.^{2,3} Volumes of gas in the body cavities will be affected by changing pressure, and inert gas components of the breathing-gas mixture will dissolve in body tissues and blood with increasing alveolar inert-gas partial pressure.^{4,5} During decompression, a free gas phase may form in supersaturated tissues, resulting in the generation of inert gas microbubbles that are eliminated by venous return to the lungs under normal circumstances.⁴ It has been reported that more air bubbles were detected in divers when dives were performed in the open sea rather than in hyperbaric chambers.^{6,7} Both dry and wet dives are associated with hyperoxia, increased density of breathing gas, and decompression stress, with possible formation of venous bubbles and enhancement of the inflammatory cascade.^{4,8}

However, open-water dives are also associated with immersion, the mechanical load of the breathing apparatus, a high level of physical activity, and exposure to a cold environment. Immersion in cold water results in breathing colder and denser gas and may also, by inducing peripheral cutaneous vasoconstriction in conjunction with the immersion effect, potentiate central pooling of blood more than in dry dives.^{1,3,5} Water immersion-induced changes in haemodynamic, neuroendocrine and autonomic activities have been reviewed previously.¹ Cardiovascular conditions may have an impact on these physiological changes, increasing the risk of suffering adverse events from scuba diving. Systemic hypertension may be aggravated by underwater exercise and immersion. Metabolic disorders are also of concern, since obesity is associated with both higher bubble grades in Doppler ultrasound detection after scuba dives when compared to normal subjects and with an increased risk of decompression illness.⁴

Thus, the diver's cardiovascular status is important in the assessment of fitness to dive, and some cardiovascular conditions, such as symptomatic coronary artery disease and heart rhythm disorders, should preclude scuba diving.⁹ Any history of cardiac disease or abnormalities detected during routine physical examination should prompt further evaluation and specialist referral. Recreational scuba diving

is usually performed without accurate medical examination. In 2003, the only countries requiring pre-diving medical examination were France, the UK and Australia (no longer the case in Australia), while it is still required for commercial diving.⁹ Swimming in rough water and strong currents can induce fatigue, anxiety or panic in divers.

A question of fundamental importance is: what is a level of physical fitness needed to deal with the reasonable, expected and unexpected demands of a recreational dive? The paper by Buzzacott et al investigates this topic, confirming previous research that the mean aerobic need is about 7 METs (metabolic equivalents).¹⁰⁻¹³ Nonetheless, we know that a US Navy diver must swim at least at 1.3 knots, which means 13 METs and a recreational scuba diver usually swims at 0.5 knots (5 METs) but during an emergency he could reach up to 1.0 knot (10 METs). Moreover, we have to take into account that an expert scuba diver has a better exercise efficiency compared to the non-expert, so these conclusions appear reasonable.¹⁴

Many unresolved questions remain open: for example, the reliability of the value of MET. A recent paper reported that the mean rate of resting oxygen consumption ($\dot{V}O_2$) in a sample of healthy men was 3.21 mL·kg⁻¹·min⁻¹, significantly lower than the standard resting MET value of 3.5 mL·kg⁻¹·min⁻¹.¹⁵ Also, another prediction model which included body surface area and percent body fat as predictors demonstrated relatively poor predictive ability.¹⁶ Moreover, the error in estimating resting $\dot{V}O_2$ from 1 MET increases with increasing adiposity but the 1-MET value also overestimates resting $\dot{V}O_2$ values in normal-weight persons. Therefore, the use of a more correct raw value in mL O₂·kg⁻¹·min⁻¹ might be preferable in addition to having a wider safety margin.¹⁶ Others have suggested that a peak capacity of 11 to 12 METs could be an appropriate goal for recreational divers.¹⁷ Another issue to be considered is the experience of the diver.⁹ Paradoxically a more expert diver needs a lower $\dot{V}O_{2max}$ than a non-expert.¹⁸ As Buzzacott reports: "*Dwyer's methods included swimming at a fixed depth for four minutes while pushing a board and his gas collection took place during only the last minute of steady-rate exercise, whereas our study involved free-swimming recreational divers and our data were averaged over much longer and more variable periods.*"^{10,19}

The role of oxygen demands, with or without exercise, in immersion is implicated in decompression physiology as well, particularly in processes defined as 'denucleation' and 'denitrogenation'.^{20,21} The increased oxygen content rapidly diffuses into micronuclei in exchange for nitrogen,

which is then eliminated from the body via the lungs. The oxygen is then absorbed by the surrounding tissue to cause rapid decay of the micronuclei. This theory has been supported by studies that demonstrate significantly reduced decompression-induced bubble formation in animals pre-treated with hyperbaric oxygen (HBO), believed to be owing to the elimination of bubble nuclei.²³ HBO has been observed to eliminate most of the gas nuclei in decompressed animals, thus reducing the number and size of bubbles during decompression. Reduction in the inflammatory cascade in humans has also been reported.⁸ A previous study showed that HBO pre-breathing significantly reduced decompression-induced bubble formation and platelet activation in simulated dives in an HBO chamber.⁶ Recent studies demonstrated that pre-breathing normobaric and hyperbaric oxygen in open water also decreased venous gas emboli formation, with a prolonged protective effect and repercussions on platelet activation and intracellular calcium accumulation in lymphocytes.^{7,23-26}

In our recently completed Tremiti Islands experiment to quantify underwater exercise variables, all subjects were asked to perform the same mild workload at a depth of 30 metres' sea water on an underwater bicycle ergometer at a pedalling rate of 25 rpm to ensure no difference of ventilation and gas exchange in all dives, guided by the Borg category ratio 0-10 scale at an intensity level of 3.^{7,26-27} Basic activities associated with scuba diving, such as surface swimming or walking with heavy equipment, may be enough to allow the passage of venous gas emboli through intrapulmonary arterial-venous anastomoses.²⁸ Some of the differences observed between the aerobic exercise and a non-exercise control dive related to a decompression-induced inflammatory pattern, and provide additional insight into the potential protective benefits of exercise performed before a dive.²⁹ Further study is needed to understand the potential of these benefits. In our opinion, the preventive measures to reduce decompression complications of diving include the acceptance of safe diving procedures, particularly related to descent and ascent and the exclusion of individuals with specific medical conditions. A more specific and in-water activity-related medical examination might be desirable for recreational scuba divers.

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