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Exercise effects on central venous nitrogen tensions after simulated non-decompression dives

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Muth C-M, Staschen C-M, Warninghoff V, Van Laak U, Radermacher P. Exercise effects on central venous nitrogen tensions after simulated non-decompression dives. Undersea Hyperbaric Med 1994; 21(3):297–303.—In five subjects we examined the effect of exercise on the pattern of central venous (right atrial) N_2 tensions (Pv_{N_2}) after ascent from simulated non-decompression dives. The dives consisted of exposure to air at 3 bar for 20 min with 10 min of exercise (workload 75 W) at depth to achieve near-complete N_2 saturation of the muscles. After the dive the subjects rested or, on another day, exercised for 30 min (workload 100 W) starting 10 min after completing the ascent. Blood samples taken every 10 min until the 60th min and 90 min after the dive were analyzed for Pv_{N_2} using a manometric Van Slyke apparatus. The amount of N_2 eliminated was estimated from the Pv_{N_2} by adapting the Fick principle. Immediately after the ascent, Pv_{N_2} were 950 ± 39 and 942 ± 27 mmHg, respectively, in the rest and experiment series. In the rest experiments Pv_{N_2} continuously decreased to 606 ± 8 mmHg 90 min after the dive, remaining significantly higher ($P < 0.05$) than before the dive. Exercise caused the Pv_{N_2} to increase beyond the corresponding levels of the rest experiments ($P < 0.05$ at 20 and 30 min exercise). After the exercise Pv_{N_2} rapidly declined, reaching predive levels 60 min after the ascent. Exercise increased N_2 elimination to 970 ± 143 ml, whereas it had been 311 ± 61 ml ($P < 0.05$) in the corresponding phase of the rest experiments. We conclude that if extensive supersaturation and bubble formation can be avoided, such as probably was the case in our shallow non-decompression dives, exercise after the ascent accelerates N_2 elimination.

decompression, exercise, central venous nitrogen tensions, nitrogen elimination

Divers breathing compressed air absorb N_2 while at underwater pressure and eliminate it during and after decompression. Exercise substantially varies the N_2 uptake and elimination rates (1): although exercise at depth increases the risk of decompression sickness (2), exercise during ascent shortens the decompression stops (3). No data are available on the effects of postdive exercise on the incidence of decompression sickness, but textbooks of diving medicine suggest that exercise after completing the ascent should be avoided (3, 4) because of bubble formation resulting from tribonucleation (5), since these bubbles are slowly eliminated due to isolation

from the circulation (4). If extensive supersaturation and bubble formation during the dive do not occur, however, such as is likely in shallow non-decompression dives, exercise after the ascent should accelerate N_2 elimination.

Therefore, we investigated the effect of moderate exercise on N_2 release after ascent from simulated non-decompression dives. Nitrogen elimination was monitored by measuring central venous (right atrial) N_2 tensions (Pv_{N_2}) because to a first approximation central or mixed venous N_2 partial pressures represent a perfusion-weighted average of tissue P_{N_2} at a given time (6).

METHODS

Subject characteristics

Five males [age 37 ± 8 (SD) yr, height 178 ± 8 cm, weight 75 ± 9 kg], all nonsmokers with normal pulmonary function (vital capacity 6.1 ± 0.7 liter), participated in this study. The subjects were experienced scuba divers with regular diving activity. Before the investigation they underwent medical examination according to the standards of the German Navy diving and compressed air work regulations (divers, submariners, and frogmen adaptability). All procedures were explained in detail, and informed consent was obtained.

Before the simulated dives, central venous catheters were advanced into the right atrium after puncture of a large forearm vein, and two blood samples were obtained for determination of predive central venous P_{N_2} (Pv_{N_2}) and for equilibration with air. Further blood samples were drawn immediately after the simulated dive, i.e., 1–2 min after ambient pressure of 1 bar had been reached. Subsequent sampling followed 10, 20, 30, 40, 50, 60, and 90 min after the dive.

Nitrogen pressure

The central venous N_2 partial pressures were determined with a manometric Van Slyke apparatus (7), as described previously (8), using a technique developed by Klocke and Rahn (9) for urine analysis and adjusted for blood samples (10). The maximum N_2 tensions in the blood were expected to be 2–3 times as high as the normal value and, consequently, the extractable amounts of N_2 too large to handle. Therefore the volume of a single blood sample was reduced from 25 to 10 ml. After collection, samples were analyzed within 6 h.

The manometric measurements were performed by the same persons (P.R and C-M.M) as in previous studies (8, 11, 12) where the reproducibility of the P_{N_2} in blood samples equilibrated with the air had been shown to be 0.9% (566 ± 5 mmHg). Intraindividual variations of baseline Pv_{N_2} were 10–20 mmHg depending on the concomitant day-to-day differences of barometric pressure ranging from 748 to 781 mmHg. Therefore, a difference of at least 10 mmHg between two corresponding intraindividual measurements was regarded as significant.

Diving profile

The simulated dives were performed in a dry hyperbaric chamber (HYDRA, Schiffahrtmedizinisches Institut der Marine, Kiel–Kronshagen, Germany) and consisted

of exposure to air at 3 bar corresponding to a depth of 20 m. The chamber was pressurized at a constant rate of 1 bar/min. Five minutes after reaching depth, the subjects began to exercise, the workload consisting of a 75-W step exercise (13) for 10 min. This workload approximates that of sport dives at this depth (1). The exercise period was limited to 10 min to allow heart rate to return to normal before ascent. After a bottom time of 20 min, the chamber was decompressed at a constant rate of −0.4 bar/min. During the entire ascent the subject remained at rest in a comfortable, semisupine position. All subjects were investigated twice; on one day remaining at rest after the dive and on a second day exercising after completing the ascent. This exercise consisted of a bicycle ergometer workload of 100 W for 30 min beginning 10 min after the end of the simulated dive. The order of the rest and exercise investigations was randomized. To ensure complete N_2 washout after the first investigation, at least 48 h elapsed between two experiments in each test person.

For each subject the area under the Pv_{N_2}/time curve in the exercise time interval was integrated to derive a mean Pv_{N_2} in the two experimental settings. The amount of nitrogen eliminated (V_{N_2}) was then estimated in analogy to the Fick principle: assuming a cardiac output (CO) of 5.5 liters/min at rest and 12 liters/min during exercise (14), V_{N_2} was calculated as

$$V_{N_2} = (\text{mean } Pv_{N_2} - \text{arterial } P_{N_2}) \cdot \alpha \cdot CO \cdot 30$$

where α is the solubility of N_2 in blood (0.0149 ml/ml blood at 1 atm abs) (15) and 30 is the duration of the exercise period in minutes. For this purpose, arterial P_{N_2} was substituted by the predive Pv_{N_2}, because the arterial P_{N_2} equals the Pv_{N_2} under steady state atmosphere conditions (16) and returns to predive levels within a few minutes after ascent (12).

All data are expressed as mean SEM. Statistical analysis was performed using the non-parametric Friedman rank-sign analysis of variance and the Page test (17) for multiple comparisons to detect differences between pre- and postdive Pv_{N_2}. As variations between the two experimental settings, if present, were expected to be induced by the exercise challenge, the differences between corresponding Pv_{N_2} values in the two experimental settings were evaluated during and immediately after this exercise period, i.e., from the 10th to 40th min after ascent. These differences were analyzed with the Friedman test and, subsequently, the non-parametric Wilcoxon rank-sign test for paired variables. The estimated amount of N_2 eliminated was tested with the Wilcoxon rank-sign test for paired variables. Significance was assumed when $P < 0.05$.

RESULTS

Figure 1 shows the time course of Pv_{N_2} (all values reported as mean ± SEM). Before the dive as well as immediately after the ascent, Pv_{N_2} were not statistically different in the rest (579 ± 4 and 950 ± 39 mmHg, respectively) and the exercise (583 ± 4 and 942 ± 27 mmHg, respectively) experiments (both $P < 0.05$). In the rest experiments Pv_{N_2} levels continuously decreased to 606 ± 8 mmHg 90 min after the ascent, remaining significantly higher ($P < 0.05$) than before the dive. In contrast, in the exercise series, Pv_{N_2} levels increased again after the initial postdive decline. The difference between the corresponding Pv_{N_2} of the two experimental series was

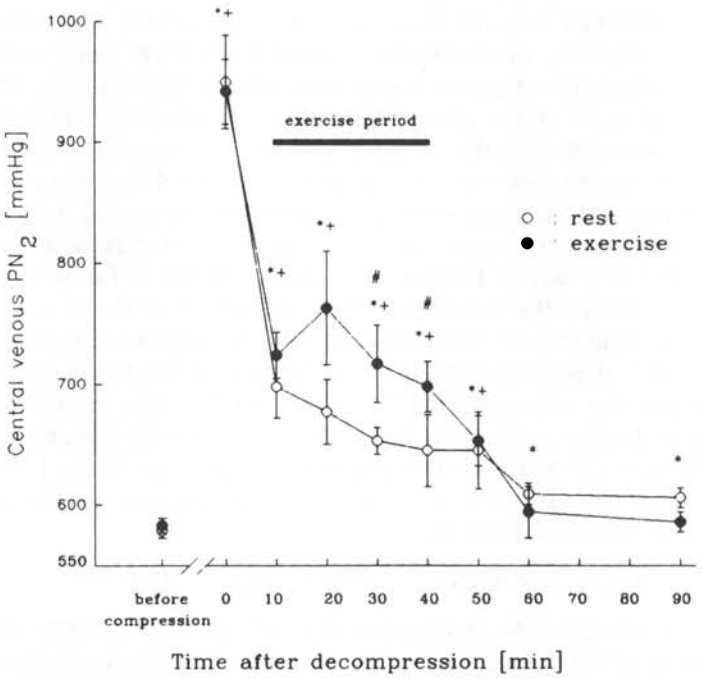

FIG. 1—Time course of central venous P_{N_2} (v_{N_2}) before and after diving with the subjects resting (*open circles*) or exercising (*solid circles*) after the ascent. Data are presented as mean ± SEM ($n = 5$), time "0 min" refers to surfacing time. # Denotes a significant difference ($P << 0.05$) between the rest and exercise experiments; + and * denote significant differences when compared with the values before the dives in the exercise (+) and the rest (*) series, respectively. *Note* that 60 and 90 min after the ascent v_{N_2} was still significantly higher ($P < 0.05$) than the predive values in the rest experiments, whereas there was no difference in the exercise series.

significant ($P < 0.05$) 20 min after the start and at the end of the exercise period, i.e., 30 and 40 min after completing the ascent. After the exercise challenge Pv_{N_2} values rapidly decreased and 60 min after the dive they had returned to baseline levels (586 ± 8 mmHg). Between the 10th and 40th min after the ascent, i.e., during the exercise time interval, the amount of N_2 eliminated was estimated to be 311 ± 61 ml in the rest experiments and 970 ± 143 ml (mean ± SEM, $P < 0.05$) during the exercise challenge (Fig. 2).

DISCUSSION

The major finding of this study was that, similar to the effects of exercise during decompression, exercise accelerated N_2 elimination after non-decompression dives that probably did not result in substantial bubble formation.

Nitrogen elimination was monitored by measuring central venous N_2 tensions (Pv_{N_2}), because to a first approximation Pv_{N_2} reflects a perfusion-weighted average of tissue P_{N_2} at a given time (6). Pv_{N_2} is a relatively crude index of N_2 washout because it cannot take into account all the different factors affecting N_2 elimination

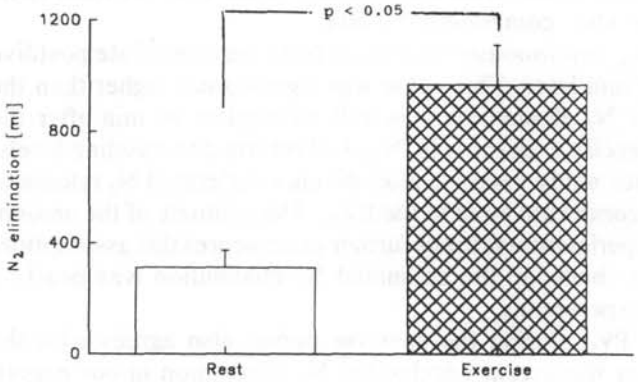

FIG. 2—Estimated amount of N_2 eliminated during the postdive exercise period, i.e., between the 10th and 40th min after completing the ascent in the rest (*open columns*) and exercise (*hatched column*) series (n = 5, mean ± SEM).

from the tissues, among which are variable tissue volumes, blood flow, N_2 solubilities, or an exercise-induced redistribution of blood flow to the muscles at the expense of other organs such as, for instance, in the splanchnic region. The latter, however, was unlikely to assume major importance in our experiment: upright exercise with a workload similar to our study (18) only reduced the blood volume in the abdominal organs by about 10% in healthy subjects, corresponding to a 15–20% reduction in regional blood flow (19). Hence, the postdive exercise challenge in our study probably did not substantially alter the overall N_2 release from the tissues. Further argument is in favor of measuring Pv_{N_2} as an appropriate monitor of overall N_2 washout: Pv_{N_2} as a perfusion-weighted average of tissue P_{N_2} at a given time reflects the different tissue blood flows, and adapting the Fick principle allowed us to grossly estimate the amount of N_2 eliminated (V_{N_2}) during exercise. The latter assumption is underscored by previous results on direct V_{N_2} measurements: the average amount of N_2 eliminated was estimated to be 311 ± 61 ml in our subjects between the 10th and 40th min after the dive in the rest experiments, which agrees well with the directly measured V_{N_2} of a resting subject ranging from 270 to 370 ml in the same time interval after a 60-min exposure to air at 2.8 bar of ambient pressure (1).

Before the dives Pv_{N_2} was 579 ± 8 mmHg in the rest and 583 ± 8 mmHg in the exercise experiments, respectively, which agrees with normal values reported by others for healthy subjects (1, 7). The relatively high variability of these measurements, when compared with previous studies (8, 10, 12), was caused by the day-to-day concomitant changes of the atmospheric pressure: barometric pressures on the experiment days ranged from 748 to 781 mmHg, resulting in concomitant 25 mmHg differences of alveolar P_{N_2} and, consequently, arterial P_{N_2}. Inasmuch as arterial P_{N_2} equals Pv_{N_2} (16), these different atmospheric pressures are reflected in the Pv_{N_2} measurements.

Immediately after the dive the mean Pv_{N_2} was about 950 mmHg in the two groups, with the highest individual value being 1,039 mmHg. With an ambient pressure of 3 bar at depth, a maximum Pv_{N_2} of about 1,700 mmHg was theoretically possible provided that all tissues had been saturated with inert gas. The difference is due to the short exposure to the hyperbaric environment. After 20 min of bottom time, "slow" tissues such as skin or fat (3) were incompletely saturated with N_2. Furthermore, the 5 min of ascent from 3 bar to atmospheric pressure had already induced desaturation in "fast" tissues (3). Since Pv_{N_2} is the perfusion-weighted average of

all tissue P_{N_2} at a given time (6), it had to be substantially lower than the theoretical maximum levels immediately after completing the dive.

In the rest experiments $P_{V_{N_2}}$ continuously decreased from the immediate postdive levels to 606 ± 8 mmHg 90 min later. This value was significantly higher than the predive $P_{V_{N_2}}$, indicating that N_2 elimination was still incomplete 90 min after the ascent. In contrast, in the exercise experiments $P_{V_{N_2}}$ had returned to baseline levels. This result suggests that in our study exercise after diving accelerated N_2 release at least from those tissues that contribute most to the $P_{V_{N_2}}$. The estimate of the amount of N_2 eliminated in the two experimental settings further underscores this assumption: during the postdive exercise challenge the estimated N_2 elimination was nearly 3 times as high as in the rest experiments.

The transient increase in $P_{V_{N_2}}$ during the exercise period also agrees with the assumption that exercise after the ascent accelerated N_2 elimination in our experiments. Muscles of the legs had probably been almost saturated with N_2 during the bottom time: a workload of 75 W at this depth is equivalent to about half maximal exercise (1) corresponding to a muscle perfusion rate of about 30 ml · min^{-1} · 100 g^{-1} (20). Since N_2 exchange is perfusion limited (21), N_2 tissue half time would be 2.3 min (1). The 10 min of exercise together with the total bottom time of 20 min would yield a theoretical muscle N_2 saturation of about 95% (21) at the end of the dive. In the resting subjects, muscle perfusion can be assumed to decrease to about 3 ml · min^{-1} · 100 g^{-1} (20) after the dive, with a corresponding N_2 tissue half time of 23 min (1). Hence, 10 min after the dive, when the subjects began to exercise, only about 25% of the excess muscle N_2 had been washed out (21). Increased muscle perfusion rate then enhanced N_2 release into the venous circulation, and $P_{V_{N_2}}$ as a perfusion-weighted mirror of tissue P_{N_2} at a given time increased because of greater contribution of tissues with relatively elevated P_{N_2}.

We can only speculate whether the exercise-induced rise in $P_{V_{N_2}}$ led to an increased chance of bubble formation, but under our investigational conditions of a safe, routine, non-decompression diving profile with experienced scuba divers no sign of decompression sickness was observed. In addition, in a previous investigation $P_{V_{N_2}}$ levels similar to those during the exercise period had not caused bubble formation detectable with ultrasound (12). It could be conjectured that the transient increase in $P_{V_{N_2}}$ might contribute to enhanced risk of decompression sickness after more strenuous dives, but to date there is no evidence that this really occurs.

In summary, we investigated the effect of exercise on the kinetics of central venous P_{N_2} after ascent from simulated, non-decompression dives. $P_{V_{N_2}}$ was measured to monitor N_2 washout from the tissues because $P_{V_{N_2}}$ is a perfusion-weighted average of tissue P_{N_2} at a given time. In contrast to the rest experiments, $P_{V_{N_2}}$ returned to baseline levels within 90 min after diving when the test person exercised, indicating more rapid N_2 washout. During the exercise period the amount of N_2 eliminated was estimated by adapting the Fick principle 3 times as high as in the corresponding time of the rest experiments. Exercise caused a transient increase in $P_{V_{N_2}}$ which, in our experiments, was not associated with any symptoms of decompression sickness.

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