Two questions arise. The first is: Was the opinion based on solid information from previous cases and accepted statistics—or was it extrapolated from thinking on other neurologic pathologies, itself perhaps somewhat theoretical? At the symposium in Bethesda, an audience of perhaps 100 diving doctors was asked if any had seen divers treated for CNS hits who returned to diving and had a second hit in the same location. Only two replied in the affirmative and one of these (to my neighbhor and me, at least) sounded more like recurrent sinus squeeze affecting the infraorbital nerve.

The second is: Assuming the opinion is not challenged, can the employer satisfy the duty of reasonable accommodation? If he can show there is no adequate accommodation that will meet his safety standard, he can legitimately refuse to employ the diver. Would more conservative decompression for this diver be "reasonable?" Is it known this would adequately protect the diver?

These questions may start coming up soon. At the seminar in Bethesda, an attorney who represents the diving industry suggested some divers might sue under ADA to regain their jobs. Whether they do or not, diving doctors will inevitably be pulled in and forced to consider what they really know. I believe all of us who deal with professional diving should peruse our medical standards. How many of them will satisfy ADA? Interesting question.

For those interested, the May 1992 issue of the *Journal of Occupational Medicine* contains two good articles and an editorial on ADA and the examining physician. In addition, the EEOC publishes a Technical Assistance Manual on the provisions of ADA, publication EEOC-M-1A.

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Saturation decompression schedules based on a critical tissue supersaturation criterion

To the Editor:

Modified Haldane concepts (1), commonly used for a calculation of decompression schedules, propose that decompression sickness (DCS) in divers occurs when a critical pressure ratio is exceeded. Recent works have connected DCS with gas bubbles that are formed from spherical gas nuclei stabilized by skin of surface-active substances (2, 3), and a number of mathematical models of bubble growth have been presented (4, 5). Since the body eliminates dissolved gas more quickly than undissolved (4), decompression will be more optimal when tissue bubble radii are minimal. This strategy was used in a model by Voytsekhovich (6, 7), based on the principle of limiting tissue supersaturation to values that do not permit bubble growth due to gas diffusion. In this report, we have adapted this model for saturation decompression after dives of 20–350 m (1 m of fresh water = 9.8067 kPa).

Decompression model

A gas nucleus will grow into a bubble when tissue gas supersaturation (ΔP_t) is more than the overpressure produced by a nucleus surface skin according to Laplace's law. Therefore, this overpressure for nuclei with maximal radii will be a critical tissue in supersaturation (ΔP_{cr}). Voytsekhovich (6, 7) (appendix) proposed that to prevent gas bubble growth from nuclei, tissue supersaturation should not exceed this critical tissue supersaturation during decompression:

$$\Delta P_t \leq \Delta P_{cr} \qquad (1)$$

A nucleus radius will be decreased during compression below sea level and increased during decompression, with a gas expansion according to Boyle's law. Using Laplace's and Boyle's laws and *Eq. 1*, an equation for maximal permissible tissue supersaturation was obtained:

$$\alpha_c \cdot \Delta P_t^3 - \Delta P_t = P_\alpha \qquad (2)$$

where P_α is an ambient pressure and α_c is a coefficient of tissue cavitation strength, defined as

$$\alpha_c = \frac{P_o + \Delta P_{cr_o}}{\Delta P_{cr_o}} \qquad (3)$$

where P_o = 1 atm abs, and ΔP_{cr_o} is the experimentally established critical tissue supersaturation at P_o. Coefficient α_c defines the decrease of ΔP_{cr} during decompression from a value of ΔP_{cr_o}.

The value of ΔP_{cr_o} is significantly greater for heliox than nitrox ($\Delta P_{cr_oN_2}$ and ΔP_{cr_oHe}, respectively), probably because of differences in fat/water solubility coefficients. These values may be approximately estimated from body responses to direct decompression to 1 atm abs after saturation at greater pressures. Eckenhoff et al. (8) found that decompression in 2 min to 1 atm abs after a 48-h exposure at 1.77 atm abs air (1.4 atm abs nitrogen) did not evoke definite DCS, but in 26% of subjects, symptoms occurred such as fatigue and limb and joint discomfort. The nitrogen supersaturation that was responsible for the appearance of these symptoms was 0.4 atm abs, and it is obvious that whole tissue supersaturation was more than ΔP_{cr}. Therefore, $\Delta P_{cr_oN_2}$ must be less than 0.4 atm abs.

Barnard's data (9) indicate that rapid ascent to the surface after 24 h of saturation exposure in normoxic He-O_2 mix at 2 atm abs is safe for divers. This decompression implies helium tissue supersaturation of 0.8 atm abs. Nevertheless, bubbles cannot be excluded after such a pressure decrement. Therefore, ΔP_{cr_oHe} must be no more than 0.8 atm abs.

Compression causes a decrease of nucleus radius that results in an increase of safe pressure reduction in men (9, 10) that provides an advantage for decompression from a short-term dive. Voytsekhovich's model (6, 7) (appendix) includes this phenomenon when a value of 1 atm abs is used for P_o in *Eq. 3*. However, with estimated $\Delta P_{cr_oN_2}$ and ΔP_{cr_oHe}, the model produced high values of ΔP_t, which are inconsistent with saturation decompression data. We have adapted the model using one assumption.

Substances that formed gas nuclei skins in living tissues are predominantly lipids. The interaction of lipid molecules of a different polarity in a water system provides conditions for formation of micelle structures (11). Central hydrophobic areas of micelles are obviously the most preferable places for nucleation in tissues. Hence we propose that gas nuclei are generated from lipid micelles and that renovation of the gas nucleus population results from metabolic processes that generate and destroy lipids and lipid micelles. Size distribution of newly generated gas nuclei probably will be defined by the structure of lipid molecules.

The above speculation led us to propose that in saturation dives consisting of several days under a new pressure level, size distribution of the gas nucleus population in the tissues returns to the steady-state distribution at 1 atm abs. Consequently, tissues should have the same value of ΔP_{cr} as at 1 atm abs before compression; thus, the decompression advantage induced by compression disappears.

How much time is enough to reestablish normal values of ΔP_{cr} in tissues? It was found that caisson workers adapt to DCS by regular pressure exposures and then lose the adaptation about 10 days after regular exposures stop (12). Both phenomena can probably be related to the compression of gas nuclei and the recovery of a normal size distribution of the gas nucleus population in the tissues, respectively. Ten days may be the time needed for restoring the steady state for tissue ΔP_{cr}.

According to our assumptions, the term P_o in *Eq. 3* will mean the pressure of saturation exposure at which the steady state for tissue ΔP_{cr} was reached.

Moreover, since exposure times in studies by Eckenhoff et al. (8) and Barnard (9) (1 and 2 days, respectively) probably were not sufficient for ΔP_{cr} to reach a steady state, we tentatively assumed conservative values of $\Delta P_{cr_oN_2} = 0.28$ atm abs and $\Delta P_{cr_oHe} = 0.6$ atm abs. For trimix, a mean-weighted value of ΔP_{cr_o} was calculated in proportion to N_2 and He partial tensions in the tissue during decompression:

$$\Delta P_{cr_o} = \Delta P_{cr_oN_2} \cdot \frac{P_{tN_2}}{P_{tN_2} + P_{tHe}} + P_{cr_oHe} \cdot \frac{P_{tHe}}{P_{tN_2} + P_{tHe}} + C \qquad (4)$$

where C is the sum of partial tensions of O_2, CO_2, and H_2O vapor in tissues, about 0.18 atm abs.

To compute the saturation decompression schedule, α_c was calculated (*Eq. 3*) for a special saturation pressure P_o and ΔP_{cr_o} (*Eq. 4*). Then it was used in *Eq. 2* for the maximal permissible tissue supersaturation at decompression stops. Maximal permissible tension of inert gases was defined as $\Delta P_t + P_\alpha - C$. For estimation of tissue gas transport, a perfusion model was used with half times of 240 min for helium and 640 min for nitrogen.

Figure 1 shows tissue supersaturation calculated by *Eq. 2* as a function of ambient pressure. During decompression, ΔP_t does not exceed ΔP_{cr} and is decreased from the value of ΔP_{cr_o}, which does not depend on the level of saturation pressure and varies between 0.47 and 0.59 atm abs with the gas mix (*Eq. 4*) in our dives.

Methods

Subjects were 15 healthy male volunteers, all qualified as divers. Mean age was 34 ± 1 yr (range 27–44 yr), mean weight 78 ± 2 kg (range 66–106 kg), and mean height 177 ± 1 cm (range 177–186 cm). All had experience in short hyperbaric

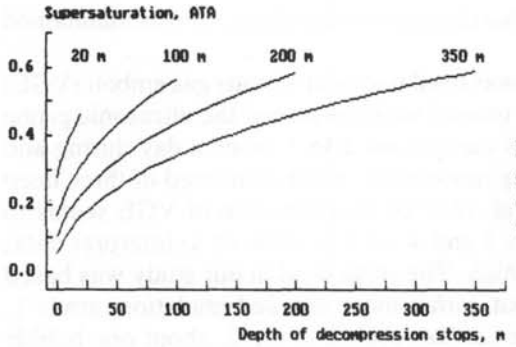

Fig. 1. Inert gas supersaturation (ΔP_t) calculated by *Eq. 2* as a function of ambient pressure at decompression steps. Supersaturation curves for different dive depths (20–350 m) vary depending on the value of P_O in *Eq. 3* (3–36 atm abs, respectively).

exposures and some took part in 2 or 3 experimental saturation dives during this study.

Simulated dives were performed in the hyperbaric complex of the South Department of P.P. Shirshov Institute of Oceanology (Gelendgic) in a 20-m^3 saturation chamber that had an external life-support system and was connected with two smaller chambers.

Details of 9 simulated saturation dives are presented in Table 1 in chronological sequence. In 7 dives (from DP-20/1 to DP-200) each diver performed 10 excursion dives under dry conditions to test excursion decompression schedules (13). These excursions were performed 20–110 m deeper than resident depth levels and took 2–4 h. There were no DCS signs during or after excursions. Saturation decompression was begun approximately 1 day after the last excursion. In dive DP-350/2, 2 subjects performed a 7-day excursion from 350 to 450 m, and saturation decompression was begun 3 days later. Only in saturation dive DP-350/1 were there no excursions.

The gas environment during saturation exposure consisted of 0.23–0.25 atm abs (1 atm abs = 98.067 kPa) of oxygen, 1.2 or 1.4 atm abs of nitrogen and helium for the rest. Temperature was maintained so that the subjects remained comfortable, and humidity was between 60 and 70%. Decompression was carried out with steps of 2 m. Partial pressure of oxygen (P_{O_2}) during ascent was maintained at a level of

TABLE 1
SIMULATED EXPERIMENTAL DIVES

Name of Dive	Depth of Dive, m	Time of Saturation, days	Time of Decompression, h	P_{O_2}, atm abs	Number of Subjects
DP-350/1	350	21	330:00	0.3	2.
DP-20/1	20	14	22:01	0.3	4
DP-20/2	20	14	22:01	0.3	3
DP-50/1	50	14	51:36	0.3	3
DP-50/2	50	14	51:24	0.3	3
DP-100	100	14	78:00	0.5	4
DP-150	150	14	115:10[a]	0.5	4
DP-200	200	14	159:10	0.5	4
DP-350/2	350	24	276:44	0.5	4

[a]Decompression was extended to 18 h because of DCS treatment.

0.3 ± 0.1 or 0.5 ± 0.1 atm abs. At the final decompression steps, O_2 was maintained at 25%.

A 5.7-MHz Doppler ultrasonic device was used to detect venous gas emboli (VGE) in the pulmonary artery. The previously trained subjects placed the ultrasonic probe in the precardial region. Monitoring was carried out 2 to 3 times a day during and after decompression, at rest, and after leg movement, which consisted of three deep knee bends. The method of Spencer et al. (14) for an estimation of VGE seems to have some discontinuity between grades 3 and 4, so it is difficult to interpret data, especially when the level of VGE is not high. The scale used in our study was based on the principle of Spencer et al. (14) but with a more detailed gradation: grade 1, less than about one bubble signal in five cardiac cycles; grade 2, about one bubble signal in two to five cardiac cycles; grade 3, about one to five bubble signals in each cardiac cycle; grade 4, more than five bubble signals, which are separated and clearly distinguished from each other in each cardiac cycle. Up to grade 4, this scale approximately corresponds to a log graph. Grade 5 designates bubble signals that have overridden each other within the cardiac and blood flow signals. The maximal bubble score from 10 cardiac cycles was recorded.

Descriptive and comparative statistics, correlation, and linear regression analyses were used. A P value of 0.05 was considered significant.

Results

Only one case of DCS occurred in 31 man-decompressions during ascent from 150 m (DP-150); a 26-yr-old subject felt discomfort in the right knee joint at 88 m which turned into pain at 78 m. He had not participated in saturation dives before. The pain disappeared after recompression treatment to 98 m while breathing 0.6 atm abs of oxygen. Decompression was continued after 2 h at treatment depth using the initial schedule. That subject again felt discomfort in the same site at 50 and 20 m, and at 2 m he complained of a mild pain in the knee joint. He had complete resolution of pain during recompression to 18 m while breathing 100% oxygen. Thus, incidence of DCS in our trials was 3.3 ± 3.2% (mean + SE).

Another manifestation of decompression disorders was a transient pruritis in 3 subjects immediately after ascent to the first decompression stop in DP-20/1. Two subjects in DP-50/2 and one in DP-200 had joint discomfort from 6 to 8 m up to the surface, which was felt during or immediately after ascents to the next decompression stop and lasted for 10–20 min.

Venous gas emboli in the pulmonary artery were observed in all decompressions, but not in all subjects: they were found only in 24 man-decompressions from 31 m (77.4 ± 7.5%). At rest, VGE were found in 14 man-decompressions (45.2 ± 8.9%); in most cases they were grade 1 and never more than grade 2. After movement, bubble scores varied from grades 1 to 4, and maximal bubble scores in most cases were grade 4. Figure 2 shows VGE dynamics during decompression in dive DP-350/2 in 3 subjects; in the fourth subject, VGE were absent.

Figure 3 presents VGE dynamics in a subject who had DCS during decompression from 150 m. Just before recompression treatment, bubble score was grade 2 at rest and grade 4 after knee bends. VGE disappeared in a few minutes at the treatment depth and returned during subsequent decompression at 50 m.

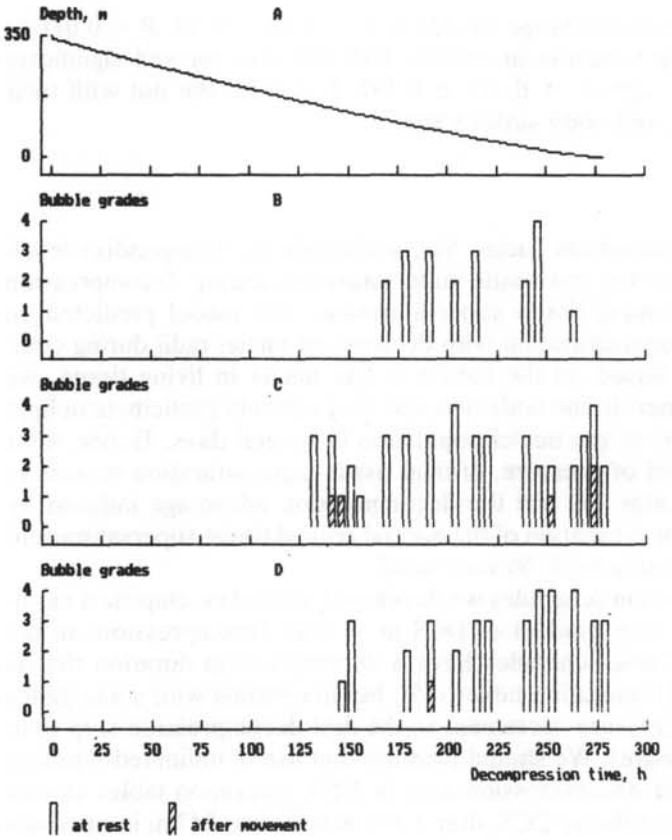

Fig. 2 Venous gas emboli in the pulmonary artery in 3 subjects (*B, C, D*) during decompression in simulated experimental dive DP-350/2. *A*, decompression profile. *Solid bars* = bubble score at rest; *open bars* = after knee bends.

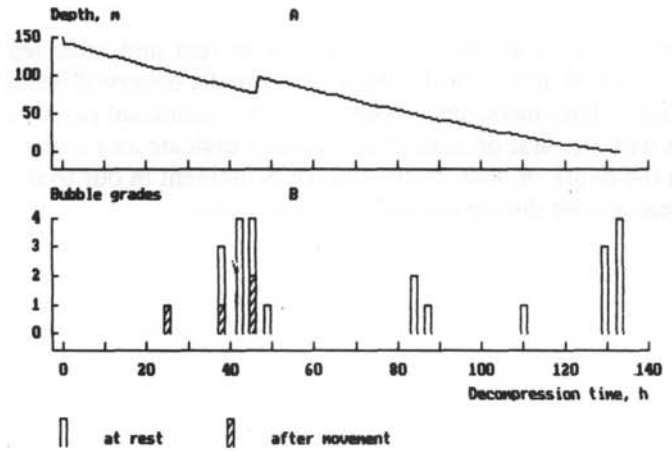

Fig. 3. Venous gas emboli in the pulmonary artery during decompression (*B*), who suffered decompression sickness in simulated experimental dive DP-150. *A*, decompression profile. *Solid bars* = bubble score at rest; *open bars* = after knee bends.

To compare the degree of decompression load, we used the ratio of the depth of a first VGE appearance to the depth of dive: relative depth of first bubbles detected (RDFDB). In most cases, these bubbles were after movement. For all dives, mean RDFDB was 47 ± 5% (range 12–92%, $n = 25$). This index showed positive correlation

with the depth of saturation in the range 20–200 m ($r = 0.63 \pm 0.18$, $P < 0.01$), at 350 m we observed a large variation in results. RDFDB also showed significant correlation with the divers' ages ($r = 0.471 \pm 0.197$, $P < 0.05$) but not with their weight, height, fat content, and body surface area.

Discussion

Using the concept of stabilized gas nuclei, Voytsekhovich (6, 7) (appendix) developed a model that provides that gas tissue supersaturation during decompression should be no more than critical tissue supersaturation. The model predicted an increase of critical tissue supersaturation with decrease of nuclei radii during compression from 1 atm abs. Based on the nature of gas nuclei in living tissue, we assumed that they are not inert in the body and that they actively participate in lipid metabolism, with renovation of gas nuclei population in several days. Hence, after a certain time at a new level of pressure, critical tissue supersaturation returns to its steady-state value at 1 atm abs and the decompression advantage induced by compression disappears. For estimation of the normal critical tissue supersaturation, experimental data of other authors (8, 9) were used.

The saturation decompression schedules we developed needed no empirical modification and exhibited only one incident of DCS in 31 man-decompressions in the range from 20 to 350 m. These schedules have a decompression duration that is similar to U.S. Navy (USN) saturation tables (15), but in contrast with some tables they have a much smaller pressure decrement to the first decompression stop (8 m after 350 m saturation exposure). We should mention that use of unlimited-duration excursion tables for the first decompression stop in USN saturation tables cannot be safe. We observed (13) vestibular DCS after a 119-min dive to 247 m in open sea followed by a 94-min decompression, although our profile was more conservative than direct ascent to 201 m from saturation at 248 m shown in the USN Diving Manual (15).

The VGE incidence in our study was about 45 and 77% at rest and after leg movement, respectively, and the first detectable bubbles were usually observed when ambient pressure was reduced by half during decompression. The significant positive correlation of relative depth with the first detected bubbles may indicate an increase of decompression load with the depth of dive. In the one DCS incident in our trials, the bubble score was no greater than during normal decompression.

APPENDIX

Model of I.A. Voytsekhovich (1981) for a permissible gas tissue tension during decompression

Pressure (P) in a spherical gas nucleus according to Laplace's law is:

$$P = \frac{2\sigma}{R} + P_a \tag{1}$$

where R = a nucleus radius, σ = surface tension, and P_a = ambient pressure. The nucleus will grow when tissue supersaturation ($\Delta P_t = P_a - P$, where P_a = gas tissue tension) is more then $2\sigma/R$. So $2\sigma/R$ is a critical tissue supersaturation (ΔP_{cr}).

Tissue supersaturation during decompression should be no more than critical tissue supersaturation:

$$\Delta P_t \leq \Delta P_{cr} \tag{2}$$

or

$$P_t \leq \frac{2\sigma}{R} + P_a \tag{2a}$$

Pressure increases from a value of P_0 = 1 atm abs to P_1 when the step is done for saturation. Now $P_t \approx P_1$ In accordance with *Eq. 2a*, pressure for the first decompression stop (P_2) was chosen to be:

$$P_1 = \frac{2\sigma}{R_2} + P_2 \tag{3}$$

and

$$R_2 = \frac{2\sigma}{P_1 - P_2} \tag{4}$$

Pressures inside gas nuclei and their radii at pressures P_0 and P_2 will be related by the next equation:

$$\left(\frac{2\sigma}{R_0} + P_0\right) \cdot R_0^3 = \left(\frac{2\sigma}{R_2} + P_2\right) \cdot R_2^3 \tag{5}$$

After substitution of *Eqs. 3* and *4* in *Eq. 5:*

$$\left(\frac{2\sigma}{R_0} + P_0\right) \cdot R_0^3 = P_1 \cdot \left(\frac{2\sigma}{P_1 - P_2}\right)^3 \tag{6}$$

or

$$P_1 = \left(\frac{2\sigma}{R_0} + P_0\right) \cdot \left(\frac{R_0}{2\sigma}\right)^3 \cdot \left(P_1 - P_2\right)^3 \tag{6a}$$

Now we define the product of the first two terms in *Eq. 6a* as the coefficient of tissue strength (α_c) at P_0 = 1 atm abs. Then

$$P_1 = \alpha_c \cdot (P_1 - P_2)^3 \tag{6b}$$

when

$$\alpha_c = \left(\frac{2\sigma}{R_0} + P_0\right) \cdot \left(\frac{R_0}{2\sigma}\right)^3 \tag{7}$$

Eq. 6b will be presented more conveniently using the definition $\Delta P_t = P_1 - P_2$:

$$\Delta P_t + P_2 = \alpha_c \cdot \Delta P_t^3 \tag{8}$$

For all subsequent decompression stops:

$$\Delta P_t + P_a = \alpha_c \cdot \Delta P_t^3 \tag{8a}$$

A value of $2\sigma/R_0$ presents critical supersaturation at 1 atm abs (ΔP_{cr_o}), so *Eq. 5* will be presented more conveniently too:

$$\alpha_c = \frac{P_0 + \Delta P_{cr_0}}{\Delta P_{cr_0}^3} \tag{9}$$

ΔP_{cr_0} can be found from safe pressure decrements to 1 atm abs. With known α_c, we can find ΔP_t at each decompression step using *Eq. 8a*.

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