

Pre-Dive Vibration Effect on Bubble Formation After a 30-m Dive Requiring a Decompression Stop

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Introduction: The preconditioning of divers to reduce post-dive decompression sickness (DCS) has gained increased interest in diving medical research over the last few years. The beneficial effects of physical exercise, oxygen breathing, hyperbaric exposure, heat exposure, hydration, or nitroglycerin administration before the dive are only a few examples of ongoing research. In this work, we investigated the effects of pre-dive whole-body vibration on post-dive bubble formation. **Methods:** Following French Navy standard dive procedures, 14 healthy male military divers performed 2 identical dives 1 wk apart to 30 m of seawater (msw) for 30 min. One of the dives was randomly preceded by a 30-min whole-body vibration session (frequencies 35-40 Hz), 1 h before the dive. Post-dive bubbles were measured precordially 30, 60, and 90 min after the dive and were graded according to the Kissman Integrated Severity Score (KISS) protocol, with and without knee flexing. Arterial endothelial function was measured before and after vibration using flow mediated dilation (FMD) measurement. **Results:** A significant reduction in bubble scores was observed after the "vibration" dive. **Conclusion:** As there was no observed change in FMD after vibration, we do not believe a nitric oxide mediated mechanism is involved; rather, a mechanical dislodgement or enhanced lymphatic elimination of gas nuclei is hypothesized.

Keywords: scuba diving, decompression sickness prevention, bubble formation, whole-body vibration.

THE PREVENTION OF decompression sickness (DCS) in scuba diving is a matter of ongoing research. Although currently available decompression tables and algorithms are capable of reducing the risk of DCS, they cannot eliminate it completely (16). A substantial proportion of DCS cases are classified "unexplained" when the diver has complied with the procedures imposed by the chosen decompression model (dive computer or dive table). Because of this, a large body of research is currently directed at reducing the incidence of (micro) gas bubbles during decompression. However, research into the optimization of decompression procedures is hampered by the large inter- (and even intra-) individual variability of post-dive decompression bubble formation (4).

Another approach to reducing bubble formation after a dive is to "pre-condition" the diver prior to immersion. The general idea behind this approach is that post-dive decompression bubbles originate from the endothelial surface. Reports have been published on the

beneficial effects of pre-dive exercise (5,14), oxygen breathing (9), pre-dive hyperbaric sessions (19,24), heat preconditioning (6), hydration (15), and nitric oxide (NO) donor administration (13). Most of these experiments try to influence bubble formation by modifying biophysical or chemical properties of the endothelial surface, on which gas bubbles or nuclei are presumed to be forming. In this paper, we report the possibility of reducing post-dive bubble formation by a short bout of mechanical low-frequency vibrations of the whole body 1 h before the dive.

METHODS

The study protocol was approved by the Academic Ethical Committee of the Brussels Free University and by the French Navy Experimental Ethical Committee. After consent, 14 healthy male military divers (age range: 23-44, mean 29 yr, height: 177 ± 6 cm, weight: 79.44 ± 10.7 kg) participated in this prospective study. All divers were in good health, did not take any medication, and had never suffered from DCS. They performed two identical dives 1 wk apart. Each dive was made in open (sea) water, to a depth of 30 m, for 30 min on the bottom with a calibrated effort during the dive (fin swimming at a determined leg frequency and speed). A 9-min decompression stop was made at a depth of 3 msw according to the French Navy standard diving procedures (MN90 diving tables). All subjects abstained from diving, smoking, or physical exercise for 48 h before each dive.

In a randomized fashion, one of the dives was preceded by a whole-body vibration session of 30 min duration using a commercially available vibration mattress

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(VM 9100RM®, HHP Products, Karlsruhe, Germany). Vibration frequencies ranged from 35 to 40 Hz (rms $2.0\text{--}4.0\text{ m} \cdot \text{s}^{-2}$) along the whole body thanks to 11 motors embedded in the mattress. The subject lay motionless on the mattress during the entire vibration session, which ended 1 h before the start of the dive. Before and after the vibration session, a measurement of flow-mediated dilation (FMD) was performed at the level of the brachial artery 3-5 cm above the elbow fold according to the standard protocol described by Coretti et al. (10). In order to optimize this measurement, we simultaneously recorded the digital arterial pulse by means of a photoelectric plethysmograph in order to freeze the echographic image at the exact time of maximal dilation.

After the dive, precordial venous gas emboli (VGE) were measured with a two-dimensional Doppler (EZ-Dop®, Compumedics, Singen, Germany) by a single, experienced Doppler operator who was blinded as to whether a vibration had preceded the dive. Doppler measurements were made at 30, 60, and 90 min after the dive, both at rest and after knee flexions, and graded according to the Kissman Integrated Severity Score (KISS) methodology (32). Bubbles scores were also graded according to our previously described simplified Doppler Bubble Grading System (DBGS). In short, VGE are classified Low Bubble Grade (LBG) for Spencer scores lower than 2 (occasional bubble signals), High Bubble Grade (HBG) for Spencer score 2 (frequent to continuous bubble signals), and Very High Bubble Grade (HBG+) for Spencer Scores reaching 3 and above. For easy comparison, DBGS can be grouped into High Bubble Grades (HBG and HBG+) and Low Bubble Grades (LBG and Zero bubbles) (31). KISS scores and FMD measurements were statistically analyzed using a nonparametric test (Wilcoxon) after Kolmogorov-Smirnoff verification on GraphPad Prism 4.0 on the PC. DBGS were compared using Fisher's exact test, two-tailed.

RESULTS

None of the dives resulted in DCS symptoms. After the control dives, the mean KISS score ranged from 7.69 (SD 6.20) at rest to 22.82 (SD 14.36) after knee flexion. After the "vibration dive," the mean KISS scores were 5.29 (SD 6.80) and 13.62 (SD 13.58), respectively, without and with knee flexion. A reduction in KISS score was observed after the "vibration dive," both without and with knee flexion (Fig. 1). For the measurements at rest, this difference just failed to meet statistical significance ($P = 0.06$); after knee flexion the difference was statistically significant ($P = 0.02$).

After the vibration dive, DBGS scores were lower both for measurements at rest, with Low Bubble Grades 71.4% (30/42) vs. 54.8% (23/42). After knee flexion, Low Bubble Grades were 45.2% (19/42) vs. 21.4% (9/43). For rest, this difference was not statistically significant ($P = 0.1745$); however, after knee flexion, the difference was significant ($P = 0.0363$). A small reduction of FMD was observed after vibration [9.41 (SD 2.9) vs. 10.18 (SD 3.95)], but this failed to reach statistical significance ($P = 0.18$).

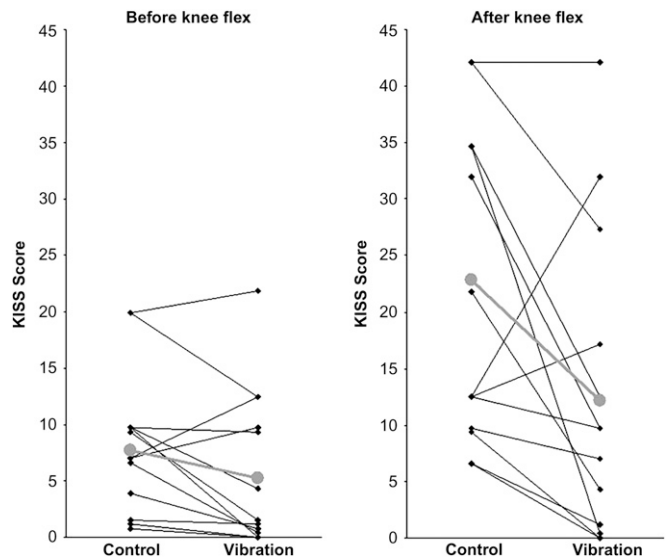


Fig. 1. Individual Kissman Integrated Severity Scores (KISS) after a dive without (Control) and with (Vibration) whole body vibration for 30 min, ending 1 h before the dive. Bubble scores are shown before (left panel) and after (right panel) knee flexion. Black diamonds are individual KISS scores, large grey circles represent the mean KISS scores.

DISCUSSION

Free nitrogen bubble formation during the decompression phase of a dive is considered the principal cause of DCS. VGE are the observable phenomena by which the decompression "stress" is generally measured (32). The origin and formation of these VGE is incompletely understood. It is generally hypothesized that they emanate from gas nuclei that are present in endothelial crevices between or within the endothelial wall surface. Nitrogen, diffusing out of the tissues during decompression, would preferably fill these gas nuclei rather than transfer as molecular nitrogen into the blood. This causes the gas nuclei to "grow" and spill out nitrogen gas bubbles into the bloodstream, where they either grow or shrink depending on surface tension and free gas tensions (4).

There is, as such, only indirect evidence of the presence of these gas nuclei; however, if this hypothesis is correct, eliminating gas nuclei before the dive would result in lower bubble production after the dive. Many research efforts have focused on the role of nitric oxide as a marker of the general "health" of the endothelium, and as a mediator of the hydrophilic/hydrophobic properties of the endothelial wall (8,13,23,38). Furthermore, the respiration of oxygen before the dive (9), or again, a short hyperbaric exposure (19,24), seems to be capable of reducing the nitrogen content of these nuclei and exerting a protective role on post-dive VGE.

Other research has focused on the consequences of (microscopic) bubble formation: even if no or little VGE can be detected by Doppler or echocardiography examination, significant alterations in arterial endothelial function (as measured by FMD) have been observed after a decompression dive (8)—these can (at least par-

tially) be prevented by intense physical activity some hours before the dive (5,3,12), or exposure to high environmental temperature (6). The role of heat shock proteins as a primary mediator of diving acclimatization has been investigated (17).

For this study, we hypothesized that gas nuclei may be also depleted by mechanical means, rather than biochemical means. If a gas nucleus is adherent to the endothelial wall, then a vibration might be able to dislodge it. This effect is commonly observed during standard Doppler VGE detection: usually, much higher bubble grades are observed after the subjects have performed several deep knee flex exercises. We have also observed that applying a local or whole-body vibration after a dive resulted in a sometimes dramatic increase in the number of VGE observed in the heart cavities by echocardiography (data not shown). If liberated into the venous blood stream before the dive, these gas bubbles would be rapidly eliminated by the combined effects of its surface tension and the gas tensions in the blood (4). The end result would be less available gas nuclei capable of growing during the decompression phase, and less VGE.

The choice of vibration frequency (35-40 Hz) and duration (30 min, ending 1 h before the dive) was arbitrary and based on practical considerations (commercially available vibration mattress, experiment planning time schedule). However, several indications were available to justify these choices. First, our clinical experience of DCS cases occurring immediately following a vibrating boat ride after a dive and, conversely, divers seemingly protected from post-dive VGE after very provocative dives when a fast inflatable boat ride transported them to the dive site, led us to hypothesize that low-frequency vibrations may play a role. Secondly, previous observations from our research group had shown that acute exposure to these vibration frequencies after a dive is capable of increasing the number of observed VGE using echocardiography. Thirdly, it has been reported that in professional divers and caisson workers, the risk for DCS is higher during the first days after returning from leave and subsequently decreases—this is attributed to progressive depletion of adherent gas nuclei by repeated decompressions (11,21). The protective effect of any means of nuclei depletion might thus last for a couple of days (the time for the “stock” of gas nuclei to return to baseline value).

The reduction in VGE after a “vibration” dive, as demonstrated by the KISS score reduction, just failed to reach significance at rest ($P = 0.06$), but was significant after knee flexion ($P = 0.02$). The knee flexion maneuver, by increasing the shear stress on the vessel wall, increases the liberation of any existing, adherent gas bubbles. Therefore, the amount of VGE observed after knee flexion reflects more accurately the amount of gas bubbles actually present than the values at rest. Our results seem to suggest that as a result of pre-dive vibration, the sources of potential post-dive gas bubbles (i.e., vessel wall gas nuclei) have been reduced in number. The exact mechanism by which gas nuclei are eliminated from the

vessel wall by mechanical vibration remains to be clarified.

It has been shown that external limb vibrations cause locally turbulent flows in blood vessels (36). These create shear forces that could mechanically dislodge and “sweep” away the gas nuclei. This is illustrated by our personal observations of the prompt increase of post-dive VGE after a few seconds of exposure on the vibrating mattress (unpublished).

The same shear forces might modify the vessel wall in a biochemical way by stimulating or inhibiting enzymatic processes, such as endothelial nitric oxide production or heat shock proteins. Whole-body vibration has been shown to decrease arterial stiffness and increase regional blood flow (27,33) through an increase in NO production (30). However, as our vibration sessions did not result in a significant modification of the endothelial reactivity, as indicated by the FMD measurements, we do not think NO production or vessel wall reactivity was significantly altered in our study subjects. Previously it has been shown that administration of nitroglycerin prior to a dive may reduce bubble formation after the dive and the use of short-acting NO-donors has been suggested as a protective measure (13). Whether the combination of NO-donors and low-intensity vibration would lead to an even greater reduction in VGE remains to be investigated.

Prolonged and intense local vibration has been shown to induce local and regional vasoconstriction by means of an as yet unclear mechanism [possibly an increase in endothelin production, a reduction in endothelial nitric oxide synthetase (eNOS) activity, and/or an increase in α -2 adrenoceptor expression] (18,20,22). Vibration effects are extensively studied in the context of occupational medicine as a cause of vibration-induced Raynaud’s syndrome. The frequency, intensity, and duration of vibrations needed to induce these phenomena, however, seem to be much higher than the vibration protocol applied in our study (37).

It may be speculative, but it seems that the effects of vibration on vascular tone exhibit a dose-response curve, with positive effects when short and low intensity, almost neutral effects in the protocol we have used, and negative effects when more prolonged or at higher intensities or frequencies (7,26,29). The vibration frequencies used in our study are several orders of magnitude lower than those that have been shown in animal experiments to induce gas bubble growth from micronuclei (1,35) and are substantially lower than the resonance frequency of microbubbles in blood vessels (34). Therefore, a purely resonance effect on micronuclei by these low-frequency vibrations is unlikely.

Finally, apart from a mechanical effect and an effect on endothelial enzymatic processes, a third, regional effect may be involved. Vibrations applied to a limb have been shown to increase the rate of lymphatic drainage (25,28). Moreover, oxygen breathing improves lymphatic drainage of microparticles and this may partly explain the beneficial effects of oxygen in the treatment of decompression disease (2). Evacuation of gas bubbles

(and nuclei) is possibly happening in part by means of lymphatic fluid. Therefore, it cannot be excluded that tissue and endothelial gas nuclei are eliminated by an increased lymphatic flow during the whole-body vibration session.

In this carefully controlled prospective, observer-blinded randomized experiment, a single vibration session before the dive has been shown to have a protective effect on bubble formation after the dive. This observation could represent a simple and very efficient way to improve diving safety by reducing the pre-existing nitrogen nuclei from which VGE originate during decompression. Further study is needed to exactly determine the mechanism of this reduction.

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