Aerobic Exercise 2 Hours Before a Dive to 30 msw Decreases Bubble Formation After Decompression

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Background: A single bout of aerobic exercise 24 h before a dive significantly reduces the formation of circulating venous gas emboli (VGE) on decompression. The purpose of this investigation was to determine the effect of aerobic exercise 2 h before a dive. ***Methods:*** There were 16 trained military divers who were compressed to 30 msw (400 kPa) for 30 min breathing air in a dry hyperbaric chamber at rest, then decompressed at a rate of 10 $m \cdot min^{-1}$ with a 9-min stop at 3 msw. Each diver performed two dives 3 d apart, one with and one without exercise that consisted of running for 45 min at 60–80% of maximum heart rate (estimated as 220 − age). VGE were graded according to the Spencer scale using a pulsed Doppler detector on the precordium at 30 min (T30) and 60 min (T60) after surfacing. ***Results:*** Mean bubble grades at T60 were 1.25 for control dives and 0.44 for dives preceded by exercise, the difference being highly significant. None of the divers showed an increase in venous bubble grade after exercise. ***Conclusion:*** Like exercise 24 h ahead, 45 min of running 2 h before a dive decreases bubble formation after diving, suggesting a protective effect of aerobic exercise against DCS. The threshold of exercise intensity and duration necessary to change venous circulating bubbles is unknown. Mechanisms underlying the protective effect of exercise remain unclear. Rather than altering the nitrogen elimination rate, exercise may affect the population of gaseous nuclei from which bubbles form.

Keywords: diving, gas nuclei, decompression sickness, heat shock protein, nitric oxide.

DECOMPRESSION sickness (DCS) is caused by circulating bubbles of inert gas in blood and tissues resulting from supersaturation during decompression. At present, Doppler-detected venous gas emboli (VGE) are widely used as an indicator of decompression stress. Although bubbles are frequent after symptom-free dives, the occurrence of many bubbles is clearly linked to a high risk of DCS (16). Regular activity, like running, is a common practice in the military diving community. The objectives are to stay in a good physical shape and to maintain a high level of cardiovascular fitness.

Intense physical exercise before diving has long been considered an additional risk factor for DCS (22). It is suggested that muscle contraction and tissue movement produce gas nuclei leading to bubble formation and a corresponding increase in the risk of DCS (10). Recently, several studies indicate this notion needs updating.

It has been reported that exercise training weeks before dives could reduce the incidence of neurological DCS in pigs (3) and rats (19). Similarly in humans, data about aerobic fitness as a DCS protective factor are described. It has been demonstrated that aerobically trained runners appeared to be at lower risk for venous bubbling than sedentary subjects (1,6). Moreover, recent studies in rats have shown that a single bout of high-intensity aerobic exercise 20 h before the dive suppressed bubble formation and prevented death with no effect at any other time (48, 10, 5, and 0.5 h prior to the dive) (23,25). In a study of 12 divers, a single bout of aerobic exercise 24 h before a dive significantly reduced venous gas emboli and consequently could have a preventive effect on occurrence of DCS (8). It was also observed that the incidence of VGE decreased when the rest interval from an anaerobic exercise (150 deep knee squats over a 10-min period) to altitude depressurization lengthened and was performed 1–2 h before exposure (7). Thus, it appeared relevant to determine the effect of a single bout of aerobic exercise 2 h before a dive on VGE formation in human volunteers.

METHODS

We recruited 16 trained military divers, ages 24–41 yr (mean: 33.4), who were medically fit for diving. The subjects were all experienced divers with 300–3000 dives (mean: 970). Their body mass index varied between 20.4 and 28 $kg \cdot m^{-2}$ (mean: 24). None of them had experienced DCS in the past. The protocol was conformed to the principles of the declaration of Helsinki and all subjects gave written, informed consent. Subjects were asked to avoid physical exertion during the 2 d that preceded the dive.

Each subject performed a single bout of submaximal exercise consisting of endurance running at an intensity

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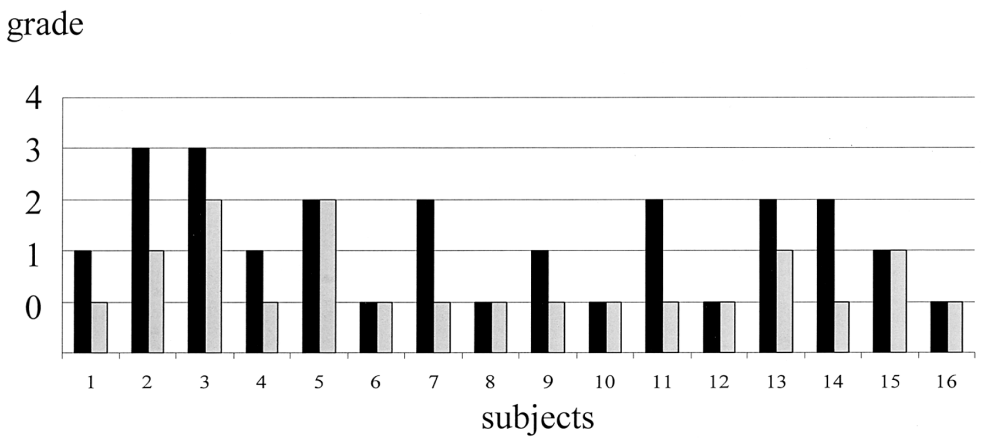

Fig. 1. Individual Spencer scores for venous gas emboli 60 min after simulated dives to 30 msw for 30 min. Black bars represent control dives and gray bars represent dives preceded by exercise.

of 60–80% of maximum theoretical heart rate (220 − age) for a total exercise session of 45 min. Heart rate was recorded continuously during the test using a HR monitor (Polar Vantage, Kempele, Finland). The divers were then compressed in a hyperbaric chamber (Sainte-Anne hospital, Toulon, France) to 30 msw (400 kPa) at a rate of 15 m · min^{-1}, breathing air and remaining at rest at pressure for 30 min. They were decompressed at a rate of 10 m · min^{-1} to 3 msw, where they remained for 9 min before they were decompressed to the surface at the same rate (French Navy MN90 procedure). Each diver performed two dives 3 d apart, one with and one without exercise 2 h before the dive. The order of the two dives was randomly allocated.

VGE detection on the precordial area was performed with a pulsed Doppler 2 Mhz by an experienced operator. We have previously shown that pulsed Doppler is more sensitive and less operator dependant than continuous Doppler (2). Moreover, it is suggested that there is a good correlation between different pulsed Doppler methods, with and without image-assisted detection (4).

During bubble detection, divers were supine for 3 min at rest, then, in order to improve the detection, two successive lower limbs flexions were performed. The Spencer scale was used to evaluate the signals of bubbles and the determination of the bubble grade was carried out at 30 and 60 min after surfacing (21). A persistent and stable bubble score during at least 10 systoles after 2 successive lower limbs flexions was considered a consistent finding. Differences in bubble grade (at peak) between groups were determined using the non-parametric Wilcoxon test with $p < 0.05$ as the level of significance.

RESULTS

None of the divers suffered from DCS after the dives. For all the dives, the maximum bubble count (bubble peak) was observed 60 min after surfacing. Mean bubble grade was 1.25 at rest and 0.44 after exercise ($p = 0.0062$). None of the divers showed an increase in venous bubble grade after performing the exercise (**Fig. 1**).

DISCUSSION

The present study puts forward a previously unreported finding that a single bout of an aerobic exercise 2 h before a dive results in lower bubble scores in human volunteers following decompression. The mechanisms underlying the protective effect of exercise remain unclear and numerous parameters should be evaluated. Rather than altering nitrogen elimination, exercise may alter the population of gaseous nuclei from which bubbles form (23). It has been generally postulated that these preformed gaseous nuclei must be present for bubbles to develop at the modest gas supersaturations encountered by divers, aviators, and astronauts (10,27). These nuclei are not stable in blood and two major stabilizing factors are suggested: 1) the existence of intercellular hydrophobic crevices on the endothelial surface that trap gas nuclei (10,12); and 2) the concept of surface-active molecules like surfactant, platelets, or proteins that coat gas nuclei (27). The half-life and the faculty for nuclei to initiate bubble formation during decompression depend on these stabilizing factors. Previous studies have hypothesized the lifetime of those bubbles could be on the order of minutes to a few hours (7), and it took about 10–100 h to regenerate the nuclei population (27).

It has been suggested that the protection from bubble formation by appropriately timed exercise could be related to biochemical processes. A large number of studies have shown that in normal control animals (20) and healthy subjects (11), exercise training improves endothelial function and protection against cardiovascular diseases (11). The likely mechanism by which aerobic exercise activates endothelial function is an increase in vascular shear stress resulting from increased blood flow. This beneficial effect seems essentially related to an increase in vascular endothelial nitric oxide (NO) bioavailability (increase in NO production and/or decrease in NO inactivation) (11). Aside from effects on vascular tone, it has been established that NO inhibits leukocyte and platelet adhesion under low and high shear conditions (17) and could reduce hydrophobicity of the endothelial wall, reducing the number of nuclei adhering to the surface (23). It is speculated that physical activity may trigger synthesis of a molecular species that is expressed in the endothelium about 20 h later, leading to an increase in endothelial NO synthase (eNOS) activity through activation of several signal transduction pathways (25). However, it has been shown that bubble production is increased by NO

blockade in sedentary but not in exercised rats (24). This indicates that the exercise effect may be mediated by factors other than nitric oxide.

A few studies have focused on other endothelial protective mechanisms induced by exercise. In vitro application of laminar shear stress to cultured endothelial cells involves changes to the architecture of the vascular wall that decrease the turbulence of blood flow, upregulate antioxidant defenses, and increase mediators of anticoagulant pathways such as protacyclin (15). Heat shock proteins (HSP), present in most cells, including endothelial cells, play a key role in normal cellular homeostasis and protection from cell damage in response to stress stimuli. However, the precise functions of these proteins have not been completely delineated (14).

It is well documented that endurance exercise is a stressor that increases the HSP expression (26). Human studies have shown that HSP 70 mRNA concentration in muscle cells was significantly increased 30 min and 3 h after the end of a single exercise bout (18). HSP 70 levels were also increased in peripheral blood leukocytes 0, 3, and 24 h after running (9). It has also been demonstrated that heat shock pretreatment before diving enhanced the expression of HSP70 and protected rats from air-embolism-induced lung injury (13). Moreover, several investigators have focused on the interaction of eNOS with HSP90, emphasizing a possible close link between HSP and the endogenous NO pathway (11). However, a recent study on heat shock preconditioning before diving supports the findings that HSP90 is less heat induced than HSP70 and that HSP90 and eNOS are probably less important for the protective mechanism against bubble formation (5). Thus, it is conceivable that exercise-induced HSP70 production affects bubble formation after diving by a different mechanism than the NO pathway. The real bioprotective mechanism of HSP70 against DCS has not yet been described and requires further research.

Regarding the limitations of our study, we did not use a precise calibration for the physical exertion protocol. In the future, it would be interesting to determine each subject's aerobic fitness (maximal oxygen uptake; $\dot{V}O_{2max}$) by a maximal exercise test before the experimental procedure. Moreover, our findings may be considered limited because of the small sample size. It would also be useful to perform a longer air dive in order to produce a significant and a reproducible amount of bubbles.

To date, the threshold of exercise intensity and duration of exercise necessary for changes in venous circulating bubbles is unknown. Further investigations are needed to determine the real pre-dive/exercise protective latency in divers.

CONCLUSION

This study demonstrates that a single bout of aerobic exercise 2 h predive decreases venous gas bubble formation in man. These results could have considerable implications for DCS prevention. Further work is needed to elucidate the mechanisms underlying this exercise-induced reduction in bubble formation.

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