Cardiovascular Deconditioning Occurs During a 7-Day Saturation Dive at 31 ATA

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Cardiovascular deconditioning (CD) has been reported to occur within 24–48 h of exposure to 4, 11, or 31 ATA environment and following decompression to sea level pressure. The CD was indicated by orthostatic intolerance, exaggerated cardiovascular responses to a passive tilt, an elevated resting heart rate and a reduced stroke volume postdive. In this dive, one of the New Seatopia series, we used a non-syncope criterion, the cardiovascular index of deconditioning (CID; Bungo MW, Johnson PJ Jr. Aviat Space Environ Med 1983; 54:1001), to evaluate CD in 3 male subjects. The CID sums the changes in heart rate and blood pressure in response to orthostatic stress. An elevated CID indicates CD. We used a passive 70° head up tilt as the orthostatic stress. The CID was measured before and after a bout of underwater exercise at predive, during the early, mid, and late exposure of the 7-d 31 ATA, and after the dive. The CID and circulatory responses to tilt were similar before and after the exercise. The CID increased ($p < 0.05$) from the predive value of 20 ± 1.6 to 25 ± 0.9 on the 2nd day, to 25 ± 0.8 on the 4th day at 31 ATA, indicating the presence of CD at the early and mid periods of hyperbaric exposure. However, CID was indifferent (18 ± 0.6) from the predive on the 7th day at 31 ATA. The increased CID corresponded to decreases in plasma volume during the early and mid periods of 31 ATA exposure. However, CID rose to 29 ± 2 ($p < 0.05$) postdive in agreement with other indicators of CD, but the plasma volume was normal. We concluded that hyperbaric diuresis is one major factor in the development of the acute phase of hyperbaric CD, and the prolonged relative inactivity may account for the late phase of hyperbaric CD. Daily exercises at a moderate intensity were not effective in intervening the development of hyperbaric CD.

CARDIOVASCULAR deconditioning (CD) has been observed during a 7-d saturation dive at 31 ATA in a previous study (1). The CD was demonstrated by the occurrence of syncopal episodes and by examining circulatory responses to a passive head-up tilt. The acute phase of CD at hyperbaria cannot be attributed to a prolonged inactivity, because CD was observed within 24–48 h of the pressure exposure. However, a prolonged (3 weeks) relatively low activity could not be ruled out as a causative factor, because CD persisted postdive. Among the indicators of CD, the cardiovascular index of deconditioning (CID) of Bungo and Johnson (4) is the simplest one to determine, and requires no syncope as the end point. Other indicators that suggest the presence of CD after a saturation dive include an elevated resting heart rate, a reduced work capacity, and a reduced stroke volume (1,14).

Since the first demonstration of CD by Arita and associates (1), others have reported CD during and following a hyperbaric exposure. Mateev and colleagues (12) observed an elevated resting heart rate and a reduced fitness score after dives of 48–50 h at 4 or 11 ATA. They concluded that "the ability of the cardiorespiratory system to readapt to normobaric conditions is hindered and delayed." Holthaus (6) reported increased incidence of postdive vagovasal syncopes in dives at the German Underwater Simulator. In the present dive, underwater exercise at 31 ATA was an essential component of the New Seatopia project. It provided an opportunity to confirm hyperbaric-induced CD and to examine whether a moderate underwater exercise could modify the development of CD. An examination of the effect of underwater exercise on the orthostatic tolerance is important for deep-sea divers, because if orthostatic intolerance occurred after an underwater exercise in hyperbaria, additional cautions must be taken to prevent accidents in actual open sea excursions at depth.

METHODS

Facility and Diving Profile

The experiment, a part of the New Seatopia Project (1982–1991), was carried out in the hyperbaric simulation chamber at the Japan Marine Science and Technology Center (JAMSTEC), Yokosuka, Japan. The main purpose of the New Seatopia dive series was to prepare for and carry out open-sea lockout of divers at 300 m depth. Therefore, underwater exercise was an essential component of the Project. This dive (Nov. 20–Dec. 11, 1985) consisted of 3 d of predive, 7 d of 31 ATA saturation, 12 d of decompression, and a 2-d postdive period (Fig.

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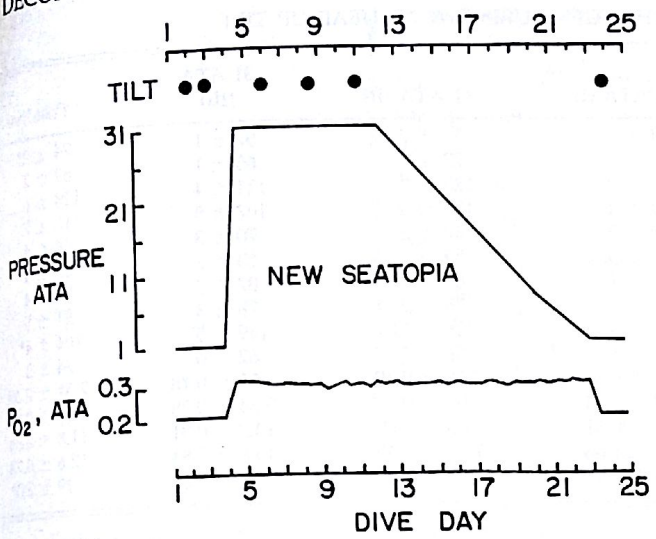

Fig. 1. Diving profile and ambient P_{O_2} of a New Seatopia dive, which was carried out between November 20 and December 11, 1985, at the Japan Marine Science and Technology Center (JAMSTEC), Yokosuka, Japan. Dots indicate where tilt tests were conducted.

1). The temperature and relative humidity inside the chamber were maintained at 26 ± 0.5°C, and 60 ± 10% during the pre- and postdive periods. At 31 ATA, the chamber gas was composed of 0.4 ATA O_2, 0.79 ATA N_2, less than 0.0004 ATA CO_2, and the balance, helium. Chamber temperature and humidity were maintained at 31 ± 0.5°C and 60%, respectively.

Subjects: Three male divers, between the ages 21 and 34 years, served as subjects. The anthropometric characteristics were similar for the three subjects. The ranges of body weight, height, chest circumference, and body surface area were 63.0–67.5 kg, 166.5–167.8 cm, 89–93 cm, and 1.73–1.77 m^2, respectively. All divers entered the chamber at 1000 h on dive day 1 (Fig. 1).

The subjects were professional saturation divers. They were trained in hyperbaric operations and procedures. They also received training for the experimental protocols, were informed of possible risks, and signed a consent form and contract with the Japanese Government. JAMSTEC's Human Experimentation Review Board reviewed and approved all procedures. The subjects could withdraw from this portion of the study. However, immediate removal from the chamber was impossible after the first day of compression.

Tilt table and orthostatic test protocol: The tilt table was constructed of aluminum (180 cm long, 53 cm wide) with a 25-cm wide foot-board and was designed to fit the aisle between two rows of bunk beds in the living chamber and to tilt between 0° (supine) and 70°. The subject was asked to lie supine quietly for 10 min on the tilt table and measurements were made during the following 10 min (pre-tilt period). Heart rate (HR), arterial blood pressure (ABP), and cardiac output (CO) were obtained at 5-min intervals during pre-tilt period. The table was then tilted to 70° within 3 s and maintained in the head-up position for 15 min (tilt period). During the tilt period, the HR and ABP were measured at 2.5 min intervals, and CO at 5-min intervals. Care was taken to insure that the subjects were completely relaxed during the tilt.

Measurements: An Omron sphygmomanometer (Model HEM-50, Tateishi-Denki, Kyoto, Japan) with digital display was used for the determination of systolic (sABP) and diastolic (dABP) arterial blood pressures and HR. These values were transcribed manually from the digital display. Mean arterial blood pressure (mABP) was calculated as one-third of pulse pressure plus the dABP. We estimated cardiac output (CO) by using an impedance cardiograph (AI-601G, Nihonkohden, Tokyo, Japan) with the standard four-band electrodes arrangement. Although there is disagreement on using impedance cardiography for CO in absolute terms, this method provides reliable measurement of CO changes in postural stresses (20). The impedance cardiograph was located outside the chamber and the impedance measurement was obtained by way of through-hull connections. The impedance was measured during a brief apnea (less than 5 s) at the functional residual capacity. We followed Kubicek's formula (9) for the calculation of stroke volume (SV). We multiplied SV by HR to obtain CO, and divided mABP by CO to obtain the total peripheral resistance (TPR). The index of cardiovascular deconditioning (CID) was calculated according to Bungo and Johnson (4), as follows:

$$CID = \Delta HR - \Delta sABP + \Delta dABP$$

where ΔHR, ΔsABP, and ΔdABP are the differences in HR, sABP, and dABP, respectively, between pre-tilt and tilt values.

Experimental design: Each subject was tested twice (before and after exercise) at predive on 2 separate days, 6 times (3 before exercise and 3 after exercise) in 3 test days at 31 ATA (subject A on days 2, 5, and 7; subject B on days 3, 4, and 6; and subject C on days 2, 4, and 6), and twice (before and after exercise) postdive on 2 separate days. The 3 test days at 31 ATA represented the early (days 2 and 3), 31 ATA (I); the mid (days 4 and 5), 31 ATA (II); and the late (days 6 and 7), 31 ATA (III). Tilt at 31 ATA was carried out before and after a bout of moderate exercise (2–3 met exercise) underwater which lasted for 21–28 min during a total immersion of 42–53 min. The post-exercise tilt was conducted after removing the wet suit and breathing apparatus, and after confirming the return of respiratory and cardiac parameters to the pre-exercise level. For other studies in this dive, blood was withdrawn from the antecubital vein once predive, three times at 31 ATA (day 2, 5, and 7), and once postdive. Details of blood chemistry and hormonal analysis appeared elsewhere (15).

The relative change in plasma volume was estimated by using Van Beaumont's equation (21). The hematocrit value was determined in triplicate by using the microhematocrit method.

Statistics: For paired comparisons between pre-tilt and tilt, values were averaged over the last 5 min of pre-tilt period and over the entire 15 min tilt period. Longitudinal comparisons were made with one-way ANOVA for repeated measures followed by Student-Neuman-Keuls tests for multiple comparison against the predive control. Values before and after underwater exercise were compared by using paired *t*-tests. We rejected the null hypothesis at $p < 0.05$.

TABLE I. SUMMARY OF CIRCULATORY CHANGES DURING A 70° HEAD-UP TILT.

Measurements	Angle	Predive	31 ATA (I)	31 ATA (II)	31 ATA (III)	Postdive
Heart rate, b·min^{-1}	S	59 ± 3	54 ± 1	59 ± 2	57 ± 1	74 ± 2*
	T	72 ± 4	66 ± 2	73 ± 2	65 ± 1	89 ± 2
Systolic ABP, mm Hg	S	118 ± 3	123 ± 2	123 ± 2	121 ± 4	128 ± 6
	T	103 ± 3	104 ± 4	107 ± 3	107 ± 5	112 ± 7
Diastolic ABP, mm Hg	S	71 ± 3	69 ± 3	62 ± 2	70 ± 3	68 ± 4
	T	63 ± 3	63 ± 3	59 ± 3	63 ± 2	66 ± 4
Mean ABP, mm Hg	S	87 ± 2	87 ± 1	82 ± 1	87 ± 2	88 ± 4
	T	77 ± 3	77 ± 2	75 ± 2	78 ± 3	81 ± 5
Stroke volume, ml·b^{-1}	S	124 ± 9	133 ± 4	126 ± 11	119 ± 5	104 ± 4*
	T	86 ± 12	87 ± 6	86 ± 3	82 ± 6	74 ± 5
Cardiac output, L·min^{-1}	S	6.52 ± 0.24	6.93 ± 0.37	7.26 ± 0.47	6.57 ± 0.18	7.51 ± 0.39
	T	5.52 ± 0.70	5.69 ± 0.44	6.01 ± 0.49	5.34 ± 0.28	6.51 ± 0.36
TPR, mm Hg·L^{-1}·min^{-1}	S	12.0 ± 0.75	12.7 ± 0.61	11.6 ± 0.67	13.3 ± 0.31	11.8 ± 0.55
	T	12.7 ± 1.13	13.9 ± 1.06	12.6 ± 0.45	14.6 ± 0.84	12.6 ± 0.78
CID		20 ± 1.6	25 ± 0.9*	25 ± 0.8*	18 ± 0.6	29 ± 2.0*

Values are mean ± SE for 6 measurements in 3 subjects. S = supine and T = tilt; TPR total peripheral resistance; CID = cardiovascular index of deconditioning; 31 ATA (I), (II), and (III) refer to early, mid, and late periods at 31 ATA, respectively; * represents $p < 0.05$ comparing S and T by paired *t*-tests.

RESULTS

Resting cardiovascular parameters: We observed no significant differences in mABP, SV, CO, and TPR in pretilt throughout the dive, except for a higher HR ($p < 0.01$) and a lower SV ($p < 0.05$) after the dive (**Table I**).

Effect of exercise on circulatory responses to tilt: Comparison of circulatory responses to the tilt test before and after exercise showed no significant differences throughout the dive periods, except during the late 31 ATA period where a greater systolic hypotension ($p < 0.05$) but a lesser diastolic hypotension ($p < 0.05$) occurred during postexercise tilts (**Table II**). Since data obtained before and after the exercise were similar with these two exceptions, we pooled the pre- and postexercise data for further comparisons (Table I).

Fig. 2 shows temporary responses of HR, sABP, and dABP during the 70° tilt throughout the course of the dive. The dABP changed insignificantly from the control values during the tilt, while sABP decreased and HR increased. This trend remained regardless whether the tilt test was conducted before or after the underwater exercise (Table I). Table I summarizes means and standard errors of supine and tilt values for various dive periods. The resting HR rose ($p < 0.01$) but SV fell ($p < 0.05$) postdive compared to the predive values. The predive postexercise values for CO, TPR were not available due to technical problems. However, the available data (ABP and HR) during this period showed no exercise related differences in response to tilt (Table II).

Cardiovascular index of deconditioning: Although we observed no syncopal episodes with the orthostatic test, CD was shown to exist during the early and mid periods at 31 ATA, and after the dive, because CID increased significantly at ATA (I), ATA (II), and postdive compared to the predive values (Table I).

Hematocrit and plasma volume: The hematocrit value increased ($p < 0.05$) at days 2 and 5 at 31 ATA, indicating hemoconcentration. Urine output increased by 100% from the average predive value of 1.03 L·d^{-1} (15). Since the red blood cell volume had not changed during the dive (17), we estimated that the plasma volume decreased by 8.3

TABLE II. EFFECT OF UNDERWATER EXERCISE ON CIRCULATORY CHANGES DURING A 70° HEAD-UP TILT.

Measurements	Work	Predive	31 ATA (I)	31 ATA (II)	31 ATA (III)	Postdive
Heat rate, b·min^{-1}	B	25 ± 3	22 ± 6	26 ± 3	13 ± 3	22 ± 4
	A	35 ± 9	22 ± 2	20 ± 1	17 ± 3	18 ± 4
Systolic ABP, mm Hg	B	−12 ± 5	−12 ± 1	−13 ± 6	−7 ± 3	−14 ± 2
	A	−15 ± 3	−18 ± 2	−11 ± 3	−17 ± 3*	−12 ± 3
Diastolic ABP, mm Hg	B	−1 ± 6	−10 ± 3	−8 ± 5	−15 ± 4	−9 ± 3
	A	−1 ± 11	−4 ± 5	−2 ± 3	−3 ± 4*	2 ± 5
Mean ABP, mm Hg	B	−6 ± 5	−11 ± 1	−10 ± 4	−10 ± 3	−12 ± 1
	A	−8 ± 7	−11 ± 3	−7 ± 1	−11 ± 1	−6 ± 3
Stroke volume, ml·b^{-1}	B	−32 ± 3	−30 ± 2	−30 ± 8	−26 ± 5	−31 ± 6
	A	NA	−40 ± 4	−31 ± 6	−36 ± 4	−28 ± 3
Cardiac output, L·min^{-1}	B	−16 ± 8	−6 ± 12	−11 ± 11	−14 ± 5	−10 ± 4
	A	NA	−27 ± 3	−20 ± 8	−23 ± 3	−18 ± 3
TPR, mm Hg·L^{-1}·min^{-1}	B	−1 ± 13	0 ± 16	2 ± 10	2 ± 8	0 ± 5
	A	NA	21 ± 2	16 ± 10	18 ± 4	13 ± 2

Values are mean ± SE for 3 measurements in 3 subjects. B = before exercise and A = afer exercise; TPR = total peripheral resistance; 31 ATA (I), (II), and (III) refer to early, mid, and late periods at 31 ATA, respectively; NA = not available; * represents $p < 0.05$ comparing B and A by paired *t*-tests.

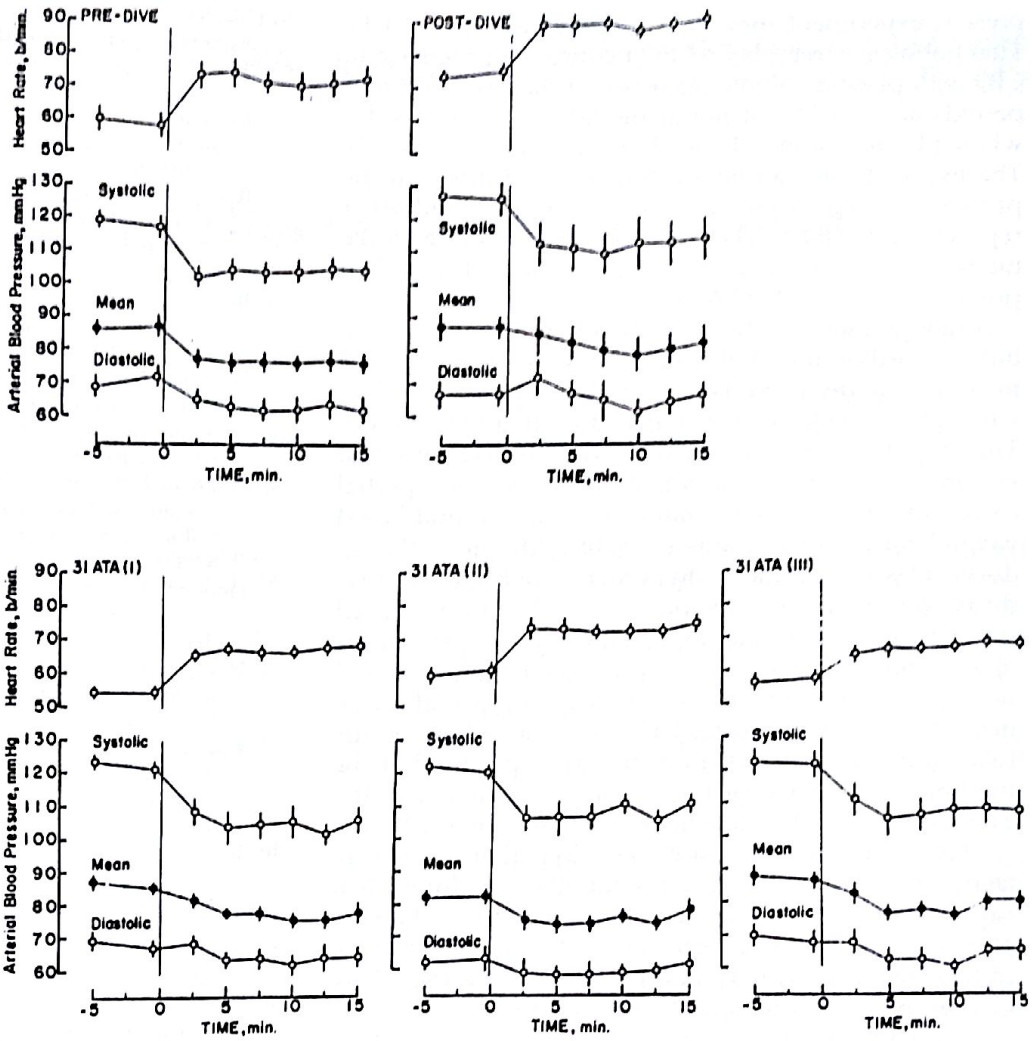

Fig. 2. Circulatory responses to a 70° head-up tilt. Values are mean responses ± SE for 6 tilt tests in 3 subjects. Tilt-induced changes are significantly different from supine values at $p < 0.05$ for all measurements except TPR. The upper panel depicts predive and postdive responses. The lower panel shows early, mid, and late periods of 31 ATA exposure which were represented by 31 ATA (I), 31 ATA (II), and 31 ATA (III), respectively. There were no temporal differences in responses to tilt throughout the dive. Average values for pre-tilt and during the tilt are presented in Table III.

and 10.3% ($p < 0.05$) during these periods (**Table III**). Compared to the predive values, there was no difference in hematocrit values and estimated plasma volume changes at late 31 ATA period (day 7) and postdive.

DISCUSSION

The present study showed an increase in CID at early and mid periods of 31 ATA exposure in agreement with a previous study (1). In addition, the resting HR was elevated and SV lowered following the dive. These results strongly suggest the existence of CD upon exposure to hyperbaric environment. In an in-house report, Holthaus (6) listed incidence of postdive vagovasal syncope, postdive circulatory instability, and vagovasal presyncope during i.v. puncture at the German Underwater Simulator (GUSI) dive series I, III, VI, and IX. Results of a recent study by Mateev et al. (12) also support the occurrence of CD following a short exposure to hyperbaric conditions.

TABLE III. CHANGES IN HEMATOCRIT AND PLASMA VOLUME DURING EXPOSURE TO 31 ATA.

Periods	Hematocrit (%)	ΔPV (%)
Predive	45.0 ± 3.0	0.0
31 ATA (day 2)	47.3 ± 2.9*	−8.3 ± 1.6*
31 ATA (day 4)	47.9 ± 2.1*	−10.3 ± 3.7*
31 ATA (day 7)	46.2 ± 2.4	−4.0 ± 2.3
Postdive	44.5 ± 3.3	3.1 ± 4.9

Values are means ± SE. ΔPV is percent change in plasma volume from predive value.
* $p < 0.05$ compared to predive values.

Classic indicators of CD following a period of inactivity include an elevation of resting HR, a decreased SV, reduced plasma volume, lowered left ventricular diastolic volume, an orthostatic intolerance, and a reduced exercise capacity (16). Although syncope and shortened time to syncope in a passive tilt are positive indications and have been used to define the existence of CD, other indicators of CD may also be used. Among the extensive list of potential indicators reviewed (10), the CID of Bungo and Johnson (4) appears suitable. The reason is that the stability of mABP during a tilt depends on the mobilization of cardiac and vascular actions to prevent syncope. A large change in these parameters reduces functional reserve and induces instability in the cardiovascular system. Therefore, indices that include antihypotentive factors, such as tachycardia and vasoconstriction (elevated dABP and reduced decrease in sABP) could be used. Among them, CID is useful and simple to obtain.

The rapidity by which the CD occurs upon exposure to a hyperbaric environment rules out the prolonged inactivity as the cause (1,12). Hypovolemia observed in the

present experiment may be a major cause for the CD. This notion is strengthened by a concurrent increase in CID with plasma volume decreases at the early and mid periods at 31 ATA but not at the late stage of 31 ATA where plasma volume changed insignificantly (Table III). The hypovolemia can be attributed to a diuresis in the present experiment published elsewhere (15), and other reports (7,8,13,18,19). The close relationship between the increased CID and hypovolemia was also observed in previous dives to 31 ATA (1,19).

Although there is little doubt that hypovolemia contributes to orthostatic intolerance (2), some other factor(s) must also be involved, because CID increased postdive when plasma volume had returned to the predive level. This hypothesis is supported by the observations that volume-to-volume replacement achieves only partial correction of orthostatic intolerance in ground-based weightlessness simulations (3,5). In addition to the reduced plasma volume, at hyperbaric environment a reduced cardiovascular responsiveness due to decreased sympathetic activity may have played a role (1). On the other hand, CD may follow a prolonged confinement (3 weeks in the present study) and reduced physical activities. The inactivity-induced CD is clear, which occurs following bed rest and microgravity exposure, but the mechanism responsible for is not yet understood (16). The hyperbaric CD warrants further investigation.

Plasma volume depletion upon hyperbaric exposure shares a common mechanism with other conditions that cause central hypervolemia and diuresis (10). CD occurs after a space sojourn, head-out water immersion (6 h in thermoneutral water), and prolonged bed rest, where central hypervolemia and diuresis also prevail.

Circulatory responses to the 70° tilt in the present experiment differed from that of 90° tilt used in our previous study (1). Vasoconstriction (judged from TPR, and changes in dABP) was smaller in the 70°-tilt compared to the 90°-tilt, however changes in HR, SV, and CO were similar in both studies. Metalon and Farhi (11) have demonstrated the indifference in cardiovascular responses to a tilt between 60° and 90°. The smaller vasoconstrictor reflex during tilt in the present study corresponded to the unchanged dABP as opposed to an increase in our previous dive (90° tilt). We have no apparent explanation for the difference at present.

In summary, the results of the present study showed an elevation of CID, indicating CD, at the early phase of hyperbaric exposure and a persisted CD after the dive, confirming the existence of a hyperbaric-induced CD. Mechanisms underlying CD are not well understood. However, the acute hyperbaric CD upon hyperbaric exposure involves hypovolemia, and the reduced physical activity over a prolonged period may account for the CD that we observed postdive.

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