

Nitrogen partial pressures in man after decompression from simulated scuba dives at rest and during exercise

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Radermacher P, Santak B, Muth CM, Wenzel J, Hampe P, Vogt L, Hahn M, Falke KJ. Nitrogen partial pressures in man after decompression from simulated scuba dives at rest and during exercise. *Undersea Biomed Res* 17(6):495-501.—In 5 subjects arterial and central venous nitrogen partial pressures (PN_2) were measured after decompression from a chamber dive following a decompression schedule for scuba diving. The simulated dives consisted of exposure to air at 6 bar for 30 min corresponding to a depth of 50 m. Afterward the subjects were decompressed with decompression stops at 2.5, 2.2, 1.9, 1.6, and 1.3 bar with a total decompression time of 67 min. In 3 of the subjects the measurements were repeated after they had exercised (workload 75 W) during bottom time. Immediately after decompression and every 40 min until Minute 240 arterial and central venous blood samples were analyzed for PN_2 using a manometric Van Slyke apparatus. Venous PN_2 remained elevated until 160 min after decompression, indicating still incomplete nitrogen washout for at least 2 h after decompression had been accomplished. We did not find any difference in PN_2 values after decompression from dives at rest and after exercise. Applying a computer program based on a wide range of theoretical tissue half-times nitrogen elimination proved to be consistent with Haldanian theories when using our decompression profile. Our data confirm that nitrogen elimination is prolonged after decompression from simulated dives at rest and after exercise.

blood nitrogen partial pressure
decompression

exercise
simulated diving

Divers breathing compressed air absorb nitrogen while at underwater pressure and eliminate it during and after decompression. The rates of uptake and elimination of nitrogen vary, depending on several factors such as exercise (1) or the shape of the compression and decompression profile (2). Too rapid a decompression may result in decompression sickness (DCS) due to intra- and extravascular bubble formation (3).

To avoid this problem, several decompression schedules have been established (4) based on decompression trials with volunteers. But despite thorough testing, even precisely following such a schedule does not always prevent symptoms of DCS or bubble formation in symptom-free dives (5).

Since decompression precedes the elimination of N_2 in the diving situation, measuring the mixed or central venous nitrogen partial pressures (P_{vN_2}) is one possibility to monitor decompression because the P_{vN_2} represents an average of the tissue P_{N_2} at a given time (2).

Although P_{N_2} have been measured in dogs after saturation dives, to our knowledge no data are available on P_{N_2} values in humans. The aim of our study was to describe the time course of arterial and central venous P_{N_2} after decompression from a simulated "bounce" dive following a common decompression schedule.

METHOD

Five males, all nonsmokers and all with normal pulmonary functions (Table 1), were investigated in this study. All had previous scuba diving experience, subjects KF, BS, and PR being occasional and subjects CM and AR being regular scuba divers. Before the investigation all subjects underwent medical examinations according to the standards of the German compressed air work regulations (G31-Vorsorgeuntersuchung Überdruck).

The simulated dives were performed in a dry hyperbaric chamber (TITAN, Deutsche Forschungsanstalt für Luft- und Raumfahrt, DLR, Cologne, FRG). The dives consisted of exposure to air at 6 bar corresponding to a depth of 50 m (Fig. 1). The chamber was pressurized within 2.5 min, the pressure being doubled every minute. After 30 min of bottom time at 6 bar the chamber was decompressed at a rate of $-1 \text{ bar} \cdot \text{min}^{-1}$ until a pressure of 2.5 bar corresponding to a depth of 15 m was reached (Fig. 1). In earlier chamber dives the same profile, but with a 12-min wet excursion to 6.8 bar corresponding to 58 m, resulted in 4 cases of skin rashes per 15 man-dives (6). For the present study we left out the excursion to 6.8 bar and kept the decompression profile, which consisted of decompression holds of 3, 4, 8, 18, and 34 min at 2.5, 2.2, 1.9, 1.6, and 1.3 bar, respectively (corresponding to 15, 12, 9, 6, 3 m) (Fig. 1). Decompression rate between two subsequent decompression stops was $-0.6 \text{ bar} \cdot \text{min}^{-1}$ each.

TABLE 1
MORPHOMETRIC DATA OF THE SUBJECTS

Subject	Age, years	Height, cm	Weight, kg	Vital capacity, liter
PR	30	171	65	5.1
BS	31	171	70	5.0
CM	27	171	71	5.5
AR	35	185	72	6.6
KF	48	180	74	5.4

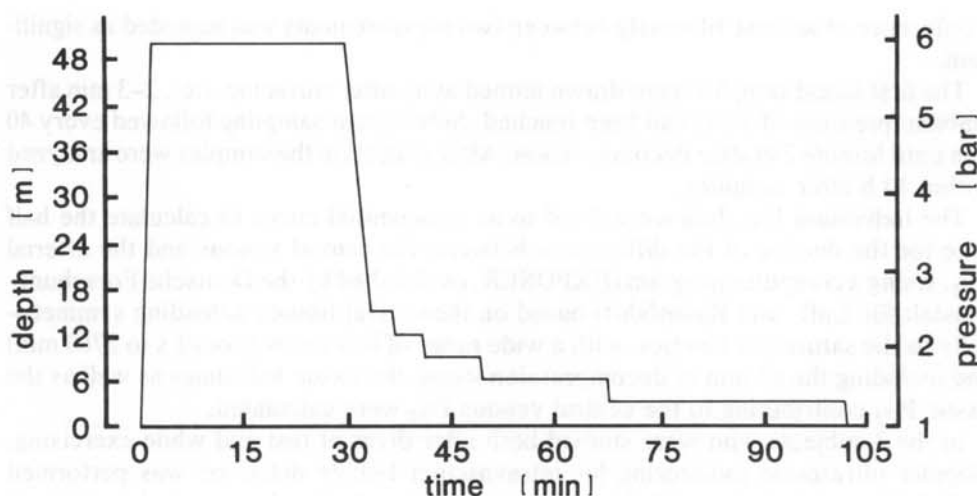


Fig. 1. Profile of the simulated dive. The y-axes show the ambient pressure in bar and the corresponding depth in meters.

Dives at rest were investigated in all 5 subjects, subjects CM, BS, and PR were studied both after resting and exercising dives. The subjects exercised on a bicycle ergometer, the workload being 75 W. This exercise rate approximates those of common sport dives at this depth (1). The exercise periods were limited to 20 min of bottom time to allow heart rate to return to normal before decompression. The entire decompression took place with the subject resting in a comfortable, supine position.

For blood sampling in all subjects radial artery and central venous catheters had been placed. The latter were positioned after puncture of a large forearm vein, and the position of the tip of the catheter in the superior vena cava was controlled by chest x-ray.

Nitrogen partial pressures were measured with a manometric Van Slyke apparatus (7) according to a manometric technique described by Klocke and Rahn (8). Simultaneous 15-ml aliquots of arterial and central venous blood were anaerobically taken in heparinized gas-tight syringes (Hamilton, Bonaduz, Switzerland). After vacuum extraction and chemical absorption of O₂ and CO₂ the total amount of dissolved nonabsorbable gas was determined. This gas was referred to as nitrogen (N₂). In each subject additional samples of arterial and venous blood were equilibrated with air at 37°C and the amount of dissolved gas was measured. The PN₂ values (expressed in mmHg) were derived from the quotient of the N₂ content in the directly measured blood sample divided by the N₂ content of the sample equilibrated with room air. It is unnecessary to determine the Ostwald coefficient when comparing directly measured and equilibrated blood samples, and thus inter- and intraindividual (between arterial and central venous blood) N₂ solubility differences assume no importance.

For a previous study (9) the reproducibility of the PN₂ in blood samples equilibrated with room air had been shown to be 566.4 ± 5.1 mmHg ($n = 12$), i.e., 0.9%. Therefore

a difference of at least 10 mmHg between two measurements was regarded as significant.

The first blood samples were drawn immediately after surfacing, i.e., 2–3 min after ambient pressure of 1 bar had been reached. Subsequent sampling followed every 40 min until Minute 240 after decompression. After collection the samples were analyzed within 10 h after sampling.

The individual P_{N_2} data were fitted to an exponential curve to calculate the half time for the decline of the differences between the central venous and the arterial P_{N_2} . Using a computer program (EXPOSER, established by the Deutsche Forschungsanstalt für Luft- und Raumfahrt) based on theoretical tissues unloading symmetrically to the saturation kinetics, with a wide range of half times (from 1 s to 2760 min) and including the 67 min of decompression stops, the tissue half-times as well as the tissue P_{N_2} contributing to the central venous P_{N_2} were calculated.

In the 3 subjects who were studied both after dives at rest and while exercising, Doppler ultrasound monitoring for intravascular bubble detection was performed (subclavian vein) during each decompression stop and after decompression using an ultrasound device constructed by SODELEC (Marseille, France). Minimal size for bubbles detectable with this instrument is 40–50 μ m.

Statistical analysis was performed using a two-tailed analysis of variance to detect significant differences between decompression and baseline as well as between arterial and central venous P_{N_2} values.

RESULTS

The mean baseline arterial and central venous P_{N_2} were 568 ± 13 and 569 ± 13 mmHg. Mean arterial P_{N_2} was 592 ± 33 mmHg immediately after decompression and had returned to baseline at the second measurement, i.e., 40 min after decompression. In contrast, venous P_{N_2} remained elevated until the fourth measurement after decompression, falling from 720 ± 36 to 561 ± 21 mmHg at 160 min. The half-time for the decline of the differences between the central venous and arterial P_{N_2} values was 27 min when fitted to the exponential curve.

The computer analysis yielded a theoretical tissue half-time of 50 min with a tissue P_{N_2} of 1150 mmHg contributing most to the central venous P_{N_2} immediately after decompression. The corresponding tissue half-time was about 150 min with a tissue P_{N_2} of 750 mmHg 120 min after decompression.

No differences were found between either arterial or central venous P_{N_2} values after dives at rest and while exercising. Figure 2 shows the time course of arterial and central venous P_{N_2} after resting and exercising dives for the subjects taken together.

None of the subjects exhibited any symptoms of DCS during or after decompression, and no intravascular bubbles could be detected either.

DISCUSSION

The aim of this study was to describe the time course of P_{N_2} after a simulated scuba dive following a routine decompression schedule in humans.

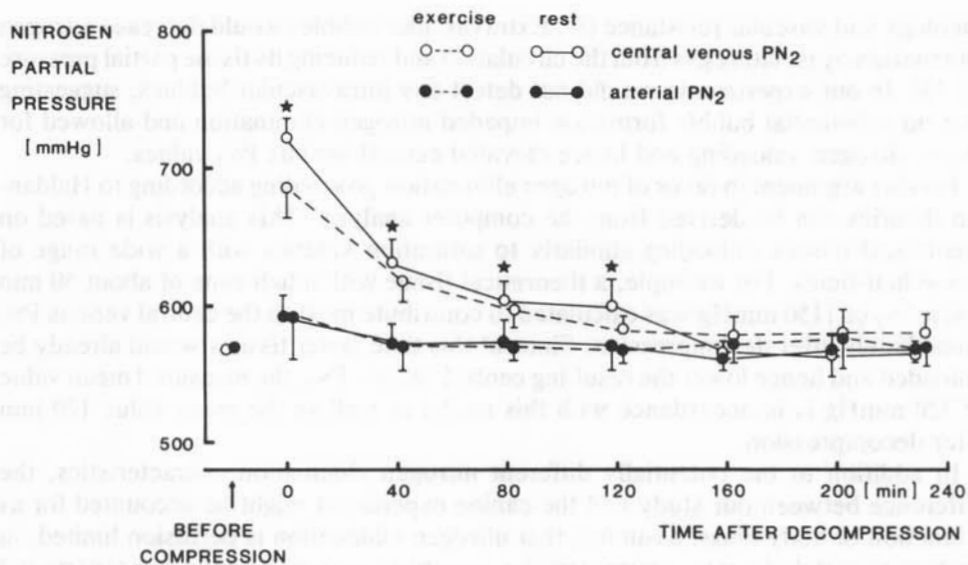


Fig. 2. Time course of arterial (solid circles) and central venous (open circles) PN₂ in mmHg after resting (solid lines) and exercising (dotted lines) dives. Data are presented as mean \pm SEM. Time "0 minutes" refers to surfacing time. Asterisks denote significant differences between the arterial and the central venous PN₂ values. Note that the arterial PN₂ at surfacing time was significantly higher than the baseline value and that there was no difference between rest and exercise dives. When fitted to an exponential curve the equation for the time function of the difference $P_{vN_2} - P_{aN_2}$ was $(P_{vN_2} - P_{aN_2})(t) = 116 \text{ mmHg} \cdot e^{-t/38.4 \text{ min}}$.

The average baseline PN₂ in the 5 subjects was 568 mmHg, which agrees well with those published elsewhere for normal subjects (10). The mean difference between the arterial and venous PN₂ was 1 mmHg, i.e., 0.18%. This negligible difference is in accordance with theoretical assumptions emphasizing that arterial and venous PN₂ should be equal inasmuch as there is neither production nor consumption of N₂ by systemic tissues or organs (11).

Venous PN₂ markedly exceeded the arterial and alveolar PN₂ until 160 min after decompression. These data indicate that nitrogen elimination was still incomplete at least 120 min after decompression.

The long-lasting elevation of PN₂ values in our subjects differs from the results of a previous experiment using incremental decompression in dogs (2). In these experiments venous PN₂ reached baseline values within 40 min after decompression. It has to be noted however, that in these experiments during the decompression stops the venous PN₂ rose from values below the expected levels to values markedly above the arterial ones. This phenomenon was explained by "a recovery of the tissue to blood nitrogen clearance" which initially had been reduced, possibly due to bubble formation (2, 12). Intravascular bubbles might decrease the elimination rate by causing capillary stasis and reducing tissue nitrogen clearance (2) or by changing blood

rheology and vascular resistance (3). Extravascular bubbles would decrease nitrogen elimination by isolating gas from the circulation and reducing its tissue partial pressure (4, 13). In our experiments we did not detect any intravascular bubbles, suggesting that no substantial bubble formation impeded nitrogen elimination and allowed for tissue nitrogen unloading and hence elevated central venous PN_2 values.

Further argument in favor of nitrogen elimination proceeding according to Haldanian theories can be derived from the computer analysis. This analysis is based on theoretical tissues unloading similarly to saturation kinetics with a wide range of tissue half-times. For example, a theoretical tissue with a half-time of about 50 min and a PN_2 of 1150 mmHg was calculated to contribute most to the central venous PN_2 immediately after decompression. Since at this time faster tissues would already be unloaded and hence lower the resulting central venous PN_2 , the measured mean value of 720 mmHg is in accordance with this model as well as the mean value 120 min after decompression.

In addition to the potentially different nitrogen elimination characteristics, the difference between our study and the canine experiment might be accounted for as a function of body mass: assuming that nitrogen elimination is perfusion limited, in analogy to metabolic rate, oxygen uptake, or cardiac output, it would be proportional to body mass raised to the 0.75 power (14). Hence, the nitrogen elimination could be calculated as $V_{N_2} = a \cdot BW^{0.75}$, which would yield 24.2 arterial and 7.6 arterial for V_{N_2} in a 70-kg man and a 15-kg dog, respectively. The ratio 24.2:7.6 or 3.2 approximates the ratio of the two elimination times 160 and 40 min, respectively, i.e., 160:40 or 4.0 assuming that the blood flow distribution is similar during simulated dives in dogs and humans.

In the studies following exercise dives, the PN_2 time course did not differ from the one after dives at rest. It is well established theoretically and experimentally by decompression trials for DCS that exercise during diving increases nitrogen uptake (4). A difference for the PN_2 time course after resting and exercising dives, however, was unlikely to be detected when following our decompression schedule. Muscle perfusion at rest is about $3 \text{ ml} \cdot \text{min}^{-1} \cdot 100 \text{ g}^{-1}$ and $60 \text{ ml} \cdot \text{min}^{-1} \cdot 100 \text{ g}^{-1}$ at maximal exercise (15). These values correspond to tissue half-times of about 20 and 1 min, respectively, if nitrogen uptake is perfusion-dependent. With a half-time of 20 min, resting muscle would be 70% saturated after 30 min (14) of bottom time, as in our experiment. In contrast to that, exercising muscle at a half-maximal perfusion corresponding to a half-time of about 3 min would be 100% saturated after 30 min of bottom time (16). Thus half-maximal exercise could increase nitrogen uptake by only 30%. Taking into consideration that our measurements after decompression could only depict the slower part of the PN_2 time course, these differences were unlikely to be detected.

Recently, Dick and coworkers (1) suggested that different levels of physical fitness might influence nitrogen elimination: slower elimination was observed during repeated investigations after 1 subject had reduced his physical activity within several months. We did not find any interindividual differences in the PN_2 curves in our subjects, although 2 of them were regular divers with superior training levels, suggesting potentially different blood flow distributions (1). It has to be noted, however, that the more pronounced anthropometric variation in our subjects (Table 1) compared to the above-cited study may have made different nitrogen curves due to interindividual fitness differences nondetectable by our methods.

In conclusion, our study clearly confirms that after simulated dives precisely following a routine decompression schedule, complete nitrogen elimination requires several hours after decompression. Following this decompression schedule, a light-to-moderate workload does not further delay normalization of blood nitrogen content. Further investigation is warranted on the time course of PN₂ values after resting and exercising dives within the no-stop limits in "high-bubbler" subjects (5).

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