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Direct ascent from shallow air saturation exposures

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Eckenhoff RG, Osborne SF, Parker JW, Bondi KR. Direct ascent from shallow air saturation exposures. Undersea Biomed Res 1986; 13(3):305–316.—Thirty-four healthy human subjects were exposed to shallow air saturation for 48 h [1.77 ATA (25.5 fsw) n = 19, 1.89 ATA (29.5 fsw) n = 15] and then decompressed to 1 ATA (0 fsw) in about 2 min. Symptoms included fatigue, limb and joint pain, headache, myalgias, and pruritus. No subject of 19 was diagnosed as having decompression sickness (DCS) after the shallower exposure, but 4 of 15 were diagnosed and treated for DCS subsequent to the deeper exposure. Almost all subjects in both groups had Doppler-detectable venous gas emboli (VGE) lasting up to 12 h postdecompression. Treated subjects had a recurrence of VGE several hours after the hyperbaric oxygen treatment. Only the duration of VGE, and not the VGE score, correlated with symptoms; and only the subjects body weight and age correlated with the VGE variables. This study indicates that hyperbaric air exposures of this magnitude are not as benign as previously thought.

decompression sickness
venous gas emboli
hyperbaric oxygen

The magnitude of direct pressure decrements that humans can safely tolerate without incurring the risk of decompression sickness is of immense practical and theoretical importance. Practical, because the alternative is lengthy decompression, and theoretical, because all modern decompression schedule formulations necessarily use this point as a beginning. Application of this information is found in diving, pressurized construction, submersible operations, space flight, and certain medical therapeutics. Despite the importance of such information, remarkably little solid data exist to suggest what degree of pressure a human can be exposed to for infinite periods and return immediately to 1 atmosphere absolute (ATA) without the risk of the various decompression sickness syndromes (DCS). Early estimates based largely on animal work suggested that this point (for air) is about 2 ATA (33 fsw) (1). More recent experience has shown that DCS can occur on direct ascent from lengthy stays at lesser pressures. For example, at least 1 of 4 subjects exposed to 1.94 ATA (31 fsw) for 24 h was diagnosed with DCS (2). Similarly, anecdotal reports from pressurized tunnel workers has suggested that DCS can occur after direct decompression from

prolonged exposures at less than 1.9 ATA (30 fsw). At least one small field study showed no DCS, however, in 2 subjects after direct decompression from 1.79 ATA (26 fsw) (3). In other studies, interpretation of the decompression results are muddied because of oxygen breathing or insufficient time at pressure for saturation (4, 5). A "safe" level may have been established recently as 63 human subjects have been asymptomatic after direct decompression from 24-h exposures to 1.56 ATA (18.5 fsw), and another 188 subjects after similar exposures to 1.52 ATA (17 fsw), in a field educational project (personal communication, N. Monney, C. Olstad; Marine Resources Development Foundation, Key Largo, FL). Unfortunately, none of these previous studies attempted to quantify blood or tissue gas phase separation (bubbles) with, for example, a Doppler ultrasound apparatus. This would have provided additional objective evidence as to the stressfulness of the decompression. This report describes both symptoms and Doppler results from no-stop decompression in 34 human subjects after prolonged exposure to air in between this "safe" pressure level and levels where DCS has been well documented.

METHODS AND MATERIALS

Subjects

The subjects for these exposures were Navy divers or qualified hyperbaric chamber personnel, none of whom had been exposed to pressure or diving for at least a month before these exposures. The subjects were divided into 2 groups for exposure at different pressure levels. No subject was used twice at the same pressure level; 4 subjects were exposed to both pressure levels (exposures separated by about 4 mo.). No significant differences in age or physical characteristics existed between the 2 groups (*see* Table 4).

Facility

All exposures were performed in a large double-lock, steel hyperbaric chamber located at the Naval Submarine Medical Research Laboratory in Groton, CT. It is cylindrical, about 3 m in diameter and about 9 m long, allowing uncramped habitability for 5 subjects at a time. Temperature was adjusted to subject comfort, while humidity generally remained at about 60%. Chamber pressure was monitored through calibrated Heise gauges.

Exposures

The experimental protocol was approved by the laboratory Committee for the Protection of Human Subjects, and informed consent was obtained from each subject before confinement in the chamber. All exposures were dry. Two different exposure conditions were employed, identical in all respects except for the pressure level. Group 1 consisted of 19 subjects (4 separate exposures) exposed to 1.77 ATA of air (1.4 ATA nitrogen). Group 2 consisted of 15 subjects (3 separate exposures) exposed to 1.89 ATA (1.5 ATA nitrogen). Compression was generally complete in less than 1 min. This pressure was maintained $\pm$ 0.015 ATA for the next 48 h. The oxygen level

was maintained at 21% (0.37 ATA for Group 1 and 0.40 ATA for Group 2). The subjects ate a regular diet, performed no exercise, and took no medications while residing in the chamber. Activity consisted of rest and unhurried movement about the chamber. After 48 h of exposure, decompression back to 1.00 ATA occurred in about 2 min.

Data collection

The subjects were interviewed at regular intervals throughout the exposure and postexposure periods. All elicited and reported symptomatology was recorded and evaluated by the diving medical officer, and he was responsible for establishing the diagnosis of DCS. Because of the protean nature of DCS, symptoms subsequent to the decompression were divided into three categories: no DCS; questionable DCS; and definite DCS. The questionable category would be similar to what has been termed "niggles" (transient symptoms consistent with DCS, but of such a nature that treatment is considered unjustified). Only subjects in the definite category were treated, and treatment followed guidelines in the U.S. Navy Diving Manual (6).

After decompression, subjects were monitored at regular intervals (30 min, 1, 2, 3, 4, 5, 6, 9, 12, and 24 h) with a precordial Doppler ultrasound probe (Sodelec) to detect the presence of venous gas emboli (VGE). Each monitoring session consisted of about 1 min with the subject standing at rest, and then 30 s after each of three deep knee bends. Neither the subject nor the medical officer responsible for making the diagnosis of DCS was aware of the VGE score. Signals were recorded and scored according to the Kisman et al. scheme (7). This scheme is similar to that of Spencer (8), in that a score of 0 represents no detectable VGE and 4, maximal (signal saturation). Recordings were coded, randomized, and scored twice by the same investigator (RGE) several weeks apart. From the mean of these scores, the VGE onset time (time of first session in which VGE were detected), the VGE peak score (both movement and rest, although movement scores were always higher), time of the peak score, and VGE duration (time from first to last session that VGE were detected) were determined.

Statistics

Comparison between the 2 groups was on the basis of Student's t test, with a P value of 0.05 being considered significant. Point-biserial correlation was used to compute the correlation coefficients in Table 5, and product-moment procedures were used for those in Table 6. The correlation procedures merely detect an association between variables, and do not imply cause and effect.

RESULTS

Symptoms. No specific, consistent symptoms occurred during the 48-h exposure to either 1.4 or 1.5 ATA nitrogen. All of the symptoms occurring after the decompression are summarized in Table 1. The predominate symptoms appear to be fatigue and limb and joint discomfort in the first 24 h, and malaise, myalgias, and headache after 24 h. In some subjects, the delayed symptoms resembled a viral syndrome without a

TABLE 1
SYMPTOMS (n = 34)

Acute Onset (<24 h)	Incidence, %
Fatigue	53
Limb Symptoms	35
Pruritus	9
Headache	6
Delayed Onset (>24 h)	
Malaise	26
Myalgias	15
Headache	15
Limb Symptoms	12
Sleep Changes	6

TABLE 2
ACUTE LIMB AND JOINT SYMPTOMS

P_{N_2}	n	Possible DCS, Not Treated	Definite DCS, Treated
1.4	19	5 (26%)	0
1.5	15	3 (20%)	4 (27%)

respiratory or febrile component, and continued for up to 4 d. The overall incidence of symptoms was similar between the 2 groups, although qualitative differences did exist.

Since limb and joint discomfort after a decompression may constitute DCS, Table 2 shows those subjects so diagnosed, along with those considered to be in the questionable category. There were no cases of definite DCS in Group 1, and 4 in Group 2. The occurrence of questionable DCS was approximately the same between the 2 groups. All 4 subjects with definite DCS (type I—pain only) in Group 2 were treated with a USN Standard Table 5, as all had complete resolution of discomfort with 10 min at the treatment depth [2.8 ATA (60 fsw)] while breathing 100% oxygen. No recurrences or retreatments occurred.

Table 3 shows the limb symptoms categorized by site. These symptoms occurred far more commonly in the lower extremities, specifically the knee and periarticular region. The cases of definite DCS were pain in the knee for 3 subjects and ankle pain in the 4th. Table 6 also shows the mean onset time of symptoms, as reported by the subjects. Group 2 developed symptoms in significantly less time than Group 1 (2 ± 1 vs. 6 ± 2 h, $P < 0.05$).

Doppler. VGE were detected in 90% of the Group 1 subjects and in all of the Group 2 subjects. Wide individual variability in the VGE scores was noted in both groups. The data are summarized in Table 4*A* and *B* and shown graphically in Fig. 1.

TABLE 3
ONSET AND SITE OF ALL LIMB AND JOINT SYMPTOMS

P_{N_2}	1.4 (n = 19)	1.5 (n = 15)
Onset, Hours*:	6 ± 2	2 ± 1
Site:		
Knee	4	5(3)
Leg or Ankle	1	2(1)
Shoulder	2	0

Cases that were treated are shown in parentheses. *Subject's best estimate of onset time ± SD.

Although no significant difference in the peak VGE score (either at rest or after movement) between Groups 1 and 2 could be detected, the VGE onset time, the time to reach the peak score, and the overall VGE duration were significantly different between the 2 groups. The treated subjects had rapid and complete disappearance of VGE during treatment. After surfacing from the treatment, VGE remained undetectable. However, in an average of 4 h later, low-grade VGE (grade 1) were detectable in 3 of the 4 treated subjects. The 1 treated subject without recurrent VGE also had the lowest VGE score before treatment.

DISCUSSION

Despite the paucity of DCS cases reported with similar exposures, and despite widely disseminated guidelines to the contrary (6), it is apparent from our results that these exposures are not benign. Definite DCS occurred in several subjects after direct decompression from 1.85 ATA (29.5 fsw) and symptoms suggestive of DCS occurred in several subjects after the 1.77 ATA (25.5 fsw) exposures. Furthermore, almost all subjects had detectable VGE after even the shallow exposure. The pressure "threshold" from which decompression would produce neither symptoms nor VGE in humans is not known, but these results suggest that it is less than 1.4 ATA nitrogen (25.5 fsw), or much shallower than previously thought. Although the idea of such a threshold is attractive for practical reasons, it is probably not representative of what is actually occurring. Our data support the notion that there is significant individual variability in DCS susceptibility, and therefore the dose-response relationship probably has a sufficiently gradual slope to make the concept of a threshold untenable. Sufficient data to define the shape of this relationship do not exist at present. Defining the dose-response relationship (P_{N_2} – DCS) for humans will require many more exposures at sufficiently provocative nitrogen doses to elicit a quantifiable incidence of DCS.

The symptoms in this study are qualitatively similar to those reported for saturation decompression in general (9–12). The periarticular and limb discomfort are well known symptoms of DCS. For unclear reasons, the lower extremity is more commonly involved than the upper in prolonged exposures, and the opposite is true for short (subsaturation) exposures. Our results are consistent with this previously noted

TABLE 4
SUMMARY OF RESULTS FROM GROUPS 1 AND 2

					VGE				DCS	
Subject	Age	Weight, kg	Height, cm	Body Fat*, %	Onset	Peak Score	Peak Time	Duration	D	Q**
GROUP 1 (P_{N_2} = 1.4 ATA)										
1	31	89	180	20	2	3.9	6	10		
2	27	67	173	17	4	3.2	6	8		X
3	21	61	165	13	2	1.7	3	4		
4	35	85	172	26	2	2.7	4	10		X
5	28	63	162	19	5	1.1	6	1		
6	42	79	180	20	1	3.0	4	11		
7	22	82	167	23	3	1.3	5	9		
8	28	71	179	21	5	0.7	6	1		
9	43	81	176	20	1	2.1	4	8		
10	46	66	163	21	1	4.0	4	11		
11	31	71	174	19	1	3.6	5	8		
12	24	81	178	21	2	1.6	9	10		X
13	31	76	175	23	—	0.0	—	0		
14	20	64	170	8	2	0.5	2	1		
15	39	63	165	8	1	2.8	2	8		
16	30	83	184	12	1	3.2	4	11		X
17	32	91	185	23	1	3.4	4	11		X
18	42	63	171	13	—	0.0	—	0		
19	37	117	179	40	1	3.3	2	11		
Mean	32	76	174	20	2	2.2	5	7		
SD	8	13	7	7	1	1.3	2	4		
GROUP 2 (P_{N_2} = 1.5 ATA)										
1	26	90	177	25	1	2.7	2	11		
2	24	99	186	16	1	3.0	5	11		
3	26	72	177	11	1	3.7	3	11		
4	23	88	179	22	1	4.0	3	11		X
5	28	80	173	14	2	2.0	3	10		X
6	22	80	166	18	0.5	0.9	1	11.5		
7	28	70	177	16	0.5	3.3	2	11.5	X	
8	20	62	171	6	0.5	1.7	3	11.5	X	
9	22	89	167	22	0.5	2.4	4	11.5	X	
10	22	73	170	16	1	2.9	2	8		
11	34	75	180	14	1	3.5	5	11	X	
12	33	82	174	28	1	3.9	5	11		
13	22	59	165	12	3	1.0	5	3		
14	38	96	179	21	1	3.9	2	11		
15	31	79	169	19	0.5	3.8	3	11.5		X
Mean	27	80	174	17	1†	2.9	3†	10†		
SD	5	11	6	6	1	1.1	1	2		

*Determined by hydrostatic weighing. **D = definite; Q = questionable. †Significantly different from Group 1 ($P < 0.05$) by unpaired t test.

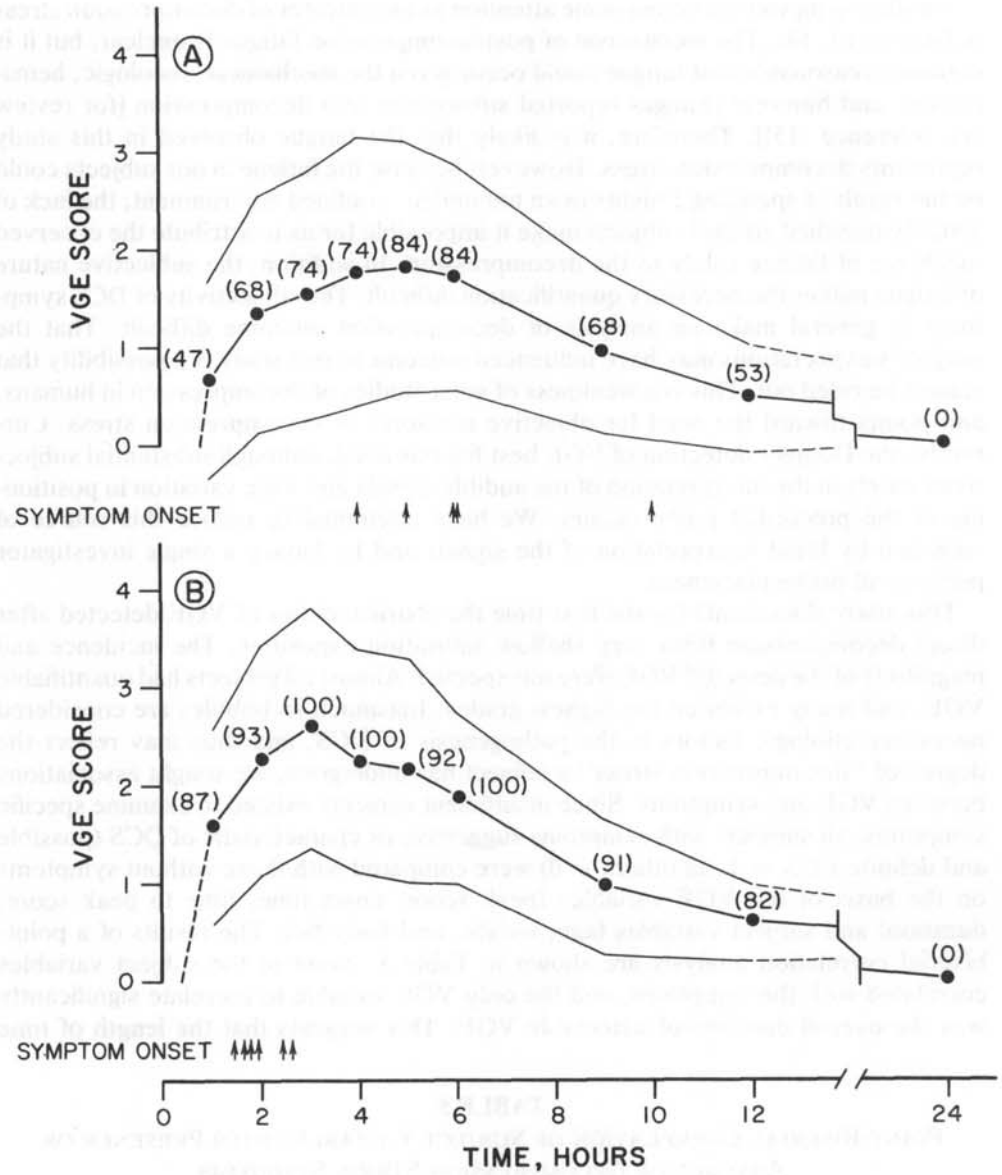

Fig. 1. VGE curve for group 1 *(A)* and Group 2 *(B)*. *Lines* represent the mean score ± SD. *Numbers* in parentheses are the percent of subjects with detectable VGE of those monitored at that time (subjects undergoing treatment not included). *Arrows* opposite "symptom onset" represent the approximate time subjects noticed either questionable or definite DCS symptoms. Notice that although VGE scores are similar after 4–6 h, VGE remained detectable in a significantly higher percentage of the Group 2 subjects, as compared to Group 1. In neither group, however, were VGE detected at 24 h.

association. Although the etiology of the pain is almost certainly a gas phase because of the immediate response to pressure treatment, the location of the gas, mechanism of the pain, and gas transfer dynamics in the joint remain largely unknown.

Another symptom receiving some attention as an indicator of decompression stress is fatigue (13, 14). The mechanism of postdecompression fatigue is unclear, but it is certainly reasonable that fatigue could occur given the mechanical, rheologic, hematologic, and humoral changes reported subsequent to a decompression [for review *see* reference (15)]. Therefore, it is likely that the fatigue observed in this study represents decompression stress. However, because the fatigue in our subjects could be the result of spending 2 nights in an unfamiliar, confined environment, the lack of suitably matched control subjects make it impossible for us to attribute the observed incidence of fatigue solely to the decompression. In addition, the subjective nature of fatigue makes the necessary quantification difficult. The subjectivity of DCS symptoms in general make an analysis of decompression outcome difficult. That the subject's expectations may have influenced outcome in this study is a possibility that cannot be ruled out. This is a weakness of most studies of decompression in humans, and points toward the need for objective measures of decompression stress. Currently, the Doppler detection of VGE best fits this need, although substantial subjectivity exists in the interpretation of the audible signals and wide variation in positioning of the precordial probe occurs. We have attempted to reduce this source of variation by blind interpretation of the signals and by having a single investigator perform all probe placement.

This study documents for the first time the characteristics of VGE detected after direct decompression from very shallow saturation exposures. The incidence and magnitude of the detected VGE were unexpected. Almost all subjects had quantifiable VGE, and many exhibited the highest grades. Inasmuch as bubbles are considered necessary etiologic factors in the pathogenesis of DCS, and thus may reflect the degree of "decompression stress" a subject has undergone, we sought associations between VGE and symptoms. Since insufficient subjects existed to examine specific symptoms, all subjects with symptoms suggestive or characteristic of DCS (possible and definite DCS = 1, all others = 0) were compared with those without symptoms on the basis of the VGE variables (peak score, onset time, time to peak score, duration) and subject variables (age, weight, and body fat). The results of a point-biserial correlation analysis are shown in Table 5. None of the subject variables correlated with the symptoms, and the only VGE variable to correlate significantly was the overall duration of detectable VGE. This suggests that the length of time

TABLE 5

POINT-BISERIAL CORRELATION OF SUBJECT VARIABLES WITH PRESENCE OR ABSENCE OF DECOMPRESSION STRESS SYMPTOMS

Variable	R	P
Age	−0.194	0.136
Weight	0.071	0.346
Body Fat	−0.072	0.342
VGE Onset	0.027	0.445
VGE Peak	0.214	0.113
VGE Peak Time	0.271	0.078
VGE Duration	0.363	0.017

venous bubbles are present may be more representative of decompression stress than the peak score. However, this analysis may be confounded by the fact that about a third of the symptomatic subjects were treated, and it is not unreasonable to postulate that VGE duration in this group may have been affected in some way by the treatment. In fact, VGE could not be detected in any of the treated subjects immediately after the treatment. However, since the correlation was positive, this factor is unlikely to explain the observed association between symptoms and VGE duration. Alternatively, the treatment could have prolonged VGE duration. In support of this possibility was the recurrence of VGE several hours after treatment in 3 of 4 subjects. Again, however, this is unlikely as the VGE returned at a much lower score than existed before treatment. It remains that VGE duration may be a more representative and objective measure of decompression stress than VGE score.

Next, associations of VGE and subject variables, regardless of whether symptoms were present, were sought. Table 6 shows the results of a correlation analysis. The subject variable to correlate the best with the VGE variables was the body weight. This is not novel, as increased body fat has long been thought to be a risk factor for DCS (16, 17), although associations with higher VGE scores have never been established. However, we only found an association with body weight and not body fat. Thus, the reason for the association with overall body weight is unclear and may be coincidental. There was a loose positive correlation of the peak VGE score with age (but not the other VGE variables), which would also be consistent with current thought on DCS susceptibility (16).

The late recurrence of VGE in subjects treated for DCS has not previously been described. The mechanism may reside in the vasoconstrictive effect of hyperbaric oxygen, which might delay inert gas elimination from poorly perfused vascular beds. Perhaps this is an indication that a lower oxygen partial pressure, with less vasoconstricive effect, is necessary for optimal treatment of DCS. A recent study has shown that a P_{O_2} of 2.0 ATA is optimal for the treatment of spinal cord DCS in dogs (18), but it remains speculative that this is due to less vasoconstriction than produced by treatment at the 2.8 ATA commonly used (used here as well). The effect of hyperbaric oxygen therapy on inert gas elimination and bubble dynamics remains poorly understood. Further study is required.

Aside from the incidence and magnitude of VGE in these exposures, we were also interested in the delay, or latent period, for VGE to appear after decompression. The mean latency in Group 1 was about 2 h (in 4 subjects, no VGE were detected before

TABLE 6

PRODUCT-MOMENT CORRELATION OF VGE VARIABLES WITH SUBJECT VARIABLES

Subject Variable		Onset	Peak	Peak Time	Duration
Age	R	−0.187	0.288	−0.011	0.068
	P	0.153	0.049	0.476	0.352
Weight	R	−0.322	0.400	−0.097	0.537
	P	0.032	0.009	0.298	0.001
Body Fat	R	−0.018	0.276	0.111	0.247
	P	0.461	0.057	0.272	0.079

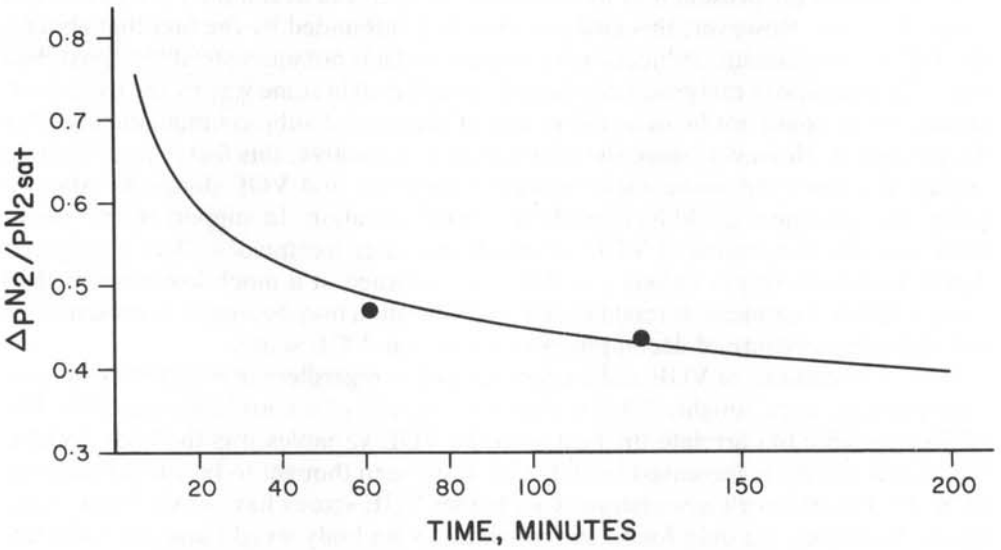

Fig. 2. Onset time of VGE in minutes vs. decompression magnitude expressed as the ratio of the change in nitrogen partial pressure to the saturation nitrogen partial pressure. The *curve* is redrawn from predictions made in the references (19), and the *points* shown represent the mean VGE onset times for Groups 1 and 2.

4 h after decompression). Latency was significantly shorter in Group 2 (about 1 h). A long latent period did not necessarily indicate a subsequent low-peak VGE score. The mean latent period in these exposures fits well with predictions derived from a previous study at this laboratory, where the relationship between VGE latency and no-stop decompression magnitude was explored (Fig. 2) (19). The mechanisms responsible for VGE latency are presumed to be related to bubble formation, growth, and distribution, but otherwise remain poorly understood.

Most decompression studies that included Doppler detection of VGE have reported only the peak VGE score. Because of the results discussed above, it is possible that other characteristics of the individual VGE "curve," such as onset and duration, may more accurately reflect the degree of decompression "stress" experienced than the peak score alone. This is reinforced by our inability to distinguish between Groups 1 and 2 using only the peak score. In this case, only the other VGE variables were sensitive enough to separate the 2 groups. It is possible that the lack of symptom correlation with peak VGE score reflects variability in Doppler probe placement, whereas this would not be expected to influence the *timing* of the VGE curve. Therefore, we would suggest that future studies of decompression which include Doppler bubble detection examine these features in addition to the peak score.

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Eckenhoff RG, Osborne SF, Parker JW, Bondi KR. Remontée directe à la suite de plongées à saturation à l'air peu profondes. Undersea Biomed Res 1986; 13(3):305–316.—Trente-quatre sujets humains en bonne santé furent soumis à des saturations à l'air peu profondes pour 48 h [1.77 ATA (25.5 pieds d'eau salée (fsw)], n = 19, 1.89 ATA (29.5 fsw) n = 15] et ensuite décomprimés à 1 ATA (0 fsw) dans à peu près 2 min. Les symptômes éprouvés comprirent la fatigue, douleur dans les membres et les articulations, maux de tête, myalgie et prurit. Aucun sujet sur 19 ne fut diagnostiqué comme ayant la maladie de décompression (DCS) après l'exposition la moins profonde, mais 4 sur 15 furent diagnostiqués et traités pour la DCS après la plongée la plus profonde. Presque tous les sujets des deux groupes subirent des embolies gazeuses veineuses (VGE) détectables par l'effet Doppler, et qui persistèrent jusqu'à 12 h après la décompression. Les sujets traités eurent une récurrence des VGE plusieurs heures après le traitement à l'oxygène hyperbare. Seulement la durée des VGE, et non le nombre des VGE, correspondit aux symptômes, et seulement le poids corporel des sujets et leur âge montrèrent une correlation avec les variables des VGE. Cette étude démontre que les expositions à l'air hyperbare de cette magnitude ne sont pas aussi bénignes que préalablement considérées.

maladie de décompression
embolie gazeuse veineuse
oxygène hyperbare

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