Doppler-echocardiography study of cardiac function during a 36 atm (3,650 kPa) human dive

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Lafay V, Boussuges A, Ambrosi P, Barthelemy P, Frances Y, Gardette B, Jammes Y. Doppler-echocardiographic study of cardiac function during a 36 atm (3,650 kPa) human dive. Undersea Hyperbaric Med 1997; 24(2):67–71.—To determine the influence of a saturation dive on cardiac function, Doppler-echocardiographic measurements were compared at sea level and during a 36 atm (3,650 kPa) He–O_2 dive (gas density: 7 g/1iter) in four healthy men. Left ventricular systolic function was studied from time–motion measurements. Transmitral flow (E:A ratio) and isovolumetric relaxation time were used to assess left ventricular diastolic function. Cardiac output was derived from systolic pulmonary and aortic valvular flows. Cardiac output decreased 4.4 ± 0.8 vs. 5.9 ± 1.2 liter/min at sea level) whereas stroke volume, left ventricular ejection fraction, atria and ventricular diameters remained unchanged. Thus, the decrease in cardiac output was attributed to bradycardia (56 ± 8 vs. 73 ± 9 beats/min at sea level) which probably resulted from the slight hyperoxia (PI_{O_2}, 0.4 atm). We found no evidence of left ventricular diastolic dysfunction. nor did we find valvular regurgitation or pulmonary hypertension. We conclude that Doppler-echocardiography can be used safely to investigate cardiac function during human saturation dives. Our results suggest that a 36 atm He–O_2 dive does not modify cardiac or systolic and diastolic function except for a slight decrease in cardiac output correlated to bradycardia.

echocardiography, Doppler, deep diving, saturation dive, cardiac function

Few hemodynamic data have been reported during deep saturation dives. They mainly concerned animal models (rat) (1–6) and used the radioactive-labeled microspheres method which does not give any information about dynamic changes throughout the dive. Two recent studies (7,8) have used the Doppler method to evaluate the variations of aortic and regional blood flows. The aforementioned observations, during normoxic dives (4,5), have shown no change in cardiac output, systemic blood pressure or peripheral resistance, and that myocardial perfusion tended to increase. Some authors have shown an increase in myocardium contractility (9) which was not antagonized by beta-blockers (6), and others have reported a positive correlation between the myocardium and the gas density (1). By contrast, other investigators reported an alteration of cardiac excitation–contraction coupling during hyperbaric exposure (10). During hyperoxic dives (2,3,5), a decrease in cardiac output and an increase in systemic resistance were coincident to bradycardia. Moreover, increased hydrostatic pressure involved bradycardia (11) which could be antagonized by nitrogen (12), and induced alterations of myocardial conduction (11,13) or arrhythmias (14).

Most hemodynamic studies in humans consisted of measurements of cardiac output using the thoracic bioimpedance method or the thermodilution method during stays at low pressure (3 atm) (15–17). They showed a stability of the stroke volume and the systemic resistance under normoxic conditions (15); furthermore, a decrease in cardiac output and an increase in systemic resistance was found under hyperoxia (16,17). Observations based on bioimpedance measurements during deep, human saturation dives (18–20), including breathing a slight hyperoxic gas mixture (PI_{O_2} = 0.4 atm), have shown no change in the systolic stroke volume. However, this method does not measure the changes in ventricular diameters and left ventricular shortening fraction, which are good markers of cardiac contractility. In addition, changes in diastolic cardiac function, resulting from loaded ventilation, have never been studied. Doppler-echocardiography (DE) is a useful method to describe both heart anatomy and cardiac function. Except for bubble detection (21,22), it has not been used for evaluation of cardiac function during human deep dives.

The aim of the present human study was to estimate both systolic and diastolic cardiac functions using DE method during a saturation dive at 36 atm (3,650 kPa) using He–O_2 gas mixture, with incursions in He–H_2–O_2 mixture.

METHODS

This experiment was conducted during the "helium in–hydrogen out" human dive (11 January to 4 February

1994) which took place in the hyperbaric center of COMEX in Marseilles. Four healthy professional divers (D1 to D4), 26, 29, 30, and 28 yr old, respectively, volunteered to participate. The protocol was approved by the ethical committee of the establishment.

Diving protocol was a 3-day compression in $He–O_2$, followed by a 9-day bottom stay at 36 atm in $He–O_2$, then a 12- day decompression. Gas density varied from 1.18 $g/liter_{ATPS}$ (sea level) to 7 $g/liter_{ATPS}$ (36 atm). During the stay at maximum pressure, eight $H_2–He–O_2$ incursions were performed. The chamber temperature was maintained at an optimum level of 31°C. PI_{O_2} value was 0.4 atm throughout the experiment.

Doppler-echocardiography was performed in each diver at Day 1 (T1) and Day 9 (T2) at maximum pressure. Data were compared to sea-level measurements performed before (C1) and after (C2) the dive. A Diasonics CFM 750 echocardiograph was localized out of the diving chamber and connected to the 2.5 MHZ probe through the hull. The transducer was positioned by the divers themselves who were trained in this method by cardiologists for 1 wk. They could adjust the position of the DE probe by looking at the DE pictures through a chamber porthole. The pictures were displayed on a video-screen placed outside the chamber. A cardiologist supervised examinations.

According to clinical practice, examinations were performed at rest in left lateral decubitus (long and short axis views) and supine (apical and subcostal views). The following data were available:

1. Time–motion echography (TM):
   *Diameters of left atrium and aorta, systolic (LVDs) and diastolic (LVDd) diameters of left ventricle. Stroke volume (Sv), left ventricular shortening (LVSF), and ejection (LVEF) fractions were calculated as follows:
   - $SV = LVDd^3 – LVDs^3$
   - $LVSF = (LVDd – LVDs) / LVDd$
   - $LVEF = (LVDd^3 – LVDs^3) / LVDd^3$

   * systolic and diastolic thickness of left ventricle walls: left ventricle contractility was calculated from these parameters.
2. Bidimensional echography, estimating fractional ventricle motion.
3. Doppler and color Doppler, which allowed us to assess valvular regurgitation and to estimate pulmonary artery pressure and left and right cardiac output (CO) from aortic and pulmonary artery flows, respectively.
   CO = Heart rate × annular area × integral value of pulsed Doppler signal. Moreover, transmitral flow allowed to obtain:
   • proto- and end-diastolic peak flows (peak E and peak A, respectively): value of peak E to peak A ratio less than or equal to 1 suggests the existence of altered diastolic function,
   • isovolumetric relaxation time (IVRT) (i.e. the time between closing of aortic valve and opening of mitral valve): increased IVRT indicates alterations in diastolic function.

All aforementioned measurements were ECG-triggered (frontal leads).

Means and standard deviations of TM values were calculated, but due to the small number of subjects it was not possible to perform a statistical comparison of these data.

RESULTS

- Heart rate (Fig 1*A*) decreased at T1 in all divers (56 + 8 beats/min vs. 73 + 9 beats/min at sea level). Bradycardia was less marked but persisted at T2 (61 + 8 bpm).
- Left ventricle diameters, wall thickness, left atrial and aortic diameters did not change (Table 1).
- Sea level measurements of LVSF (Fig. 2*A*) as well as left ventricle contractility were in normal range and did not change at T1 and T2.
- Left and right cardiac outputs (Fig. 1*B*) were equivalent at sea level and both decreased at T1 in the four subjects (mean: 4.4 + 0.8 liter/min) and in three at T2 (mean: 5.1 + 1 liter/min) compared to sea level (mean: 5.9 + 1 liter/min). Thus, the decrease in cardiac output was less marked at T2 (−14%) than T1 (−25%) and was proportional to HR variations.
- Stroke volume did not change (Fig. 2*B*).
- Diastolic function indexes: peak E/peak A ratio (Fig. 2*D*) remained in normal value range (>1), whereas a slight increase in IRVT was measured at T1 and T2 in all subjects (Fig. 2*C*).
- No significant tricuspid regurgitation was found.
- No valvular dysfunction was observed.

DISCUSSION

This study shows that reproducible echocardiographic measurements can be obtained during deep experimental human dives. The divers were able to perform adequate examinations after 1 wk of training.

One methodological limitation is the possibility of echocardiograph dysfunction under high pressure. However, if the transducer was exposed to high pressure, the echocardiograph remained at sea-level pressure. Moreover, no alterations were observed in echographic pictures or Doppler signal. Another limitation was that the small

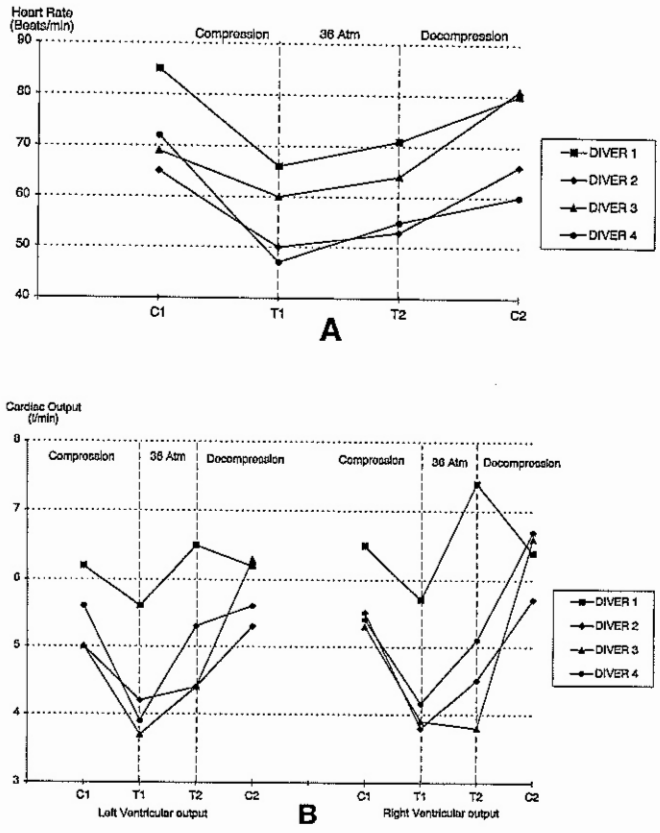

FIG. 1—Individual values: *A*, heart rate; *B*, left and right ventricular outputs. C1 and C2 = control data before and after the dive; T1 and T2 = test data at 36 atm on Days 1 and 9 of stay.

number of subjects which did not allow any statistical analysis. In consequence, our data are purely descriptive.

Our study confirms that HR decreases during human dives (23–25). This effect was already present on Day 1 of compression and was slightly accentuated when divers reached 36 atm. Then, HR changes adapted to new conditions, and the decrease in HR was only 16% after 9 days of stay at maximum depth. This time course of HR changes during a deep dive agrees with previous data (19,25–28), which showed that slowing of HR was mainly due to hyperoxia (PI_{O_2} = 0.4 atm) and ambient pressure change, and tended to disappear throughout the dive.

Cardiac output decreased during the dive in the four subjects. Doppler proved to be highly reproducible for ventricular output determination, and we found in each subject equivalent values for right and left ventricular output. Similarly, several authors using the bio-impedance method found a decrease of cardiac output during human or animal dives (3,17-19). However this was not confirmed by Smith et al.(20). The reasons for these discrepancies are not clear. Inasmuch as the stroke volume remained stable during the dive, the decrease in cardiac output with an unchanged stroke volume suggests that this change may only result from the reduced HR. This hypothesis has been proposed by Flynn et al. (18) and Ohta et al. (19), and unchanged systolic function does not corroborate previous results (1,6,9). The explanation may be the large differences between experimental conditions. The aforementioned studies were done in in vitro conditions or during acute animal experiments involving very short compression phases, i.e., in circumstances very far from a human saturation dive.

Echo-Doppler seems to be an interesting tool to describe heart–lung interactions during deep dives. Johnston et al. (29) demonstrated that increasing the intrathoracic expiratory pressure could impair the left ventricular filling. However, increasing the strength of diaphragmatic contraction in response to dense gas breathing enhanced the intrathoracic pressure toward negative value (30). This reproduces the consequences of inspiratory threshold load breathing, which facilitates the heart filling.

No significant changes in left atrial and ventricular diameters were found. This corroborates previous animal observations, which showed that the end-diastolic left ventricular pressure is not modified in rats during a 30-atm dive (6). Thus, breathing a high-density gas mixture did not involve any marked variation in left ventricular diastolic function, except an increase in isovolumetric relaxation time, probably due to bradycardia.

During this dive there was no echocardiographic evidence of pulmonary hypertension, as usually indicated by

Table 1: Mean and Standard Values, in Millimeters

	C1, 0 msw	T1, 350 msw	T2, 350 msw	C2, 0 msw
Aortic	32.7±3.3	29.2±4.5	32.3±3.3	28.8±3.1
Left atrial	31.7±3.0	32.8±5.8	31.2±5.3	27.8±2.5
Diastolic left ventricular diameters	49.4±3.7	50.1±3.5	53.1±4.1	49.3±3.5
Systolic left ventricular diameters	35.1±1.8	33.5±2.8	33.7±1.3	31.0±2.1
Diastolic thickness of the interventricular septum	8.7±1.7	8.5±0.6	9.0±1.1	8.9±1.5
Systolic thickness of the interventricular septum	12.3±1.5	12.2±1.0	12.3±0.9	12.1±0.6
Diastolic thickness of the left posterior wall	9.5±0.9	1.0±0.8	9.0±1.0	8.9±1.1
Systolic thickness of the left posterior wall	13.3±1.6	13.9±0.6	12.0±0.8	13.7±1.0

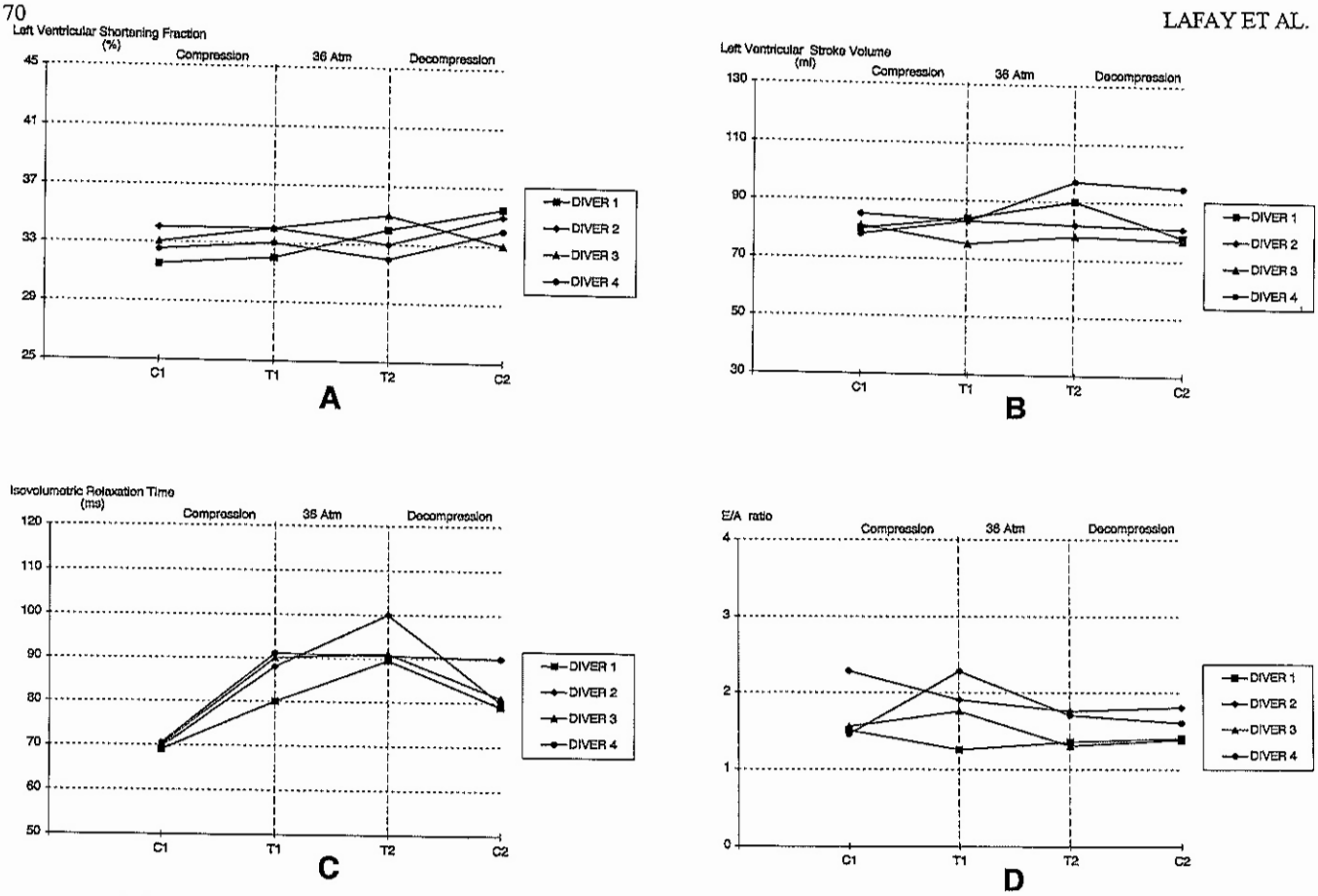

FIG. 2—Individual values of the left ventricular systolic and diastolic functions: *A*, left ventricular shortening fraction; *B*, left ventricular stroke volume; *C*, isovolumetric relaxation time; *D*, transmitral flow E:A ratio. C1 and C2 = control data before and after the dive; T1 and T2 = test data at 36 atm on Days 1 and 9 of stay.

dilation of the right ventricle or the supra-hepatic veins, high-velocity tricuspid regurgitation, or abnormalities of septal wall motion. However, this does not rule out the possibility of a moderate elevation of pulmonary arterial pressure (PAP). Previous human studies reported a slight increase (+11%) of PAP during stays at relatively low pressure (3 atm) (15), whereas, at the same pressure, PAP decreased (−23%) under hyperoxic conditions (16). No hemodynamic data on PAP changes during deep saturation dives are available, and DE seems to be the sole non-invasive method to assess pulmonary arterial pressure during experimental dives. In a previous saturation dive at 1 atm (25), we reported a rightward shift of the electrocardiographic P-wave axis with increased P-wave amplitude, suggesting an electric overload of the right atrium. However, this ECG variation could be observed in the absence of notable elevated pulmonary pressure.

In conclusion, DE can be used to assess cardiovascular changes during experimental human dives. It can be considered a good tool to evaluate heart–lung interactions due to high-density gas breathing during hyperbaric stays.

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REFERENCES

1. Bergø GW, Ask JA, Tyssebotn I. Breathing gas density influences myocardial contractility. Underwater and hyperbaric physiology IX. Proceedings of the ninth symposium on underwater physiology. In: Bove AA, Bachrach AJ, Greenbaum LJ Jr, eds. Bethesda, MD: Undersea and Hyperbaric Medical Society; 1987:471–476.
2. Bergø GW, Risberg J, Tyssebotn I. Effect of 5 bar oxygen cardiac output and organ blood flow in conscious rats. Undersea Biomed Res 1989; 1:457–470.
3. Hordnes C, Tyssebotn I. Effect of high ambient pressure and oxygen tension on organ blood flow in conscious trained rats. Undersea Biomed Res 1985; 12:115–128.
4. Risberg J, Tyssebotn I. Hyperbaric exposure to a 5 ATA He-N_2O_2 atmosphere affects the cardiac function and organ blood flow distribution in awake trained rats. Undersea Biomed Res 1986; 13:77–90.
5. Risberg J, Bergø GW, Hordness C, Tyssebotn I. Distribution of cardiac output in awake rats during exposure to 5 bar. Undersea Biomed. Res 1990; 17:503514
6. Stuhr LE, Ask JA, Tyssebotn I. Cardiovascular changes in anesthetized rats during exposure to 30 bar. Undersea Biomed. Res 1990; 17:383–393.
7. Guèrrero F, Joulia F, Barthelemy P, Burnet H. Etude des effets des hautes pressions d'helium-oxygène (91 ATA) sur les débits circu-

latoires régionaux. Arch Physiol Biochem 1990; 98:A426.
8. Slørdahl SA, Vik A, Jenssen BM, Brubakk AO. Aortic blood flow Doppler echocardiography in pigs at 5 bar ambient pressure and after decompression. Proceeding of the XVIIth annual meeting on diving and hyperbaric medicine. Crete, Greece: EUBS, 1991:163-168
9. Ask JA, Tyssebotn I. Positive inotropic effect on the rat atrial myocardium compressed to 5, 10 and 30 bar. Acta Physiol Scand 1988; 134:277–283.
10. Doubt TJ, Evans DE. Hyperbaric exposures alter cardiac excitation–contraction coupling. Undersea Biomed Res 1982; 9:131–145.
11. Örnhagen HCH, Hogan PM. Hydrostatic pressure and mammalian cardiac pacemaker function. Undersea Biomed Res 1977; 4:347–358.
12. Örnhagen HCH. Influence of nitrous oxide, nitrogen, neon and helium on the beating frequency of the mouse sinus mode at high pressure. Undersea Biomed Res 1979; 6:27–39.
13. Doubt TJ, Hogan PM. Combined influence of temperature and hydrostatic pressure on cardiac conduction. Undersea Biomed Res 1980; 7:211–223.
14. Doubt TJ, Hogan PM. Combined effect of beating rate and hydrostatic pressure on excitation in cardiac muscle. Undersea Biomed Res 1982; 9:241–253.
15. Neviere R, Mathieu D, Wattel F. Modifications hemodynamiques en milieu hyperbare. Arch Physiol Biochem 1990; 98:A426.
16. Bouachour G, Tirot P, Gouello JP, Dubourg F, Harry P, Alquier P. Pulmonary artery catheter use in the multiplace hyperbaric chamber. Cardiovascular changes at 3 ATA pressure. A case report. Proceeding of the XVIIth annual meeting on diving and hyperbaric medicine. Crete, Greece: EUBS, 1991:279–284.
17. Pelaia P, Rocco M, Malpieri MR, Gianni G, Ochigrossi F, Spinelli V. Haemodynamic changes during hyperbaric oxygen therapy. Proceeding of the XVIIth annual meeting on diving and hyperbaric medicine. Crete, Greece: EUBS, 1991:255–262.
18. Flynn ET, Doubt TJ. Cardiovascular studies during Hydra V. In: hydrogen as a diving gas. Bethesda, MD: Undersea Medical Society, 1987:199–209.
19. Ohta Y, Arita H, Nakayama H, et al. Cardiopulmonary functions and maximal aerobic power during a 14-day saturation dive at 31 ATA (Seadragon IV). In: Bachrach AJ, Matzen MM, eds. Underwater Physiology VII. Proceedings of the seventh symposium on underwater physiology. Bethesda, MD: Undersea Medical Society, 1981: 209–221.
20. Smith RM, Hong SK, Dressendorfer RH, Dwyer HJ, Hayashi E, Yelverton C. Hana-Kai II: a 17-day dry saturation dive at 18,6 ATA. IV. Cardiopulmonary functions. Undersea Biomed Res 1977; 4:267–281.
21. Eftdal O, Kotens S, Brubbak AO. Bubble grading in ultrasonic images. Undersea Hyperbaric Med. 1993; 20(suppl): 79A.
22. Boussuges, A, Lafay V, Ambrosi P, Gardette B. 2D Echocardiographic and continuous wave Doppler detections of circulating bubbles produced by isobaric counterdiffusion. In: Sipinen S, Leinio M, eds. Proceedings of the XXIst annual meeting on diving and hyperbaric medicine. Helsinki, Finland: EUBS, 1995:175–179.
23. Gosovic SM, Radovic AI. Frequency and regulation of heart rate during open-sea saturation diving. In: Bachrach AJ, Matzen MM, eds. Underwater Physiology VII. Proceedings of the seventh symposium on underwater physiology. Bethesda, MD: Undersea Medical Society. Bethesda, MD: Undersea Medical Society, 1981:209–221.
24. Salzano J, Overfield EM, Rausch DC, et al. Arterial blood gases, heart rate, and gas exchange during rest and exercise in men saturated at a simulated seawater depth of 1000 feet. In: Lambertsen CJ, ed. Underwater Physiology. Proceedings of the fourth symposium on underwater physiology. Bethesda, MD: Undersea Medical Society; 1971: 347–356.
25. Lafay V, Barthelemy P, Comet B, Frances Y, Jarnmes Y. ECG changes during the human experimental dive HYDRA 10 (7lATM/7200kPa) Undersea Hyperbaric Med 1995; 22:51–60.
26. Eckendorff RG, Knight DR Cardiac arrhythmias and heart rate changes in prolonged hyperbaric air exposures. Undersea Biomed Res 1984; 11:355–367.
27. Wilson JM, Kligfield PD, Adams GM, Harvey C, Scheaffer KE. Human ECG changes during prolonged hyperbaric exposures breathing N_2–O_2 mixtures. J Appl Physiol 1977; 42:614–623.
28. Shida KK, Lin YC. Contribution of environmental factors in development of hyperbaric bradycardia. J Appl Physiol 1981; 50:731–735.
29. Johnston WE, Viten-Johansen J, Santamore WP, Case LD, Little WC. Mechanism of reduced cardiac output during positive end-expiratory pressure in the dog. Am Rev Respir Dis 1989; 1 40:1257–1264.
30. Lenoir P, Jammes Y, Giry P, et al. Electromyographic study of respiratory muscles during human diving at 46 ATA. Undersea Biomed Res 1990; 17:121–137.