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Cardiac Response to Head-Out Water Immersion in Man

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Head-out water immersion induces marked increase in the cardiac stroke volume. The present study was undertaken to characterize the stroke volume change by analyzing the aortic blood flow and left ventricular systolic time intervals. Ten men rested on a siting position in the air and in the water at 34.5°C for 30 min each. Their stroke volume, heart rate, ventricular systolic time intervals, and aortic blood flow indices were assessed by impedance cardiography. During immersion, the stroke volume increased 56%, with a slight (4%) decrease in heart rate, thus cardiac output increased ~50%. The slight increase in R-R interval was due to an equivalent increase in the systolic and diastolic time intervals. The ventricular ejection time was 20% increased, and this was mainly due to a decrease in pre-ejection period (28%). The mean arterial pressure increased 5 mmHg, indicating that the cardiac afterload was slightly elevated by immersion. The left ventricular end-diastolic volume index increased 24%, indicating that the cardiac preload was markedly elevated during immersion. The mean velocity and the indices of peak velocity and peak acceleration of aortic blood flow were all increased by ~30%, indicating that the left ventricular contractile force was enhanced by immersion. These results suggest that the increase in stroke volume during immersion is characterized by an increase in ventricular ejection time and aortic blood flow velocity, which may be primarily attributed to the increased cardiac preload and the muscle length-dependent increase in myocardial contractile force.

Key Words: Water immersion, Stroke volume, Aortic blood flow, Ventricular ejection time, Pre-ejection period

INTRODUCTION

Head-out immersion (HOI) in thermoneutral water (34~35°C) induces an increase in stroke volume by 35~85% in humans (Arborelius et al, 1972; Löllgen et al, 1981; Lin, 1984; Christie et al, 1990; Stadeager et al, 1992; Nakamitsu et al, 1994; Miwa et al, 1997). This increase in stroke volume would be attributed to an increase in aortic blood flow velocity and/or ventricular ejection time, since the stroke volume corresponds to the aortic blood flow during the period of ventricular ejection. However, changes in these parameters during HOI have not been reported.

The aortic blood flow velocity changes with the cardiac preload and the myocardial contractility (Stein & Sabbah, 1976), whereas the ventricular ejection time is affected by the cardiac preload and afterload (Talley et al, 1971) and the heart rate (Wiessler et al, 1961; Spodick & Kumar, 1968). Numerous studies suggest that the cardiac preload increases during HOI. It has been shown that the right atrial pressure rises by 10~18 mmHg (Arborelius et al, 1972; Lin, 1984; Christie et al, 1990; Gabrielsen et al, 1993) and heart volume increases by 27~44% (Lange et al, 1974; Risch et al, 1978). The arterial blood pressure has been observed to remain unchanged, to increase slightly, or to decrease slightly during immersion (Lin, 1984; Christie et al, 1990; Gabrielsen et al, 1993), suggesting that the cardiac afterload changes a little. Many investigators reported a decrease (10~20%) in heart rate during HOI (Risch et al, 1978; Löllgen et al, 1981; Gabrielsen et al, 1993), although some investigators observed no changes in heart rate (Arborelius

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et al, 1972; Christie et al, 1990). Therefore, on the basis of increased preload and decreased heart rate, one would predict that the ventricular ejection time increases during water immersion. The myocardial contractility would be decreased with water immersion, because the plasma norepinephrine level (Krishna et al, 1983; Mano et al, 1985; O'Hare et al, 1986; Connelly et al, 1990; Nakamitsu et al, 1994) and muscle sympathetic nerve activity (Mano et al, 1985) in man were decreased with water immersion. Furthermore, Christie et al (1990) observed that the left ventricular end-systolic volume increased during water immersion. Since an increase in afterload does not cause the left ventricular end-systolic volume to increase in normal subjects, the so-called Anrep effect (Braunwald et al, 1984), they suggested that cardiac contractility might indeed be lower in water. Since the increase in cardiac preload and the decrease in myocardial contractility would affect the aortic blood flow velocity in the opposite direction, it is uncertain whether the aortic blood flow velocity remains unchanged during water immersion.

The present study was undertaken to evaluate changes in cardiac performance, especially the increase in stroke volume, by assessing the aortic blood flow velocity, ventricular ejection time and pre-ejection period corresponding to the isovolumetric contraction period by impedance cardiography.

METHODS

Subjects

Ten healthy males without a history of cardiovascular disease were recruited as the subjects. Their age was 22.6±0.4 (SE) yr, height was 172.7±1.7 cm, weight was 68.6±2.1 kg, and body surface area, estimated by the DuBois's formula (DuBois & DuBois, 1916), was 1.815±0.036 m^2. Subjects were asked to avoid strenuous physical activity and alcohol drinking, and to maintain a regular diet from the preceding day of the experiment. Permission for the study was obtained from each subject after a detailed explanation of the procedure and potential complications. No complications occurred during the experiments.

Experimental protocol

The subjects wearing swimming trunks were instrumented for impedance cardiography (Fig. 1). The skin was cleansed with alcohol and eight Ag/AgCl spot surface electrodes (Quik-Trace stress testing electrode, Quinton Instrument Co., Washington) were placed on the right and left sides of the neck and thorax in the mid-axillary line. Of the two twin inner electrodes, one pair (electrode number 2 in Fig. 1) was placed around the base of the neck and the other (electrode number 3) at the level of the xiphosternal point. Two twin outer electrodes (electrode numbers 1 and 4) were placed on the positions 5 cm apart (outwardly) from the inner electrodes. The electrodes were covered with surgical tape (Fixomull stretch, No. 2037, Beiersdorf AG, Hamburg, Germany) to prevent detachment from the skin. Vaseline and several sheets of surgical tape were applied on the spot electrodes to prevent wetting during immersion. The electrode leads were connected to the impedance cardiograph (NCCOM3-R7, BoMed Medical Manufacturing, Irvine, CA) with a "slow mode" setting. The built-in algorithm in the instrument analyzed the input signals to the NCCOM3-R7. The subject was outfitted with a blood pressure cuff on the left arm.

After instrumentation, the subject sat quietly on a stool in air at 26~27°C for 30 min and were immersed up to the neck in a circulating water of 34.5°C (thermoneutral water temperature). The subject rested on a stool immersed for 30 min. The impedance cardiograms, heart rate and arterial blood pressures were monitored at appropriate intervals. The NCCOM3-R7 impedance cardiograph provides a continuous display of artifact status. A burst of artifact symbols is displayed with an improper electrode contact, excessive body movement, irregular cardiac rhythm, abnormal respiratory pattern, and tachycardia > 150 beats/min (Introna et al, 1988). To minimize artifact, the subject was asked to avoid excessive trunk movement and breathing activity during experiment. The average values of all measured parameters for every 16 beats accepted by impedance cardiograph were stored in a computer at 2-min intervals.

Measurements

The stroke volume (SV, ml) was calculated on a beat-by-beat basis from the impedance cardiogram (Fig. 1) using the Sramek-Bernstein equation (Berntein, 1986):

$$SV = \delta \cdot (0.17\ Ht)^3/4.2 \cdot VET \cdot (dZ/dt)_{max}/Z_o$$

where $\delta \cdot (0.17\ Ht)^3/4.2$ is the physical volume of

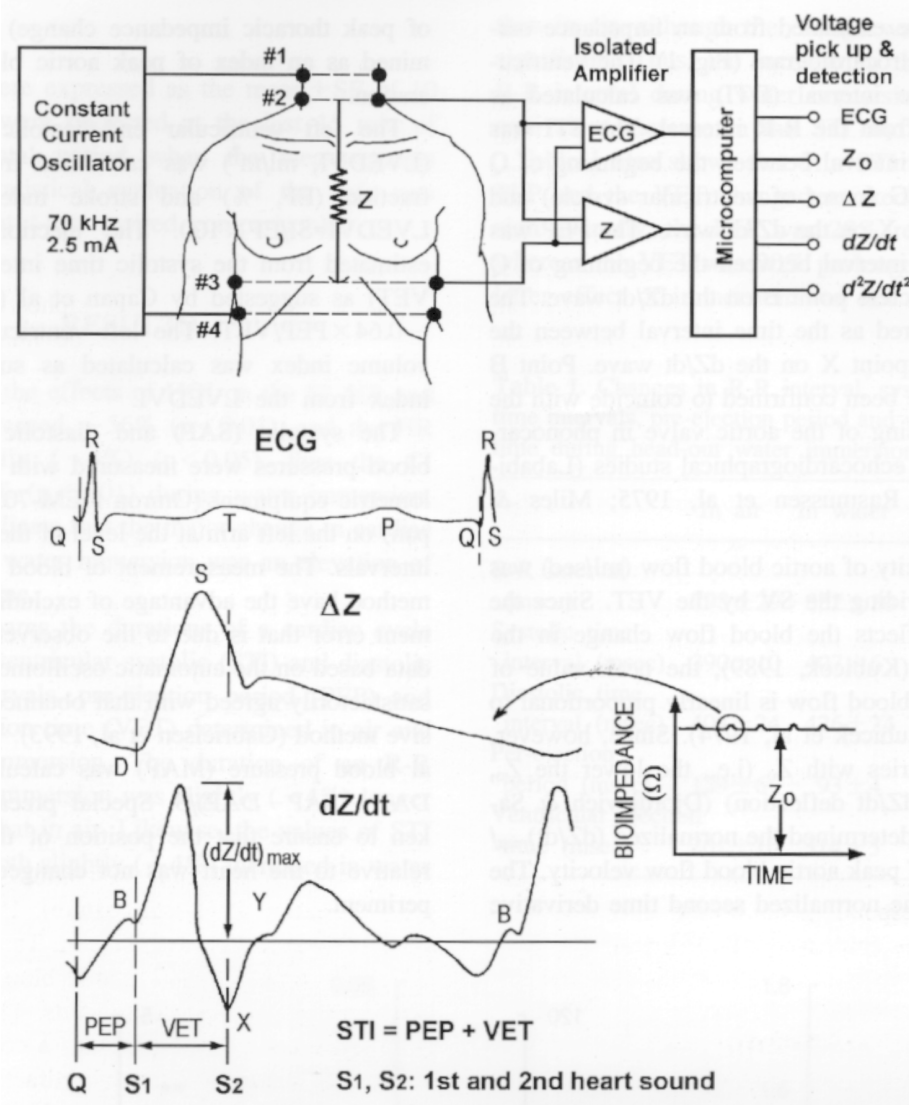

Fig. 1. Diagrams showing the main elements of the impedance cardiograph (*upper panel*) and the timing relations between ECG and dZ/dt wave (*lower panel*). A constant sinusoidal current was applied through the electrodes #1 and #4. The voltage changes reflecting the thoracic impedance changes were measured through the electrodes #2 and #3.

the electrically participating thoracic tissue in cm^3; Ht is the subject's height in cm; δ is weight correction factor, which represents the modified ratio of the subject's measured weight to his ideal weight, for the estimation of electrically participating thoracic tissue volume in Sramek-Bernstein equation; and the δ-calculating formulas was built in the microprocessor of NCCOM3-R7; Z_o is the basal thoracic impedance in ohms; $(dZ/dt)_{max}$ is the maximum rate of change in thoracic impedance in ohm/sec, which is measured as the distance between the zero impedance baseline and the peak value of the dZ/dt wave (the first time derivative of thoracic impedance change, see Fig. 1); VET is the ventricular ejection time in sec. The stroke index (SI, ml/m^2) was calculated by dividing the SV by the body surface area. The heart rate (HR) was analyzed by R-R interval on the electrocardiogram. The cardiac index (CI, $l/min/m^2$) was calculated by multiplying the SI with the HR.

Ventricular systolic time interval (STI), the pre-ejection period (PEP) corresponding to the isovolumetric contraction period and the ventricular ejection

time (VET), were estimated from an impedance cardiogram and electrocardiogram (Fig. 1). The ventricular diastolic time interval (DTI) was calculated as subtracting STI from the R-R interval. The STI was measured as the interval between the beginning of Q wave on the ECG (onset of ventricular systole) and the discrete point X on the dZ/dt wave. The PEP was measured as the interval between the beginning of Q wave and the discrete point B on the dZ/dt wave. The VET was measured as the time interval between the point B and the point X on the dZ/dt wave. Point B and point X have been confirmed to coincide with the opening and closing of the aortic valve in phonocardiographical and echocardiographical studies (Lababidi et al, 1970; Rasmussen et al, 1975; Miles & Gotshall, 1989).

The mean velocity of aortic blood flow (ml/sec) was calculated by dividing the SV by the VET. Since the dZ/dt change reflects the blood flow change in the ascending aorta (Kubicek, 1989), the peak value of ascending aortic blood flow is linearly proportional to the $(dZ/dt)_{max}$ (Kubicek et al, 1974). Since, however, the $(dZ/dt)_{max}$ varies with Z_o (i.e., the lower the Z_o, the smaller the dZ/dt deflection) (Djordjevich & Sadove, 1981), we determined the normalized $(dZ/dt)_{max}/Z_o$ as an index of peak aortic blood flow velocity. The $(d^2Z/dt^2)_{max}/Z_o$ (the normalized second time derivative of peak thoracic impedance change) was also determined as an index of peak aortic blood flow acceleration.

The left ventricular end-diastolic volume index (LVEDVI, ml/m^2) was calculated from the ejection fraction (EF, %) and stroke index (SI, ml/m^2): $LVEDVI=SI/EF\times100$. The ejection fraction was estimated from the systolic time interval ratio (PEP/VET) as suggested by Capan et al (1987): $EF=0.84-0.64\times PEP/VET$. The left ventricular end-systolic volume index was calculated as subtracting stroke index from the LVEDVI.

The systolic (SAP) and diastolic (DAP) arterial blood pressures were measured with automatic oscillometric equipment (Omron HEM-705CP, Tokyo, Japan) on the left arm at the level of the heart at 10-min intervals. The measurement of blood pressure by this method have the advantage of excluding the measurement error that is due to the observer's bias, and the data based on the automatic oscillometric method was satisfactorily agreed with that obtained from the invasive method (Gabrielsen et al, 1993). The mean arterial blood pressure (MAP) was calculated as: $MAP=DAP+(SAP-DAP)/3$. Special precautions were taken to ensure that the position of the pressure cuff relative to the heart was not changed during the experiment.

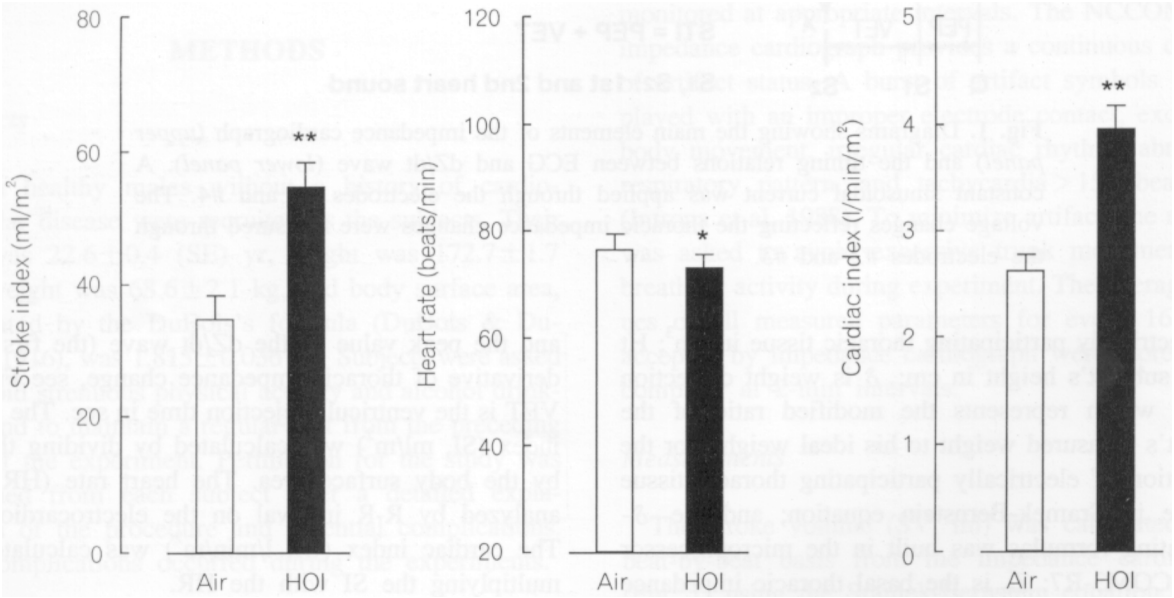

Fig. 2. Stroke index, heart rate and cardiac index in air (Air, *hollow bar*) and during head-out water immersion (HOI, *solid bar*). Data represent the mean+SE of 10 subjects. *$p<0.05$ and **$p<0.01$ compared to that in air.

Statistics

All values were expressed as the mean±SE of 10 subjects. Data were collected at the last 15 min of each experimental period when the measurements were stable. Statistical evaluation of the data was done by Student *t*-test (paired comparison).

RESULTS

Fig. 2 shows the effects of HOI on the SI, HR and CI. The SI increased ~56% ($p<0.01$), and the HR decreased slightly (~4%) ($p<0.05$), thus the CI increased ~50% ($p<0.01$) during water immersion. These results indicate that the major change in cardiac function during water immersion was an elevation of the stroke volume.

Table 1 compares the durations of a cardiac cycle (R-R interval), ventricular systolic (STI) and diastolic (DTI) time intervals, pre-ejection period (PEP) and ventricular ejection time (VET) determined in air and during water immersion. The duration of an R-R interval during immersion was slightly (~4%) longer ($p<0.05$) than that in air. Likewise, the values of STI and DTI were both slightly (~4%) increased in water than in air, although the change was not statistically significant. Thus, the increase in the duration of an R-R interval during water immersion was due to an equivalent increase in the STI and DTI.

Table 1 also shows that in a STI the lengths of the PEP and the VET changed markedly during immersion. Namely, the PEP was 28% reduced ($p<0.01$), whereas the VET was 20% prolonged ($p<0.01$). The latter effect of immersion was not associated with a

Table 1. Changes in R-R interval, systolic and diastolic time intervals, pre-ejection period and ventricular ejection time during head-out water immersion

	In air	In water	Difference	*p*
R-R interval (msec)	798±29	832±30	+34±16	<0.05
Systolic time interval (msec)	390±10	407±6	+17±8	NS
Diastolic time interval (msec)	408±24	426±24	+18±15	NS
Per-ejection period (msec)	129±6	93±4	−35±5	<0.01
Ventricular ejection time (msec)	261±10	314±5	+52±7	<0.01

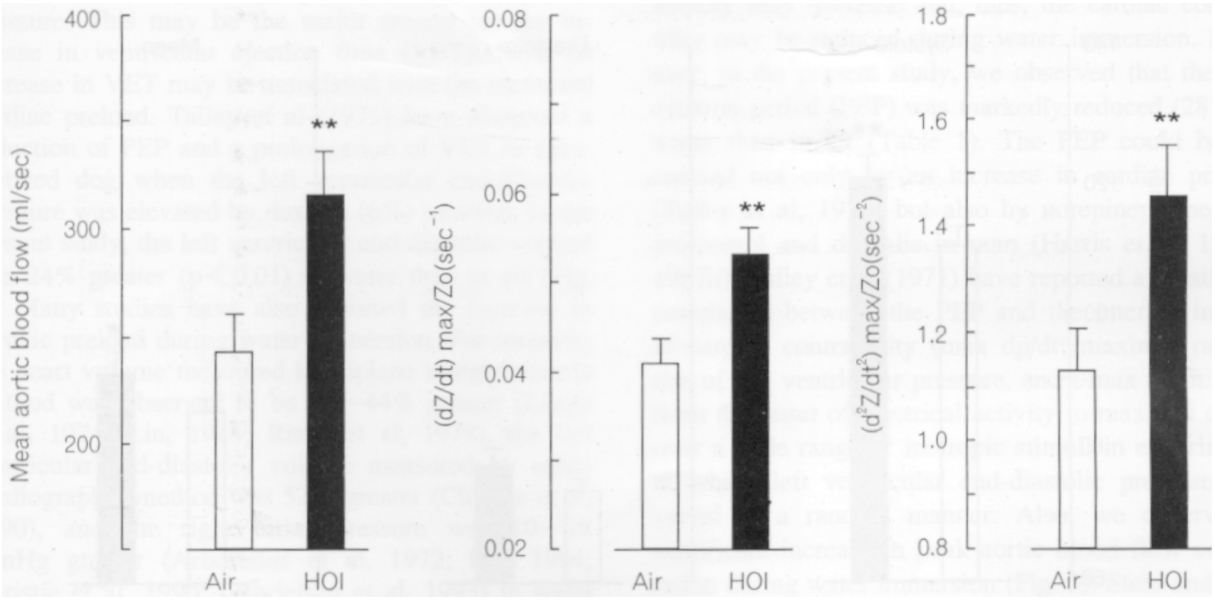

Fig. 3. Mean aortic blood flow velocity, normalized peak aortic blood flow velocity $[(dZ/dt)_{max}/Z_0]$ and normalized peak aortic blood flow acceleration $[(d^2Z/dt^2)_{max}/Z_0]$ in air (Air, *hollow bar*) and during head-out water immersion (HOI, *solid bar*). Data represent the mean+SE of 10 subjects. **$p<0.01$ compared to that in air.

change in cardiac afterload as the arterial blood pressure increased slightly rather than decreased during immersion (see below).

Fig. 3 shows the mean aortic blood flow velocity (calculated by dividing the SV by VET) and the indices of peak aortic blood flow velocity $[(dZ/dt)_{max}/Z_o]$ and peak aortic blood flow acceleration $[(d^2Z/dt^2)_{max}/Z_o]$ determined in air and during immersion. These variables appeared ~30% increased by water immersion ($p<0.01$), indicating that the left ventricular contraction was much more forceful during water immersion than in air.

Fig. 4 shows the left ventricular end-diastolic and end-systolic volume indices in air and during water immersion. The value of the left ventricular end-diastolic volume index was 24% greater in water than in air, indicating that the cardiac preload was increased by water immersion. The left ventricular end-systolic volume index during immersion was similar to that in air.

As shown in Fig. 5, the arterial systolic pressure was ~9% higher ($p<0.01$) during water immersion (121 ± 3 mmHg) than that in air (111 ± 3 mmHg), while the arterial diastolic pressure remained unchanged. Thus, the mean arterial pressure was slightly higher ($p<0.05$) during water immersion (92 ± 3 mmHg) than that in air (86 ± 2 mmHg). These data indicate that the cardiac afterload was only slightly increased during water immersion.

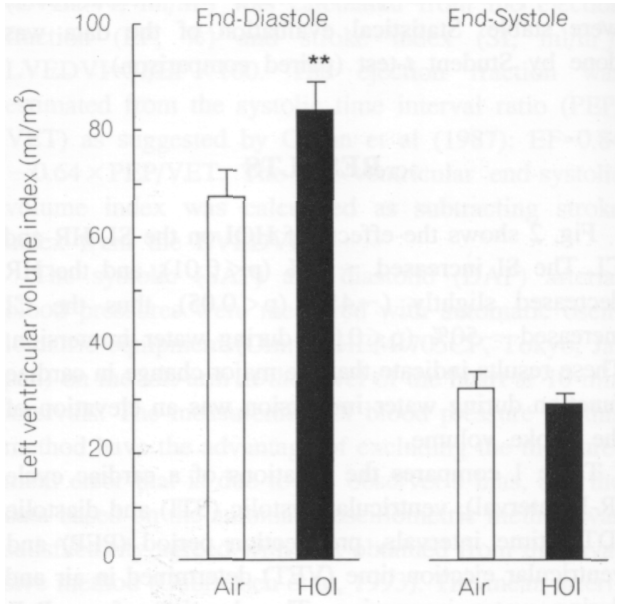

Fig. 4. Left ventricular end-diastolic and end-systolic volume indices in air (Air, *hollow bar*) and during head-out water immersion (HOI, *solid bar*). Data represent the mean + SE of 10 subjects. **$p<0.01$ compared to that in air.

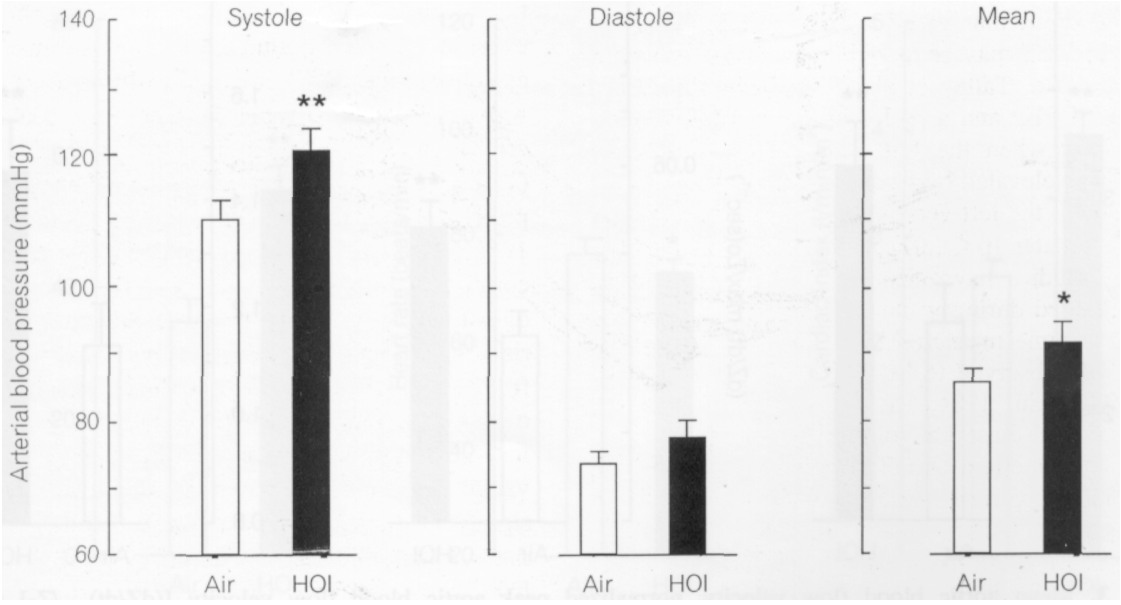

Fig. 5. Systolic, diastolic and mean arterial blood pressures in air (Air, *hollow bar*) and during head-out water immersion (HOI, *solid bar*). Data represent the mean + SE of 10 subjects. *$p<0.05$ and **$p<0.01$ compared to that in air.

DISCUSSION

In the present study, the head-out water immersion in thermoneutral water resulted in a marked (56%) increase in stroke volume measured by NCCOM3-R7 impedance cardiograph. This increase in stroke volume was in accordance with previous observations made by others using a dye-dilution method (35% increase, Arborelius et al, 1972), thermodilution method (70~80% increase, Löllgen et al, 1981; Christie et al, 1990), acetylene rebreathing method (45% increase, Stadeager et al, 1992) and other impedance cardiography (40~85% increase, Nakamitsu et al, 1994; Miwa et al, 1997).

The present study showed that the increase in stroke volume was achieved by an increase in both ventricular ejection time (20% increase, Table 1) and aortic blood flow velocity (30% increase, Fig. 3). These changes were not associated with an alternation of cardiac afterload, as the arterial blood pressure increased rather than decreased during water immersion (Fig. 5).

During water immersion, the R-R interval increased only 4% with no significant change in ventricular systolic time interval (Table 1). However, the pre-ejection period (PEP) was shortened considerably (28%) with water immersion, suggesting that the left ventricular pressure during isovolumetric contraction period reached more rapidly to the aortic diastolic pressure. This may be the major reason for the increase in ventricular ejection time (VET). Such an increase in VET may be associated with the increased cardiac preload. Talley et al (1971) have observed a reduction of PEP and a prolongation of VET in anesthetized dog when the left ventricular end-diastolic pressure was elevated by dextran (6%) infusion. In the present study, the left ventricular end-diastolic volume was 24% greater ($p < 0.01$) in water than in air (Fig. 4). Many studies have also reported the increase in cardiac preload during water immersion. For instance, the heart volume measured by biplane rentgenometric method was observed to be 27~44% greater (Lange et al, 1974; Lin, 1984; Risch et al, 1978), the left ventricular end-diastolic volume measured by echocardiographic method was 52% greater (Christie et al, 1990), and the right atrial pressure was 10~18 mmHg greater (Arborelius et al, 1972; Lin, 1984; Christie et al, 1990; Gabrielsen et al, 1993) in water than in air. Therefore, the increase in ventricular ejection time during water immersion may be a consequence of the increased cardiac preload.

The velocity of aortic blood flow greatly increased with water immersion. The increase was observed not only in the mean and peak velocities but also in the peak acceleration of aortic blood flow (Fig. 3). Stein and Sabbah (1976) observed in anesthetized dog that the peak velocity of aortic blood flow increased when the cardiac preload (left ventricular end-diastolic pressure) and the cardiac contractility were increased. Since the cardiac preload was increased during immersion (Fig. 4), the increased aortic blood flow velocity observed in the present study could also be attributed to an increase in cardiac preload.

It is unlikely that the increased aortic blood flow velocity, induced by the increased cardiac preload, is offset by the immersion-induced change in myocardial contractility, because the cardiac contractility may not decrease during water immersion. In the past, several studies have attempted to assess the sympathetic nervous activity during water immersion, since the sympathetic nerve activity is an effective mean of modifying the myocardial contractility. It has been shown that the plasma norepinephrine level of venous blood is decreased with water immersion (Krishna et al, 1983; Mano et al, 1985; O'Hare et al, 1986; Connelly et al, 1990; Nakamitsu et al, 1994). Also, Mano et al (1985) reported a suppression of muscle sympathetic nerve activity during immersion in a microneurographic study. On the basis of these facts, one may presume that the cardiac sympathetic nervous activity may decrease and, thus, the cardiac contractility may be reduced during water immersion. However, in the present study, we observed that the pre-ejection period (PEP) was markedly reduced (28%) in water than in air (Table 1). The PEP could be decreased not only by an increase in cardiac preload (Talley et al, 1971) but also by norepinephrine, isoproterenol and digitalis in man (Harris et al, 1967a, 1967b). Talley et al (1971) have reported a significant correlation between the PEP and the internal indices of cardiac contractility (max dp/dt: maximal rate of rise of left ventricular pressure, and t-max dp/dt: time from the onset of electrical activity to maximal dp/dt) over a wide range of inotropic stimuli in experiments in which left ventricular end-diastolic pressure was varied in a random manner. Also, we observed a significant increase in peak aortic blood flow acceleration during water immersion (Fig. 3). Stein and Sabbah (1976) reported that the peak aortic blood flow acceleration was markedly increased by a positive inotropic agent isoproterenol in the anesthetized dog, in contrast to a slight increase induced by a preload

increase. Finally, in the present subjects, the left ventricular end-systolic volume tended to decrease during immersion as compared with that in air (Fig. 4), although the cardiac afterload was increased during water immersion (Fig. 5). Since, however, the cardiac preload increased, it is difficult to ascertain whether the myocardial contractility was indeed increased during immersion.

Assuming that the cardiac contractility was not changed with water immersion, the increase in aortic blood flow acceleration and the decrease in pre-ejection period during immersion may be explained by an increase in contractile force of the heart following the increase in ventricular muscle length. The increase in muscle length just prior to contraction would increase contractile potential by increasing the number of cross-bridge formation through the changes in myofilament overlap (Gordon et al, 1966). In addition, the increase in myocardial fiber length is known to increase the Ca^{++}-induced Ca^{++} release from sarcoplasmic reticulum (Fabiato & Fabiato, 1975; Fabiato, 1980), the myofibrillar Ca^{++} sensitivity (Kentish et al, 1986) and the Ca^{++} binding to the fiber bundles (Hofmann & Fuchs, 1988). The cardiac contractile force generated by these mechanisms would be greater than that expected on the basis of myofilament overlap (Fuchs, 1995).

In summary, the present study demonstrated that the increase in stroke volume during water immersion resulted from an increase in aortic blood flow velocity for a longer duration of ventricular ejection. Most of the increase in ventricular ejection time during immersion was caused by a decrease in pre-ejection period. The increase in aortic blood flow velocity and the decrease in pre-ejection period may be primarily attributed to the increased cardiac preload and the attendant increase in myocardial contractile force. Whether the cardiac contractility contributed to the increase in these parameters is not certain.

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