

Ascent rate, age, maximal oxygen uptake, adiposity, and circulating venous bubbles after diving

D. CARTURAN,¹ A. BOUSSUGES,² P. VANUXEM,³
A. BAR-HEN,⁴ H. BURNET,⁵ AND B. GARDETTE⁶

¹Faculté des Sciences du Sport, Luminy, 13009 Marseille; ²Service de Réanimation Médicale et d'Hyperbarie, Hôpital Salvator, 13274 Marseille; ³Laboratoire de Physiologie et de Pathologie Respiratoire, Faculté de Médecine de Marseille, 13385 Marseille; ⁴Université Aix-Marseille III, Faculté St. Jérôme, Institut Méditerranéen d'Ecologie et de Paléoécologie, 13397 Marseille; ⁵Centre National de la Recherche Scientifique, Unité Propre de Recherche Neurobiologie et Mouvement, 13402 Marseille; and ⁶Comex, Direction Scientifique, 13275 Marseille, France

Received 20 September 1999; accepted in final form 18 March 2002

Carturan, D., A. Boussuges, P. Vanuxem, A. Bar-Hen, H. Burnet, and B. Gardette. Ascent rate, age, maximal oxygen uptake, adiposity, and circulating venous bubbles after diving. *J Appl Physiol* 93: 1349–1356, 2002. First published April 19, 2002; 10.1152/jappphysiol.00723.1999.—Decompression sickness in diving is recognized as a multifactorial phenomenon, depending on several factors, such as decompression rate and individual susceptibility. The Doppler ultrasonic detection of circulating venous bubbles after diving is considered a useful index for the safety of decompression because of the relationship between bubbles and decompression sickness risk. The aim of this study was to assess the effects of ascent rate, age, maximal oxygen uptake ($\dot{V}O_{2\text{ max}}$), and percent body fat on the production of bubbles after diving. Fifty male recreational divers performed two dives at 35 m during 25 min and then ascended in one case at 9 m/min and in the other case at 17 m/min. They performed the same decompression stops in the two cases. Twenty-eight divers were Doppler monitored at 10-min intervals, until 60 min after surfacing, and the data were analyzed by Wilcoxon signed-rank test to compare the effect of ascent rate on the kinetics of bubbles. Twenty-two divers were monitored 60 min after surfacing. The effect on bubble production 60 min after surfacing of the four variables was studied in 47 divers. The data were analyzed by multinomial log-linear model. The analysis showed that the 17 m/min ascent produced more elevated grades of bubbles than the 9 m/min ascent ($P < 0.05$), except at the 40-min interval, and showed relationships between grades of bubbles and ascent rate and age and interaction terms between $\dot{V}O_{2\text{ max}}$ and age, as well as $\dot{V}O_{2\text{ max}}$ and percent body fat. Younger, slimmer, or aerobically fitter divers produced fewer bubbles compared with older, fatter, or poorly physically fit divers. These findings and the conclusions of previous studies performed on animals and humans led us to support that ascent rate, age, aerobic fitness, and adiposity are factors of susceptibility for bubble formation after diving.

susceptibility; bubble formation; scuba diving

THE MOST LIKELY FACTOR FOR initiating decompression sickness (DCS) in scuba diving is believed to be the

formation of inert gas bubbles as a result of supersaturation of the dissolved gas in the tissues and blood.

Even if it is known that most decompressions produce gas bubbles in organisms, only intravascular bubbles with a 40- to 50- μm diameter can be detected by means of ultrasonic Doppler (12, 19). They are detected in the venous circulation, most often in the precordial region, and their sound signals are graded according to a scale of assessment ranging from 0 to 4 (21, 40). For more than 30 years, it has been evident that many postdive decompressions produce Doppler-detectable bubbles in humans (45). Their presence is not sufficient to induce DCS, but it is known that DCS risk is linked to the bubble grades (11, 14, 31, 32, 38, 40). They may be indicative of bubbles elsewhere in the body, which may be a cause of DCS (33). All of the studies have shown that the incidence of DCS is very low for grade 0 (no bubbles) or grade 1 and that the DCS risk increases when grade 2 or higher is observed, with DCS being almost always accompanied by bubbles (38).

Bove et al. (5) have shown that circulatory alterations due to air embolism (increase in right ventricular pressure, stasis in the azygos vein, and reduction in nitrogen elimination) contributed to increase the risk of spinal cord DCS in dogs. Moreover, retrospective studies (4, 46) have emphasized an increased risk of cerebral DCS in divers who had an intracardiac shunt (patent foramen ovale). One may assume that this risk is enhanced in case of numerous vascular bubbles. Because of the association between bubbles and DCS risk, it could be considered that the factors favoring the formation of bubbles are also DCS risk factors and that it is possible to use Doppler bubble detection as an index of safety for diving and decompression profiles, without any DCS symptom.

In animals, a fast decompression rate was demonstrated to be a determining factor for DCS (34, 36, 39).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

Address for reprint requests and other correspondence: D. Carturan, La Prade, 26770 Le Pègue, France (E-mail: carturan@wanadoo.fr).

It has been reported in animals and men that a slow ascent induced less venous gas bubbles than a fast ascent (26, 39). Thus there are some arguments to hypothesize that, in men, a fast ascent may be responsible for an increased bubble production and then for an increased DCS hazard.

In the different decompression tables, ascent rate up to decompression stops has been assessed arbitrarily and then modified by empiricism and intuition. The 18 m/min ascent rate of the US Navy (USN) air tables was for a long time considered as a standard from which the ascent rate of the other decompression tables was determined (23). Now, the ascent rate of the USN tables has been reduced since 1993 to 10 m/min. In the case of the French tables, the recommended ascent rate varies from 9 m/min (1992 French Labour Ministry) to 17 m/min (1990 French Navy tables). The decompression tables used in scuba diving are based on the Haldane concepts (6) and generally do not take into account the individual susceptibility to bubble formation or DCS; they are elaborated for all of the divers without distinction of age or physical fitness. Often, recreational divers use military tables that are made for young and trained men (1, 29).

The incidence of individual factors such as age and adiposity on DCS occurrence and bubble formation is recognized (8–10, 16, 22, 41). In the present practice of diving, it is known that physical fitness is a determining biological factor for DCS hazard, even if few studies have shown its statistical incidence. Gardette (14) has shown that divers who were “bubble producers” had lower scores on the Ruffier index (37) than “no bubble producers.” Rattner et al. (35) reported that rats aerobically trained had a significantly reduced risk of DCS after hyperbaric exposure. Broome et al. (7) subjected pigs to an aerobic training and found a significant reduction of the rate of DCS in the conditioned pigs, compared with the control sample. When various domestic, nonprimate animals are compared with humans, the pig seems the most physiologically similar, and several authors have considered it to be quite a good model for decompression in humans (17, 27, 42).

We have tried to confirm, using bubble Doppler detection, the findings of previous studies carried out on animals and men concerning the factors most currently mentioned in the literature as favoring bubble production and/or DCS risk. For ethical reasons, we chose to limit our study to factors whose assessment is not invasive, i.e., ascent rate, age, aerobic fitness, and adiposity.

METHODS

We have carried out a Doppler monitoring of postdive decompression in a sample of 50 male sport divers who were medically fit to dive (mean age: 37 ± 9.6 yr, mean weight: 80 ± 10.7 kg, mean height: 177 ± 5.7 cm) and who gave informed consent in accordance with the French law about biomedical research. They performed two dives in open water at 35 m for 25 min. The dives were performed on a flat, regular bottom; the descent time (30 s) was included in the dive time. One of the two dives was followed by an ascent at

9 m/min (1992 French Labour Ministry tables); the other one was followed by an ascent at 17 m/min (1990 French Navy tables). The ascent rate was linear and controlled by one investigator who performed all of the dives himself. The control was made by using a chronometer, a depth meter, and a dive computer equipped with a bar graph of ascent (Maestro Pro Beuchat). Ascent time to the first decompression stop was 195 s for the 9-m ascent and 102 s for the 17-m ascent (time difference = 93 s).

In the two cases, the divers performed the decompression stops of the 1992 French Labour Ministry Tables: 3 min at 6 m and 15 min at 3 m. The profile of the dives is represented by Fig. 1.

The investigators randomly imposed the order of the two dives. The dives were performed with a minimal 24-h interval. Only three divers performed their dives with this interval; all of the others performed their two dives with an interval between 3 and 7 days. The ascent rate of the two dives was not determined with a preferential order. Some divers performed first the ascent at 9 m/min; the others performed the ascent at 17 m/min. The temperature of the water varied during the period of the study from 15 to 20°C at the surface and from 12 to 16°C on the bottom. The divers were equipped with neoprene diving suits whose thickness was in accordance with the temperature of the water, and none reported suffering from cold. Before, during, and after the dives, the subjects were required to avoid excessive exercise. After surfacing, venous gas emboli were detected by using ultrasonic Doppler. The divers did not take a warm shower before the end of the bubble detection, in order not to bias the results.

Bubble Detection

Bubble detection was performed with a continuous-wave Doppler apparatus (DUG, Sodelec), equipped with a 5-MHz probe. The subjects were placed in left lateral decubitus, and they laid at rest 1 min before the beginning of the detections. Unlike experimental studies in hyperbaric chambers, our subjects had to exercise in accordance with actual conditions of diving, i.e., to equip themselves, swim, come back to the diving boat, take off, and tidy their equipment. They were affected by the effects of cold and immersion: diuresis, hemoconcentration, change in the repartition of the blood mass, and dehydration. Thus their level of bubble release

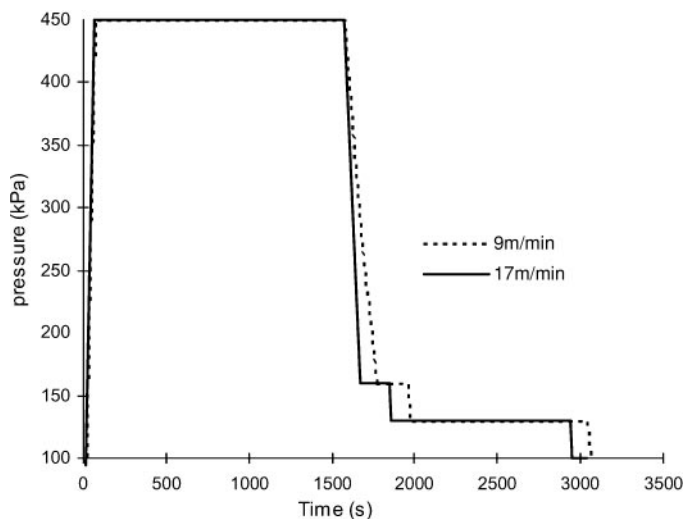


Fig. 1. Profiles of the dives.

corresponded to the actual exercise of all divers respecting the safety rule: little exercise before, during, and after diving. Consequently, we did not monitor bubbles after movement (3 deep knee bends), as it is usually performed in the protocols of the studies conducted in hyperbaric chambers, when the subjects are really at rest. The probe was placed in the precordial site, with the ultrasonic wave being directed into the pulmonary infundibulum. The signals were tape recorded for 2 min and graded in a blind manner by two experienced investigators, according to the Spencer Doppler code (40). Bubble signals were classified on a scale from 0 to 4, on the basis of the number of bubble signals per cardiac cycle and the number of cardiac cycles containing bubbles. If any discrepancy in the interpretation of the signals occurred, the recordings were studied again to reach a consensus. Before the dives, we performed a tape recording of their cardiac signal to have a reference.

Twenty-eight divers were Doppler monitored on the diving boat, at 10-min intervals, until 60 min after surfacing, whereas 22 divers were monitored on the ground 60 ± 4 min (30 s) after surfacing.

Assessment of the Individual Factors

Aerobic fitness was assessed by the maximal exercise test to assess the maximal oxygen uptake ($\dot{V}O_{2\max}$). To assess the adiposity, we used the percent body fat (PBF).

$\dot{V}O_{2\max}$. The maximal exercise test was performed in the Laboratory of Respiratory Function and Muscular Metabolism Exploration of the Sainte Marguerite Hospital (Marseille).

Protocol. Exercise was performed on a cycle ergometer (ER 900 Jaeger). Testing sessions took place in the morning, 2 or 3 h after breakfast. Ambient temperature ranged from 21 to 23°C.

After a 2-min warm-up period, the load was increased by 20 W every 2 min. The test was stopped in case of exhaustion, excessive hypertensive reaction (diastolic pressure >10 mmHg, systolic pressure >25 mmHg), or when the subject presented two of the three accepted criteria of maximal exertion: 1) maximal theoretical heart rate according to the Astrand formula, $220 \text{ beats} - \text{age (in yr)}$; 2) respiratory ratio >1.05; and 3) no increase of oxygen uptake ($\dot{V}O_2$), although the minute respiratory flow kept increasing.

The following parameters were measured (BG Electrolytes, Instrumentation Laboratory): 1) $\dot{V}O_2$ (STPD l/min and $\text{ml} \cdot \text{min}^{-1} \cdot \text{kg body wt}^{-1}$); 2) minute expiratory volume (BTPS l/min); 3) expired carbon dioxide ($\dot{V}CO_2$ STPD ml/min), respiratory ratio ($R = \dot{V}CO_2/\dot{V}O_2$); and 4) respiratory frequency.

The averages corresponding to the 1-min intervals were displayed, and the average of the second minute of each load level was retained for the study. Heart rate was recorded by means of standard electrocardiogram derivations during exertion and recovery periods. Systemic arterial blood pressure was measured at the end of each load level (Sphygmomanometrie).

Measure of the PBF. PBF was measured by electrical impedance with a Bodystat 1500 apparatus. This process is more accurate and reproducible than the classical process of skinfold thickness (18). Before the test, the subjects had not eaten for at least 4 h and had to urinate. They were placed in dorsal decubitus.

Statistical Analysis

Statistical analysis was composed of two parts. In the first analysis, we compared the effects of the two ascents on the kinetics of the bubble scores obtained at 10-min intervals

until 60 min after surfacing in the group of 28 divers. In the second analysis, we analyzed the effects of the variables, ascent rate, age, $\dot{V}O_{2\max}$, and PBF, on the bubble grades detected 60 min after surfacing, on the whole sample of 47 divers.

Effect of ascent rate on bubble kinetics. The Doppler scores were compared in the slow ascent vs. fast ascent at each period of measurement, i.e., 10, 20, 30 min, etc., until 60 min, in the group of 28 divers, using a paired test, i.e., Wilcoxon matched-pairs signed-rank test. The *P* values obtained were then adjusted and corrected according to Holm's step-down procedure (25). One may notice that, before 60 min, many bubble scores are not affected, and, therefore, these values are eliminated from the analysis. This effect on the sample sizes and the conclusions of the test concern only divers presenting differences in bubble scores for the different ascent rates.

Effect of ascent rate and individual variables on bubble production. First, a multinomial log-linear model was used to study the relationship between bubble scores and the four variables. In addition, the potential for interaction among these variables was also considered by the formulation of a multinomial log-linear model. The interactions eligible for inclusion were determined by examining the Akaike Information Criterion (AIC). It should be emphasized that lower values of AIC indicate a best fitting model (2). Wilcoxon tests were computed with Sigmasat (Jandel Scientific), and multinomial log-linear models were done with S-Plus (Ref. 44, section 7.3). We used the multinom function of the nnet library. A Poisson model could be fitted, but, in this analysis, the number of individuals is fixed. This condition on the marginals of the Poisson model leads to a multinomial model. Because the bubble score is an ordinal factor, it is also possible to try a proportional odds model. In fact, results (in term of AIC) are not good (and not presented here). A reasonable explanation is the inadequacy of the bubble scale for the proportional odds model (see Ref. 13, section 3, for details on generalized linear models).

RESULTS

None of the divers had DCS.

Effect of Ascent Rate on Bubble Kinetics

The Doppler signals, graded according to the Spencer scale, are shown in Table 1. The bubble grades were significantly increased after the 17 m/min ascent compared with the 9 m/min ascent at each measurement interval, except at the 40-min interval (Table 1). Percentages of increased bubble scores after fast ascent are as follows: 10 min, 50% (14 of 28 dives); 20 min, 39.3% (11/28); 30 min, 32.14% (9/28); 40 min, 25% (7/28); 50 min, 50% (14/28); and 60 min, 53.57% (15/28).

Effect of Ascent Rate and Individual Variables on Bubble Production

Table 2 presents the raw data to be used in the predictive model of bubble scores: ascent rate, age, $\dot{V}O_{2\max}$, and PBF. Table 3 presents estimates in the main effects model, along with their estimated standard error. AIC and residual deviance for this model are also indicated. The results of the multinomial log-linear model, i.e., including interactions between covariates, are presented in Table 4.

Table 1. Grades of bubbles detected at 10-min intervals

Diver No.	Interval, min											
	10		20		30		40		50		60	
	Ascent rate, m/min											
	9	17	9	17	9	17	9	17	9	17	9	17
1	0	1	0	1	1	1	1	1	0	1	0	0
2	0	1	0	2	2	2	1	2	1	2	1	2
3	0	0	0	1	1	2	2	2	1	1	0	0
4	0	1	1	1	1	2	2	2	1	1	0	1
5	0	2	2	2	3	3	3	3	2	3	1	2
6	2	3	3	3	3	3	3	3	3	3	3	3
7	3	4	4	4	4	4	4	4	4	4	3	4
8	0	0	0	1	0	1	0	0	0	0	0	0
9	2	2	3	3	3	3	3	3	1	3	0	3
10	2	3	3	3	3	3	3	3	3	3	2	3
11	0	1	0	1	2	2	1	1	1	2	0	0
12	0	1	0	2	1	1	1	1	0	1	0	0
13	1	0	1	1	1	2	1	2	0	2	0	1
14	0	0	0	1	1	2	1	1	0	0	0	0
15	0	0	0	0	1	1	1	2	1	1	0	0
16	0	0	0	0	1	1	0	1	0	1	0	0
17	2	2	3	3	3	3	3	3	3	2	3	3
18	1	2	2	2	3	3	3	3	3	2	3	3
19	1	1	1	3	3	3	3	3	2	3	1	3
20	3	3	3	3	3	4	4	4	3	4	3	4
21	0	1	0	1	1	2	1	2	0	1	0	1
22	3	3	3	3	3	3	3	3	3	3	3	3
23	1	0	2	1	2	2	2	3	1	3	0	2
24	3	3	3	3	3	3	3	3	3	3	3	3
25	2	3	3	3	3	3	3	3	2	3	1	3
26	1	2	2	3	2	3	3	3	3	3	3	3
27	1	3	1	3	1	3	1	3	1	3	0	2
28	2	1	2	1	2	1	1	0	1	0	0	0
<i>P</i>	0.011	0.0104	0.0196	0.0546	0.0008	0.0002						
<i>P</i> *	0.033	0.0416	0.0392	0.0546	0.004	0.0012						
<i>P</i> †	0.0416	0.0416	0.0416	0.0546	0.004	0.0012						
	S	S	S	NS	S	S						

P values are Wilcoxon's *P* values. *Holm adjusted *P* values: Wilcoxon's *P* values were arranged in increasing order. The first *P* value was calculated as (Wilcoxon's *P*) (no. of intervals), i.e., 6. The second *P* value as (Wilcoxon's *P*) (no. of intervals - 1), i.e., 5, and so on until all intervals have been compared. †Corrected *P* values in order to maintain the monotonicity of ranking. If a *P* value was lower than the previous one, then this *P* value was declared to be the same as that for the preceding comparison. S, significant; NS, not significant.

The difference of AIC criterion between the model with and without the variable follows approximately a χ^2 distribution. This is a classic way to test that a variable has a significant effect on the model (see Ref. 44, for example). It is also possible to test a particular coefficient by noting that asymptotically a coefficient divided by the standard error follows a normal law. These tests are not very interesting because coefficients are not independent and the level would be strongly affected.

To give a graphic description of the data, we have divided our sample of divers on both sides of the median of the variables, and we have realized histograms for age (Fig. 2), $\dot{V}O_{2\max}$ (Fig. 3), and PBF (Fig. 4).

DISCUSSION

We emphasize that our study was conducted in actual diving conditions, because more bubbling has been

reported when the dives were performed in open water rather than when they were performed in hyperbaric chambers (15). The first part of the statistical analysis has shown a significant relationship between a slow ascent and decreased bubble production. As ambient pressure decreases faster at 17 m/min than at 9 m/min, the increase in the gas pressure gradient between tissues and alveoli is greater as well; it may then reach the critical ratio pressure for nucleation and promote bubble formation. The ascent at 9 m/min was 93 s

Table 2. Individual factors and Doppler scores

Diver No.	Age, yr	$\dot{V}O_{2\max}$, ml·min ⁻¹ ·kg ⁻¹	PBF, %	Bubble Score	
				Ascent rate, m/min	
				9	17
1	47	53	18.1	0	1
2	54	31.5	21.3	3	3
3	47	16.4	16.2	2	3
4	30	40.6	17.5	0	0
5	26	41.2	13.4	0	1
6	32	40.8	10.8	1	1
7	54	19.7	16.1	0	0
8	45	19.8	17.2	3	3
9	48	20.2	26.3	1	2
10	19	56	6.4	1	0
11	41	21.4	24.3	1	3
12	37	23.3	21.6	3	3
13	26		8.4	0	0
14	47	31.5	19	1	3
15	26	40	18.5	0	2
16	46	26.9	20.6	3	3
17	26	44	7.4	0	0
18	20	52.4	4.3	0	0
19	26	43.7	3.9	0	0
20	23	47.1	11.5	0	0
21	46	25.5	18.1	1	2
22	31	35	14.1	0	0
23	22	43.5	20.6	0	1
24	36	60.1	12.4	1	2
25	44		17.7	3	3
26	33		21.1	4	4
27	27	56	11.6	0	0
28	37	33.9	13.2	0	3
29	46	45.6	11.8	2	3
30	30	40.7	24.9	0	0
31	37	36	11.5	0	0
32	49	52.6	15.3	0	1
33	25	52.2	14	0	0
34	30	40.7	18.9	0	0
35	32	43.9	18.3	0	0
36	48	40.3	18.7	2	3
37	30	31	20.2	2	3
38	36	45	12.8	1	3
39	42	38.3	22	3	4
40	47	46.3	12.6	0	1
41	49	32	16.6	3	3
42	33	39.5	19	0	2
43	39	36.7	18.3	3	3
44	46	37.6	18.5	3	3
45	38	41.2	20	0	1
46	43	53	17	0	2
47	43	37.2	19.6	0	0

$\dot{V}O_{2\max}$, maximal O₂ uptake; PBF, percent body fat. Average values: age = 37 ± 9.6 yr; $\dot{V}O_{2\max}$ = 38.9 ± 10.8 ml·min⁻¹·kg⁻¹; PBF = 16.2 ± 5.2%.

Table 3. *Parameter estimates of multinomial log-linear model: main effects model*

Grade	Intercept	Age	\dot{V}_{O_2}	PBF	Ascent Rate
<i>Coefficients</i>					
1	-3.992	0.069	0.005	0.029	-0.277
2	-5.401	0.093	-0.022	0.095	-0.648
3	-4.319	0.134	-0.073	0.080	-0.877
4	-38.842	0.228	0.146	0.823	-5.458
<i>Standard errors</i>					
1	2.718	0.039	0.038	0.078	0.324
2	3.606	0.048	0.045	0.103	0.397
3	3.229	0.046	0.039	0.092	0.356
4	13.504	0.203	0.201	0.674	14.899
Residual deviance	186.00				
AIC	226.00				

A log-linear model is fitted, with coefficients zero for the first class. \dot{V}_{O_2} , O_2 uptake; AIC, Akaike Information Criterion.

longer than the ascent at 17 m/min; this seems to have been sufficient to reduce bubble formation and thus DCS risk. This is consistent with previous studies on decompression (see Introduction). The analysis of the bubble kinetics (Table 1) has shown that the significance of ascent rate was higher from 50 min after surfacing. This could mean an enhanced safety for the divers in case of repetitive dives because of a reduced gas load and a reduced, preexisting gas phase in the interval.

In the second part of the statistical analysis, according to the AIC, the best fit was obtained with the interactive model rather than with the main effects model. The best fitting model includes the four covariates and interaction terms between $\dot{V}_{O_{2\max}}$ and age as well as $\dot{V}_{O_{2\max}}$ and PBF. It should be emphasized that one implication of the main effects model is that the effects of any variable do not depend on the values assumed by the other variables. Thus, had one not fit a model with interaction terms, the effect of age and PBF on $\dot{V}_{O_{2\max}}$ would have been missed. Such interactions are not surprising.

Indeed, it is well known that $\dot{V}_{O_{2\max}}$ decreases with increasing age, and generally adiposity is associated with poor physical fitness. Many studies have con-

cluded that aerobic fitness and adiposity were among the possible causes of individual susceptibility (see the introduction), but, so far, no data have shown a relationship for men between $\dot{V}_{O_{2\max}}$ and bubbles.

For Mebane and McIver (28), obesity and poor physical condition generally coexist and represent a hazard to the divers. Obesity is widely recognized as a DCS risk factor because of the high solubility of nitrogen in lipids. Dembert et al. (10) found significantly higher measures of weight and skinfold thickness in USN divers who experienced DCS compared with those who remained free of DCS. However, they did not find an association of DCS and age in studies of military divers. Broome et al. (7) commented on these findings, hypothesizing that, in military diving units, both senior and junior divers are required to maintain a high level of aerobic fitness. For Lam and Yau (22), DCS susceptibility is increased by age, but this might be due to an increase in adiposity because of age. If age and obesity are widely mentioned in the literature as DCS risk factors, there are few data about aerobic fitness. Curley et al. (9) suggested that the importance of obesity as a risk factor for DCS may be overstated. Furthermore, Broome et al. (7) hypothesized that, in epidemiological studies in which body weight has been associated with increased DCS risk, the underlying association was, in fact, with poor aerobic fitness, for which being overweight or relatively obese was a surrogate indicator. They have shown that aerobic exercise reduces the risk of DCS in swine, regardless of age, adiposity, and weight. Vann (43) had reported that aerobically trained runners appeared to be at lower risk for venous bubbling and bends than weight lifters or sedentary subjects. Rattner et al. (35) speculated that increased capillary density of muscle, as a result of training, might explain the decreased DCS rate observed in their treadmill-exercised rats. McKirnan et al. (27) have reported a reduction of 20% in cerebral blood flow at rest, in a sample of exercise conditioned pigs, compared with the untrained control pigs. Thus, Broome et al. (7) hypothesized that such changes in cerebral blood flow were representative of proportional changes in the central nervous system blood flow gen-

Table 4. *Parameter estimates of multinomial log-linear model: interactive model*

Grade	Intercept	Age	\dot{V}_{O_2}	PBF	Ascent Rate	Age \times \dot{V}_{O_2}	$\dot{V}_{O_2} \times$ PBF
<i>Coefficients</i>							
1	-17.232	-0.400	0.276	2.073	-0.429	0.012	-0.049
2	-14.804	-0.368	0.183	1.914	-0.800	0.012	-0.044
3	13.754	-0.750	-0.644	1.462	-1.094	0.024	-0.032
4	16.460	-43.944	102.162	49.688	-23.909	1.921	1.827
<i>Standard errors</i>							
1	0.028	0.228	0.031	0.608	0.356	0.006	0.015
2	0.018	0.224	0.049	0.609	0.424	0.006	0.015
3	0.015	0.208	0.084	0.589	0.396	0.006	0.015
4	0.003	0.216	0.061	0.099	0.003	0.007	0.011
Residual deviance	157.00						
AIC	213.00						

A log-linear model is fitted, with coefficients zero for the first class.

Fig. 2. Grades of bubbles according to age and ascent rate. *Left*: age <37 yr; *right*: age ≥37 yr. Higher grades of bubbles are associated with elevated age and faster ascent.

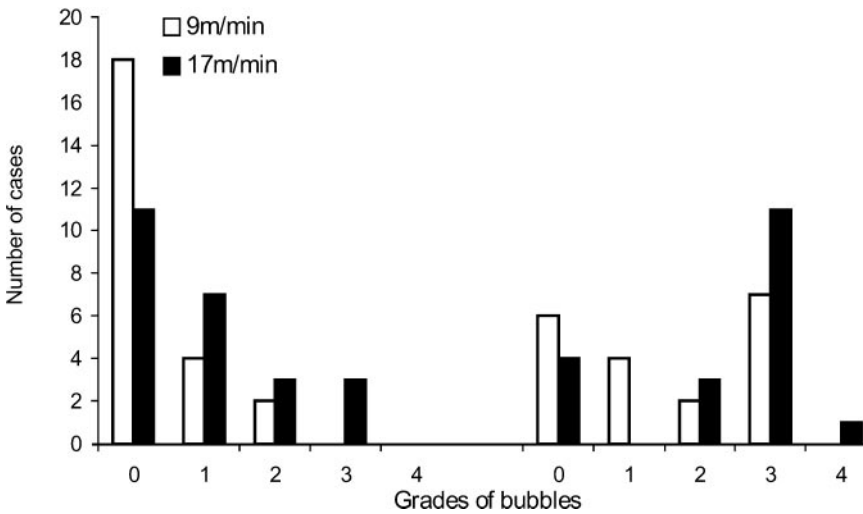
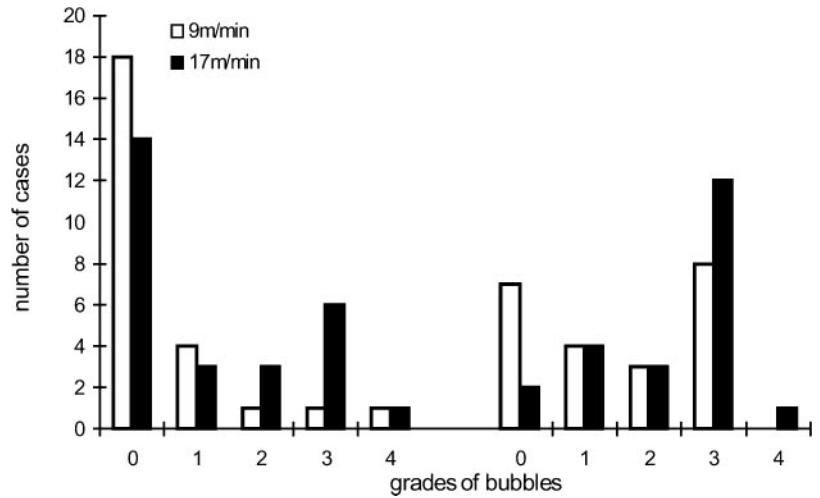
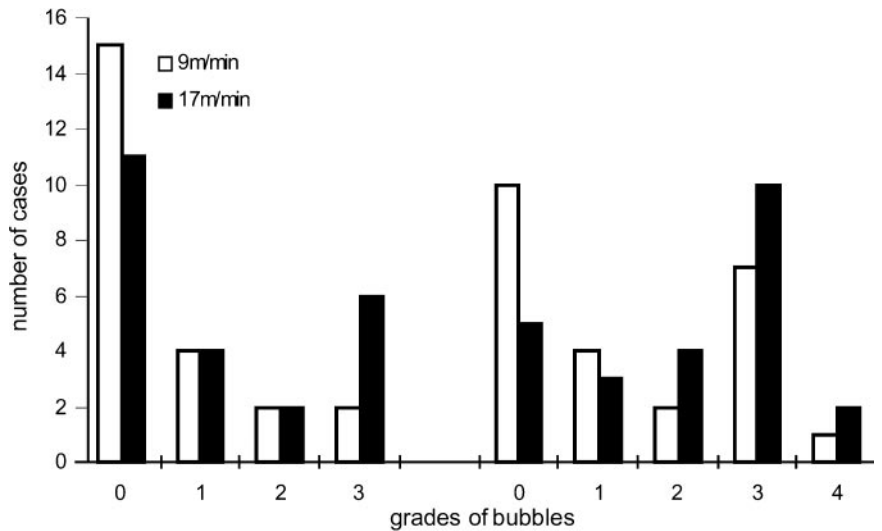


Fig. 3. Grades of bubbles according to maximal O₂ uptake ($\dot{V}O_{2max}$) and ascent rate. *Left*: $\dot{V}O_{2max} \geq 40$ ml·min⁻¹·kg⁻¹; *right*: $\dot{V}O_{2max} < 40$ ml·min⁻¹·kg⁻¹. Higher grades of bubbles are associated with poor aerobic fitness and faster ascent.

Fig. 4. Grades of bubbles according to percent body fat (PBF) and ascent rate. *Left*: PBF <17.5%; *right*: PBF ≥17.5%. Higher grades of bubbles are associated with higher PBF and faster ascent.



erally and thus also in spinal blood flow. They assumed that reduced spinal cord blood flow would lessen the gas uptake by spinal cord tissue and reduce the risk of neurological DCS. Moreover, they assumed that the rheological effects of aerobic training [a rise in plasma volume and fall in hematocrit (3, 24)] might explain this reduction of the DCS rate in aerobically trained divers.

Thus we can support that age and the combination of aerobic fitness and adiposity take a part in the bubble formation process and that the divers would be well advised to keep physically fit and slim by aerobic training. Our conclusion is consistent with Broome et al. (7), who underlined that “an individual could manipulate his personal risk by being aerobically fit or unfit.” Moreover, our findings suggest that elderly, poorly fit, and fat divers could reduce their bubble production and then their DCS risk on the one hand by improving their physical fitness and on the other hand by ascending slowly. Obviously, the presence of bubbles does not explain the whole occurrence of DCS, and some authors have reported an adaptation to decompression stress that seems to be a subsequent response of the immune system (20). Nevertheless, it is widely recognized that, because of the linkage between DCS and bubbles, it is in the divers’ best interest to prevent as much bubble formation as possible. As Moon et al. wrote (30): “the probabilistic models on which tables and computers are based should reflect the individual reality of the divers, to enable them to conduct their dives in accordance with their individual characteristics.”

It would be of interest to conduct further studies in men to check the effect on bubbles of an aerobic training that would make $\dot{V}O_{2\max}$ increase and PBF decrease and, in this way, verify whether it is possible to lighten the effects of age on bubble susceptibility.

REFERENCES

- Aharon-Peretz J, Adir Y, Gordon CR, Kol S, Gal N, and Melamed Y. Spinal cord decompression sickness in sport diving. *Arch Neurol* 50: 753–756, 1993.
- Akaike H. Factor analysis and AIC. *Psychometrika* 52: 317–332, 1997.
- [Anonymous]. “Anaemia” in athletes. *Lancet* 1: 1490–1491, 1985.
- Bove A. Risk of decompression sickness with patent foramen ovale. *Undersea Hyperb Med* 25: 175–178, 1998.
- Bove A, Hallenbeck JM, and Elliott DH. Circulatory responses to venous air embolism and decompression sickness in dogs. *Undersea Biomed Res* 1: 207–220, 1974.
- Boycott AE, Damant GCC, and Haldane JS. The prevention of compressed-air illness. *J Hyg Lond* 8: 342–443, 1908.
- Broome JR, Dutka AJ, and McNamee GA. Exercise conditioning reduces the risk of neurologic decompression illness in swine. *Undersea Hyperb Med* 22: 73–85, 1995.
- Carlioz M, Comet M, and Gardette B. About individual factors influence in man on the bubble formation in air diving decompression. In: *Proceedings of the XIth Annual Meeting of the EUBS Congress on Diving and Hyperbaric Medicine*. Göteborg, Sweden: European Underwater and Baromedical Soc., 1985, p. 229–239.
- Curley MD, Robin GJ, and Tahlmann ED. Percent body fat and human decompression sickness (Abstract). *Undersea Biomed Res* 16, Suppl: 33, 1989.
- Dembert ML, Jekel JF, and Mooley LW. Health risk factors for the development of decompression sickness among US Navy divers. *Undersea Biomed Res* 11: 395–406, 1984.
- Eatoock BC. Correspondence between intravascular bubbles and symptoms of decompression sickness. *Undersea Biomed Res* 11: 326–329, 1984.
- Eckenhoff RG, Olstad CS, and Carrod G. Human-dose response relationship for decompression and endogenous bubble formation. *J Appl Physiol* 69: 914–918, 1990.
- Fahrmeir L and Tutz G. *Multivariate Statistical Modelling Based on Generalized Linear Models* (2nd ed.). New York: Springer, 2001, p. 517.
- Gardette B. Correlation between decompression sickness and circulating bubbles in 232 divers. *Undersea Biomed Res* 6: 99–107, 1979.
- Gardette B, Le Chuitton J, Sciarli R, and Fructus X. Contrôle médico-physiologique des tables à l’air. In: *Proceedings of the VIIth Annual Meeting of the EUBS on Diving and Hyperbaric Medicine*. Cambridge, UK: European Underwater and Baromedical Soc., 1981.
- Gray JS. Constitutional factors affecting susceptibility to decompression sickness. In: *Decompression Sickness*, edited by Fulton JF. Philadelphia, PA: Saunders, 1951, p. 182–191.
- Hastings AB, White FC, Sanders TM, and Bloor CM. Comparative physiological responses to exercise stress. *J Appl Physiol* 52: 1077–1083, 1982.
- Heitmann BL. Evaluation of body fat estimated from body mass index, skinfolds and impedance. A comparative study. *Eur J Clin Nutr* 44: 831–837, 1990.
- Hills BA and Butler BD. Size distribution of intra-vascular air emboli produced by decompression. *Undersea Biomed Res* 8: 163–170, 1981.
- Kayar SR, Aukhert EO, Axley MJ, Homer LD, and Harabin AL. Lower decompression sickness in rats by intravenous injection of foreign proteins. *Undersea Hyperb Med* 24: 329–335, 1997.
- Kisman KE, Masurel G, and Guillerm R. Bubble evaluation code for Doppler ultrasonic decompression data (Abstract). *Undersea Biomed Res* 5, Suppl: 28, 1978.
- Lam TH and Yau KP. Analysis of some individual risk factors for DCS in Hong Kong. *Undersea Biomed Res* 16: 283–292, 1989.
- Lanphier EH. A historical look at ascent. In: *Biomechanics of Safe Ascents Workshops*, edited by Lang MA and Ergstrom GH. Costa Mesa, CA: American Academy of Underwater Sciences, 1990, p. 5–8. (AAUS Diving Safety Publication AAUSDSP-BSA-01-90)
- Letcher RL, Pickering TG, Chein S, and Laragh JH. Effects of exercise on plasma viscosity in athletes and sedentary normal subjects. *Clin Cardiol* 4: 172–179, 1981.
- Ludbrook J. Multiple comparison procedures updated. *Clin Exp Pharmacol Physiol* 25: 1032–1037, 1998.
- Marroni A and Zannini D. Effetti della variazione della velocità di risalita sulla produzione di bolle gassose circolanti dopo immersioni ad aria compressa. *Minerva Med* 72: 3567–3572, 1981.
- Mc Kirnan FC, White FC, Guth BD, and Bloor CM. Exercise and hemodynamic studies in swine. In: *Swine in Cardiovascular Research*, edited by Stanton HC and Mersmann HJ. Boca Raton, FL: CRC, 1986, vol. 2, p. 105–119.
- Mebane GY and McIver NKI. Fitness to dive. In: *The Physiology and Medicine of Diving* (4th ed.), edited by Bennett P and Elliott D. London: Saunders, 1993, p. 52–76.
- Méliet JL. Les tables de plongée à l’air de la Marine Nationale: Historique, nouveaux développements. In: *Physiologie et Médecine de la Plongée*, edited by Broussolle B. Paris: Ellipses, 1992, p. 565–587.
- Moon RE, Vann RD, and Bennett PB. The physiology of decompression illness. *Sci Am* 273: 70–77, 1995.
- Nashimoto I and Gotoh Y. Relationship between precordial Doppler ultrasound records and decompression sickness. In: *Proceedings of the 6th Underwater Physiology Symposium Abstracts*, edited by Shillings CW and Beckett MW. Bethesda, MD: Undersea Medical Society, 1978, p. 497–501.

32. **Neuman TS, Hall DA, and Linaweaver PG.** Gas phase separation during decompression in man: ultrasound monitoring. *Undersea Biomed Res* 3: 121–130, 1976.
33. **Nishi RY.** Doppler and ultrasonic bubble detection. In: *The Physiology and Medicine of Diving* (4th ed.), edited by Bennett PB and Elliott D. London: Saunders, 1993, p. 433–453.
34. **Pollard GW, Marsh PL, Fife CE, Smith LR, and Vann RD.** Ascent rate, post dive exercise, and decompression sickness in the rat. *Undersea Hyperb Med* 22: 367–376, 1995.
35. **Rattner BA, Gruenau SP, and Altland PD.** Cross-adaptive effects of cold, hypoxia or physical training on decompression sickness in mice. *J Appl Physiol* 47: 412–417, 1979.
36. **Reinertsen RE, Flook V, Koteng S, and Bribbakk AO.** Effect of oxygen tension and rate of pressure reduction during decompression on central gas bubbles. *J Appl Physiol* 40: 229–235, 1998.
37. **Ruffier X.** *Traité d'Education Physique*. Paris: Physique, 1943.
38. **Sawatzky KD and Nishi RY.** Intravascular Doppler-detected bubbles and decompression sickness. *Undersea Biomed Res* 17, *Suppl*: 34–35, 1990.
39. **Smith KH and Stayton L.** *Hyperbaric Decompression by Means of Bubble Detection*. Seattle, WA: Virginia Mason Research Center, 1978. (ONR Rep. N0001469-C-0402)
40. **Spencer MP and Johanson DC.** *Investigation of New Principles for Human Decompression Schedules using the Doppler Ultrasonic Blood Bubble Detector*. Technical Report to the Office of Naval Research. Seattle, WA: Invest. Environ. Med. Physiol., 1974. (Tech. Rep. ONR contract N000 14-73-C-0094)
41. **Sulaiman ZM, Pilmanis AA, and O'Connor RB.** Relationship between age and susceptibility to altitude decompression sickness. *Aviat Space Environ Med* 68: 695–698, 1997.
42. **Swindle MM, Moody DC, and Phillips LD** (Editors). *Swine as Models in Bio-medical Research*. Ames: Iowa State Univ. Press, 1992.
43. **Vann RD.** *The Proceedings of the 1990 Hypobaric Decompression Sickness Workshop*, edited by Pilmanis AA. Brooks Air Force Base, TX: Air Force Systems Command, 1992, comment session 1, discussion 4, p. 165. (Rep. AL-SR-1992-0005)
44. **Venables WN and Ripley BD.** *Modern Applied Statistics With S-Plus* (3rd ed.). New York: Springer, 1999, p. 501.
45. **Walder DN, Evans A, and Hempleman HV.** Ultrasonic monitoring of decompression. *Lancet* 1: 897–898, 1968.
46. **Wilmshurst PT, Byrne JC, and Webb-Pepploe MM.** Relation between interatrial shunts and decompression sickness in divers. *Lancet* 2: 1302–1306, 1989.

