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Circulatory functions during immersion and breath-hold dives in humans

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Lin YC. Circulatory functions during immersion and breath-hold dives in humans. Undersea Biomed Res 1984; 11(2):123–138.—An unusual circulatory state exists in humans immersed in water at or near 35°C. This circulatory state is characterized by a persistent and elevated stroke index (SI) but heart rate (HR) changes little; hence an increased cardiac index (CI) results. In comparison of measurements in air and immersion in water up to the neck level that are based on the weighted averages of 45 subjects from 8 studies, SI increased by 29%, CI increased by 24%, and HR decreased by 6%. Evidence is presented to support the finding that the increase in stroke volume is a result of enhanced preload of the heart, and that alterations in afterload and contractility play an unimportant role in affecting cardiac performance during immersion in thermoneutral water up to the level of the neck. The circulatory state represents hyperperfusion, since there is no concurrent rise in metabolic demand or heat stress. To what extent this preexisting circulatory state affects the cardiovascular responses to breath-hold (BH) diving is in most part unknown. However, the BH bradycardiac responses are more potent in a natural setting than in the laboratory where the breath hold is performed with only face immersion in water. In contrast, in the natural setting, the divers perform BH dives while they are immersed up to the neck in cold water (much lower than 35°C) and are exercising. It is desirable in future studies to compare systematically other aspects of circulatory responses to BH dives while immersed in water and while exercising.

water immersion
human
circulatory state
cardiac output
stroke volume
heart rate
preload
afterload
contractility
breath-hold diving

Head-out immersion in thermoneutral water is a convenient model in simulating the gravity-free state for investigations of fluid volume regulations (1–3), respiratory changes (4–8), cardiac mechanoreceptors, and cardiovascular functions (6, 9–14). Together with bed rest studies, water immersion provides a significant data base to account for physiological changes during and immediately after space travel (15, 16). Although neither bed rest nor water immersion duplicates the condition that exactly matches the condition of weightlessness in space, each serves as a useful partial simulation of the condition of weightlessness. Similarity and dissimilarity between these simulations and weightlessness in space have been discussed (17, 18). There are significant differences that warrant proposal of an alternate model (19, 20). Never-

theless, circulatory responses to water immersion are so prominent and persistent that interest in this model has not faded, though experience in space flights during the last two decades has turned up no major surprises of the circulatory system. Furthermore, lessons learned from water immersion and diving research have been proved valuable, not only in the management of space flights but also in practical and clinical applications (21–23).

Head-out immersion in thermoneutral water produces a variety of prominent changes in cardiovascular, respiratory, and renal functions. In addition, thermal stresses are inevitable when water temperature deviates, even minutely, from neutral conditions. Thermal stress aside, simplistic views have it that submerged tissues encounter 1) hydrostatic compression that reduces the venous capacitance in the lower extremities as well as displaces abdominal contents chestward; 2) an ambient with density resembling human tissues including blood, which thus effectively renders them weightless; and 3) a negative transthoracic pressure of about 14.7 mmHg that also promotes cephalad redistribution of blood. Combination of these conditions results in redistribution of the circulating blood volume toward the upper body. Blood volume in intrathoracic vasculature, including the heart, increases as a consequence. The distended circulatory organs in the thoracic chamber are known 1) to activate cardiac mechanoreceptors that, via the vagal afferents, inform the hypothalamus of hypervolemia, though total blood volume remains constant; 2) to encroach on the pulmonary air space and to alter respiratory mechanics; and 3) to enhance ventricular diastolic filling. Thus the functional expressions of water immersion are diuresis, restrictive ventilation, and sustained increase in stroke output of the heart (1, 3, 22).

This review is restricted to circulatory responses to immersion in thermoneutral water in resting humans. This review and that of Krasney's survey on nonhuman mammals (24) should provide an overview of immersion-induced whole-body hyperfusion. A circulatory condition that did not conform to the concept of autoregulation, i.e., hyperemia without concurrent rise in heat dissipatory or metabolic requirements (25), is a unique physiological state deserving further attention. Consideration of existing literature on circulation changes during immersion prompted these reviews. Other aspects of water immersion are reviewed separately (24, 26–29).

STROKE VOLUME AND CARDIAC OUTPUT

It is known that cardiac output ($\dot{Q}$) rises in humans during head-out immersion in thermoneutral water. Though the notion was conceived early on (30–33), convincing evidence has become available only recently. Arborelius et al. (9) compared male subjects sitting in air (28°C) and head-out immersion in 35°C water. They reported that $\dot{Q}$ in water is 28% greater than that in air. Other reports listed in Table 1 indicate $\dot{Q}$ in water ranged from 88% (34) to 162% (6) of their respective values in air. When water temperature falls below 34°C, heart rate changed either insignificantly (17, 31, 35, 36) or decreased (10, 34) during immersion. Although immersion effects on $\dot{Q}$ are not entirely agreed upon among investigators, there is no dispute on the responses of stroke volume. Stroke volume (SV) increases ranged from 9% to 77% in water over that in air (Table 1). Heart rate (HR) modifies the magnitude of increase in $\dot{Q}$, though SV was increased in all studies (Table 1).

Correlation between stroke index (SI) and cardiac index (CI) is shown in Fig. 1, with relative changes shown in Fig. 2. Open symbols signify mean values obtained in air (25°C–28°C), and the closed symbols depict mean values in water (32°C–35.5°C). In Fig. 2, $\dot{Q}$ of Rennie's subjects falls below air values as a result of HR reduction of about 25% despite elevation in SV. Heart

TABLE 1
CIRCULATORY EFFECTS OF HEAD-OUT IMMERSION IN THERMONEUTRAL WATER

Reference	No. of subjects	Temp, °C		CI		HR		SI		Remarks
		Air	Water	Air	Water	Air	Water	Air	Water	
Arborelius et al., 1972	10	28	35	3.13	4.01	73	71	44	56	Seated, dye-dilution
Bazett et al., 1937	4	25	35	2.04	2.28	60	61	34	37	Supine, acetylene rebreathing
Begin et al., 1976	5	25	34	3.08	3.90	64	63	48	62	Seated, acetylene rebreathing
"	5	25	34	3.14	3.63	62	59	51	62	Supine
Farhi & Linnarsson, 1977	6	28	35	2.64	4.30	76	70	35	62	Seated and suspended, CO_2-rebreathing
Hood et al., 1968	5	25	33	3.14	3.50	73	66	43	53	Semi-supine, dye-dilution
McArdle et al., 1976	6	25	33	2.97	3.27	70	65	42	50	Seated, CO_2-rebreathing
Matsuda et al., 1981	4	26	34.5	4.41	5.16	70	60	63	80	Seated, impedance cardiography
Rennie et al., 1971	3		<34		−12%		−25%		+17.3%	Seated, CO_2-rebreathing

Values are means of number of subjects in each study. CI, cardiac index in liters · min^{-1} · m^{-2}; HR, heart rate in beats/min; SI, stroke index in ml · $beats^{-1}$ · m^{-2}.

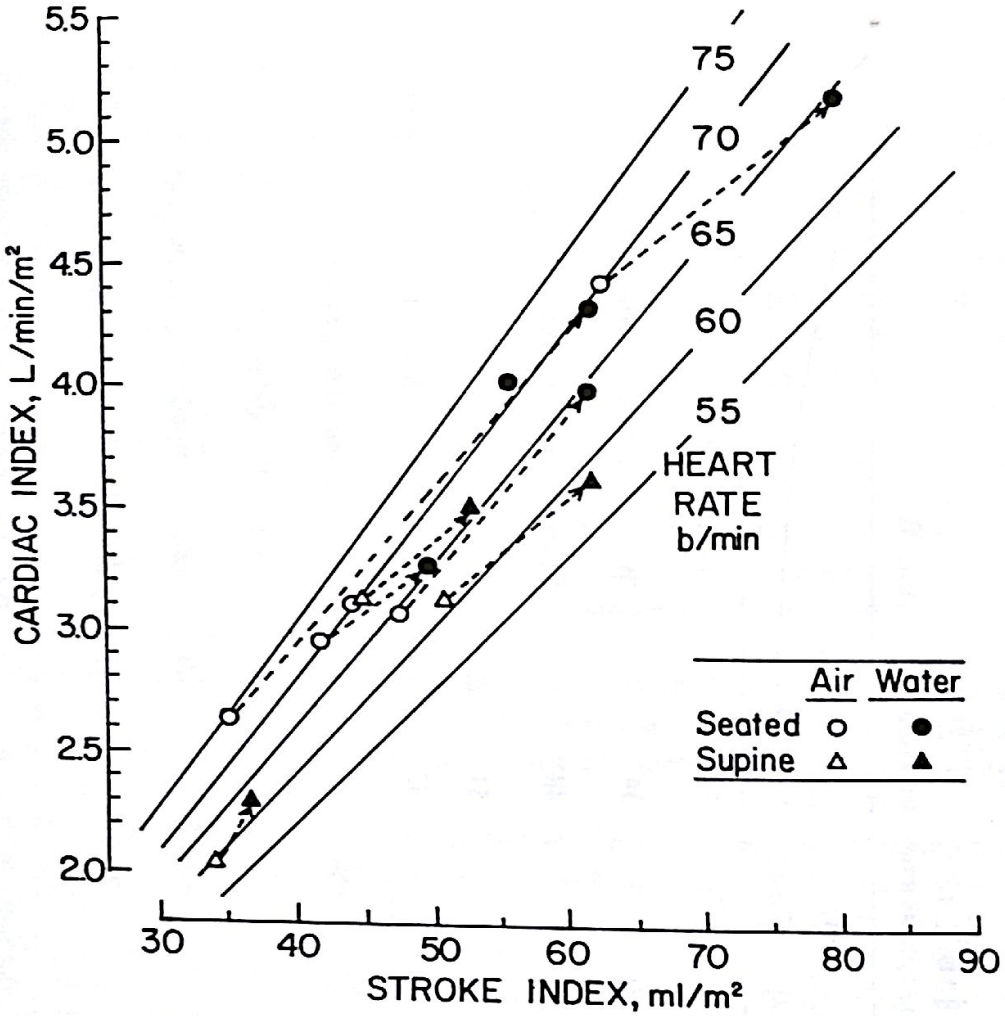

Fig. 1. Effects of head-out immersion in thermoneutral water on stroke index, cardiac index, and heart rate in human. Data for paired observations in air (25°C–28°C) and in water (33°C–35°C) are shown in Table 1. Heart rate, represented in figure as isopleths, changed little during water immersion from control value in air.

rate in water either is not changed or is lower than that observed in air (Table 1, Fig. 2; see also Table 5). However, HR plays an important role in deciding the magnitude of $\dot{Q}$ during immersion when water temperature falls below 34°C.

It is clear from this survey that water immersion elevates SV, while HR moderates increase in $\dot{Q}$ or negates it, depending on water temperature. The mechanism by which SV increases during immersion is discussed next.

MECHANISMS RESPONSIBLE FOR INCREASED STROKE VOLUME IN WATER

Stroke volume is influenced by the ventricular condition just prior to the onset of contraction, the force against which the cardiac muscle must contract during the ejection phase of systole, and the inotropic state of the myocardium. This discussion addresses the changes that occur to these three major determinants of SV during immersion in water—namely, *preload*, *afterload*, and *contractility*.

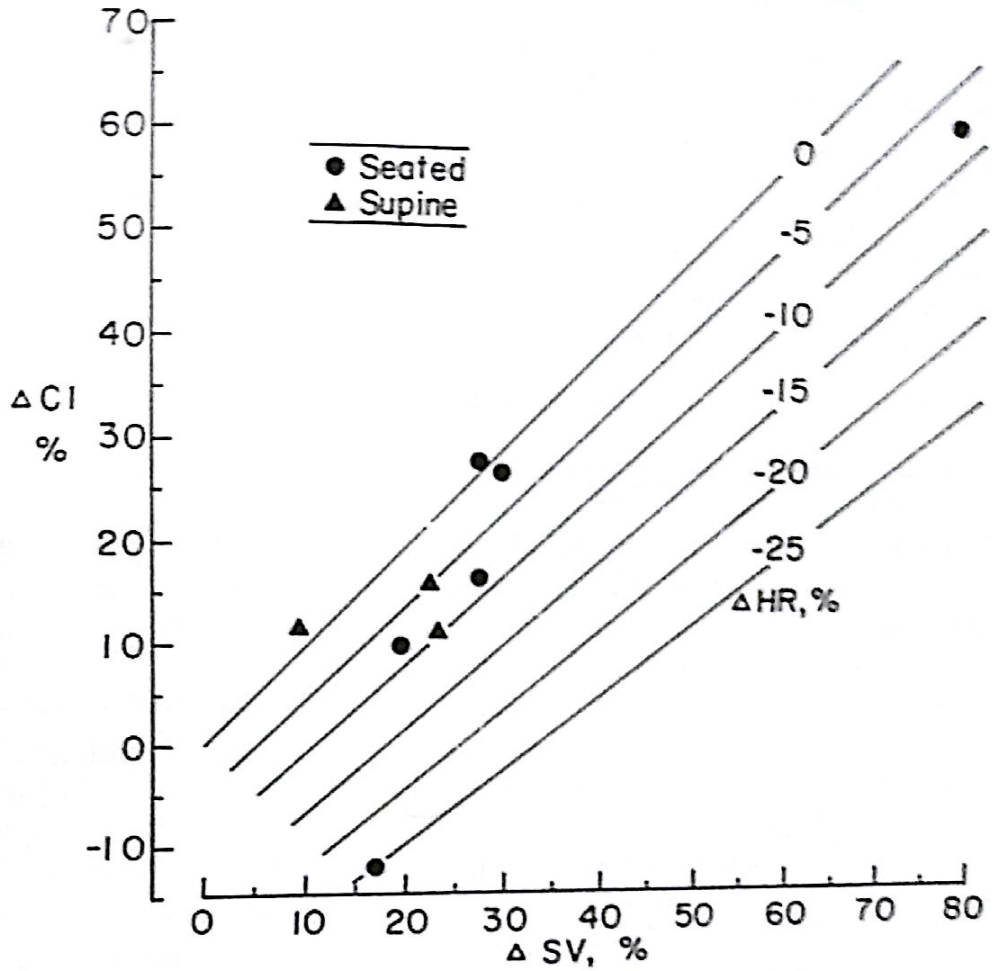

Fig. 2. Effects of head-out immersion on cardiac index (CI), stroke volume (SV), and heart rate (HR) expressed as percentage of change from their respective air controls. Rennie's data (34), which are not shown in Table 1, are represented here as −12% in CI, −25% in HR, and +17% in SV.

Preload

Within physiological limits, the strength and velocity of myocardial contraction are regulated by the extent of muscle being stretched just prior to the onset of contraction. The length of the muscle fiber at end diastole is influenced by factors affecting ventricular filling. Among them are filling time, venous tone, intrapleural pressure, intrapericardial pressure, atrial contribution, total blood volume and central blood volume (CBV), and central venous pressure (CVP). Complete discussion of all influencing factors on the preload of the heart (37) is beyond the scope of this review. Two of these, however, warrant attention: both CBV and CVP rise during water immersion.

During immersion up to the neck, CBV increases were conservatively estimated to be about 300–500 ml by Hong et al. (7) and Rahn (38), and about 700 ml by Arborelius et al. (9); ¼ of this is allotted to the cardiac chambers (1, 39). Consequently, left ventricular end-diastolic volume (LVEDV), as well as left ventricular end-diastolic pressure (LVEDP), rises. Direct measurements of LVEDV and LVEDP (indicators of myocardial fiber length and thus preload) in humans during water immersion are still lacking. But in estimation of heart size by using biplane roentgenometric technique, Lange et al. (40) reported an increase of 180 ml over the value obtained in air in 10 young men weighing 70.6 kg during immersion in 35°C water up to the neck in an upright posture. Risch et al. (41) estimated, also by roentgenographic method, that the heart enlarged by 247 ml in 6 male subjects averaging 73 kg. These values are similar to Arborelius's result by an entirely different method, if we allow ¼ of the increased CBV to

the heart, the 175 ml (700/4) in 10 subjects (9). In addition, there are several studies indicating increased pulmonary capillary blood volume and the consequent increase in CO diffusing capacity and improved gas exchange (6, 36, 42, 43). There is reason to believe that in these studies volumes in other intrapulmonary vasculatures and the heart are also increased during immersion. Risch et al. (44) disclosed that chestward translocation of blood is rapid and is virtually complete within 6 heart beats of immersion to the neck level.

Values of intrathoracic vascular compliance change little under a variety of conditions, according to Gauer and Henry (1). This is important, since

$$\Delta P = (\Delta V)/C$$

where Δ V is the volume change associated with the change in transmural pressure (Δ P) across the wall of a hollow structure on which the mechanoreceptors reside, and C is the compliance. Pressure changes reflect volume changes only if C remains constant or nearly so. Evidence indicates that veins, large or small, behave passively as long as changes in intravascular volume do not exceed 10% of normal (45). Thus, as blood is displaced into the thorax, CVP rises. More importantly, it remains elevated until the mock hypervolemia is corrected. Table 2 summarizes the magnitude of CVP changes during immersion in thermoneutral water. The average (weighted by number of subjects used in each study) of 5 studies cited here indicates that CVP increases from −1.4 mmHg in air to 15.2 mmHg in water, an increase of 16.6 mmHg. Echt et al. (46) reported also the esophagheal pressure, which increased from −0.4 mmHg before immersion to 3.4 mmHg during immersion. Thus in this study transmural pressure of the heart during diastole was estimated to be 3.8 mmHg in air and 11.8 mmHg in water (Table 2). Therefore it can be concluded that the ventricular diastolic filling is facilitated and the preload of the heart is increased during immersion. Decreased HR during immersion, which though small is the case in most studies (Table 1; see also Table 5), is another factor enhancing preload by lengthening the filling time. The favorable preload condition during immersion should result in increased SV, which was indeed found in all cases (Table 1). Unless

TABLE 2

EFFECT OF IMMERSION IN THERMONEUTRAL WATER ON CENTRAL VENOUS PRESSURE, ESOPHAGEAL PRESSURE, AND TRANSMYOCARDIAL PRESSURE

Reference	No. of subjects	CVP, mmHg		P_{es}, mmHg		TMP, mmHg	
		Air	Water	Air	Water	Air	Water
Arborelius et al 1972	10	−2	16.0				
Echt et al 1974a	5	3.4	15.2	−0.4	3.4	3.8	11.8
Koubenec et al 1978	8	−1.7	16.3				
Risch et al 1978a	6	−2	16.3				
Risch et al 1978b	8	−2.7	15.0				

CVP, central venous pressure; P_{es}, esophageal pressure; TMP, transmyocardial pressure. All subjects are males; values are means of number of subjects in each study.

drastic bradycardia intervenes, augmentation of $\dot{Q}$ should be observed during head-out immersion.

With a constant total blood volume, changes in central blood volume can be initiated by altering one or more of the following factors:

1. Capacitance of the peripheral vasculature by vasomotions, which requires the participation of autonomic nervous system. Vasomotion in veins produces greater capacitance changes than that occurring in the arterial side. Vasoconstriction reduces the capacitance and vasodilation increases it.
2. Capacity of gravity-dependent vasculatures, affected by altering body posture, gravitational orientation, or partial compression of the body.
3. Transthoracic pressure, the difference between alveolar pressure and the pressure surrounding the thorax. A negative-pressure difference, such as the case during immersion, enhances central blood pooling, and a positive pressure difference opposes it.

In Table 3 only the primary initiative factors are considered. Obviously if peripheral vasoconstriction (especially venoconstriction) occurs, it follows that the capacity of gravity-dependent vasculature also is reduced. But the initiation factor in this instance is the vasomotion. Exposure to hyperbaric environment is a special condition that warrants comment. In a helium-oxygen environment the thermoneutral zone is narrow and is higher than breathing air. Unless the ambient temperature is raised to 30°C–31°C, the subject feels cold and is vasoconstricted. Some degree of negative-pressure breathing exists in hyperbaric environments due to the high density of breathing gas. The subject encounters elevated airway resistance. It is, therefore, not surprising to observe diuresis in hyperbaric environment similar to that of water immersion (47). Breath holding is another condition that also induces increase in CBV, about 230 ml in 2 min of breath holding, attributable to the diminishing lung volume with glottis closed (48). In some respiratory problems where inspiratory efforts are elevated, the patients encounter negative transthoracic pressure; central blood volume rises for this reason.

By inspecting Table 3, it is easily seen that water immersion involves all three of these factors. First, peripheral blood flow reduces to near minimum when water temperature is at or below 35°C (49), a result of active vasoconstriction. Second, hydrostatic compression plus a diminished difference in density between tissue and its surround eliminate gravity dependency of the lower body. And finally, negative transthoracic pressure of about 14.7 mmHg promotes blood pooling in the thoracic vasculatures including the heart. Other procedures involve only one or two of the three initiation factors (Table 3). It is, therefore, not surprising that among procedures promoting central blood pooling, immersion is most effective. Increase in CBV during immersion exceeds that of assuming the supine position alone (41).

In support of the enhanced preload of the heart during immersion, the existing literatures provide sufficient data indicating increased CBV, CVP, transmural pressure of the heart, and an unaltered or lengthened filling time. As the following two sections will show that by the increase in preload alone through the Frank-Starling mechanism, it is adequate to explain the increased stroke volume (SV) during immersion in 33°C–35°C water.

Afterload

For cardiac muscle, the afterload is the force against which the myocardiac fibers must contract during ejection. The total force opposing ventricular contraction is the product of the ventricular pressure and the internal surface area of the ventricle (50). However, both pressure and area change continuously throughout systole. It is therefore difficult to assess afterload in this manner. For reasons of accessibility and practicality, alternative criteria are used. In

TABLE 3
EFFECT OF VARIOUS PROCEDURES ON INITIATION OF INCREASE IN CENTRAL BLOOD VOLUME WITHOUT CHANGING TOTAL BLOOD VOLUME

Procedure	Peripheral Vasomotion	Gravity-Dependent Capacity	Transthoracic Pressure
Immersion*	Vasoconstriction or vasodilation	Decrease	Increasing negative difference
Breath holding**	Vasoconstriction		Increasing negative difference
Hyperbaric exposure†	Vasoconstriction		Increasing negative difference
Cold	Vasoconstriction		
Head-down tilt	Vasodilation	Decrease	
Head-up tilt	Vasoconstriction	Increase	
Supine		Decrease	
Weightlessness		Decrease	
Gravity stress		Increase	
Lower body			
Negative pressure		Increase	
Positive pressure		Decrease	
Negative pressure breathing			Increasing negative difference
Positive pressure breathing			Increasing positive difference
Valsalva maneuver			Increasing positive difference
Muller maneuver			Increasing negative difference
Respiratory diseases‡			Increasing negative difference

*Peripheral vasoconstriction or vasodilation depends critically on water temperatures. **Typical breath holding induces bradycardia and peripheral vasoconstriction and also negative transthoracic pressure due to diminishing lung volume with glottis closed. †Hyperbaric exposure encounters increased density of breathing gas and hence an elevated airway resistance that increases inspiratory but not inspiratory efforts, resulting in negative pressure breathing. ‡Respiratory diseases that increase inspiratory efforts but wherein expiration remains passive, such as upper airway obstructions, especially those of extrathoracic airways.

practical terms, if one has to depend on determination of arterial pressure alone, according to Tarazi and Levy (50) systolic rather than diastolic pressure offers a value that is closest to a correct evaluation of the load imposed on the heart. Other factors being equal, in order for SV to increase, the afterload must fall. During immersion, systolic or mean arterial pressure has been reported to increase (9, 51, 52), to decrease (17, 53), or have no change (31). These changes are small in comparison to that of cardiac output. Estimation from Table 4 showed that, by weighted average, the systolic pressure increased from 116.4 to 120.5, the diastolic pressure from 70.9 to 72.4, and the mean pressure from 89.1 to 92.9 mmHg. Consequent to this small rise in blood pressure in relation to cardiac output, the calculated total peripheral resistance falls (9, 17, 31). It is therefore reasonable to assume that afterload changes little during immersion and plays no significant role in influencing cardiac performance.

Contractility

Contractile state of the myocardium is another determinant of SV. When preload and afterload are held constant, SV is a function of the contractile state. Elevation of contractility causes more forceful contraction; hence the left ventricle ends in a smaller end-systolic volume than it otherwise would, and, as a consequence, a larger SV results. But, is the contractility of the myocardium increased during immersion? There is as yet no direct assessment of myocardiac contractility in humans during water immersion. Alteration of sympathetic activity is an effective means of modifying the contractile state of the heart. Direct assessment of sympathetic activity in humans is difficult, to say the least. However, one could indirectly infer the state of sympathetic activity from measurements of plasma level or urinary excretion

TABLE 4
ARTERIAL BLOOD PRESSURE RESPONSE DURING HEAD-OUT IMMERSION IN THERMONEUTRAL WATER

		Arterial Blood Pressure, mmHg						Total Peripheral Resistance, ABP/CI	
		in air			in water				
Reference	*n*	SP	DP	ABP	SP	DP	ABP	Air	Water
Arborelius et al, 1972	10	114	71	86	128	79	98	27.5	24.4
Bazett, 1937	4	99	61	75	101	62	77	36.8	33.8
Campbell et al, 1969	18			90			100		
Craig and Dvorak, 1966	10	126	81	98	118	72	89		
Hood et al, 1968	5			95			82	30.3	23.4
Koubenec et al, 1972	8	116	63	83	124	70	90		

SP, systolic pressure; DP, diastolic pressure; ABP, arterial blood pressure; CI, cardiac index, from Table 1. Total peripheral resistance in mmHg per unit of cardiac index. Values are means of *n* subjects, all male.

of catecholamines. Reviews of plasma epinephrine (E) and norepinephrine (NE) levels, and urinary excretion of E and NE indicate that changes are insignificant when comparing conditions of being seated in air and immersion to the neck level (54, 55). If anything, urinary excretion of NE appears depressed during immersion (55–57). To the extent that the level of NE in plasma or in urine reflects sympathetic activity, and that sympathetic activity causes alteration in myocardial contractility, these results suggest that myocardial contractility is most likely unaltered or even depressed during immersion in warm water. From the foregoing discussion it is suggested that contractility plays no role in the augmentation of SV during immersion.

In summary, considering the major factors that influence cardiac performance, the enhanced preload resulting from cephalad redistribution of circulating blood appears solely responsible for the increased stroke output during immersion. The afterload and inotropic state of the heart change minimally. If anything, they change in the direction of opposing the increase of SV during immersion. Therefore they are not responsible for altered cardiac performance during immersion in thermoneutral water.

HEART RATE

Heart rate (HR) is either unchanged or decreased during immersion in thermoneutral water as compared to rate while resting in air (Table 5). The immersion bradycardia moderates the increase (Fig. 2) or even nullifies the increase in $\dot{Q}$ in the face of elevated SV (34). The data

TABLE 5
MEAN HEART RATE RESPONSE DURING HEAD-OUT IMMERSION IN THERMONEUTRAL WATER

Reference	*n*	Temp. of Water, °C	Mean Heart Rate, beats/min	
			In Air	In Water
Arborelius et al, 1972	10	35	73	71
Bazett, 1937	4	35	61	61
Begin et al, 1976	5	34	64	63
Campbell et al, 1969	18	34	72	72
Craig and Dvorak, 1966	10	35	63	63
Farhi and Linnarsson 1977	6	35	76	70
Hood et al, 1968	5	33	73	66
Keatinge and Evans 1960	12	35	80	80
Koubenec et al, 1978	8	35	74	73
Matsuda et al, 1981	4	34–35	70	60
McArdle et al, 1976	5	34	64	63
Risch et al, 1978a	8	35	80	68
Risch et al, 1978b	5	35	82	65

Values are means of *n* subjects in each study. All subjects are males.

from Keatinge and Evans (58) showed that after 19 min of immersion HR is lower when water temperature falls below 35°C, and HR rises drastically when water temperature exceeds 35°C (Fig. 3). The present survey includes studies of water temperatures between 33°C and 35°C. We should expect HR to be either not changed or slightly lower than 35°C conditions. Put simply, this is the case (Table 5). The explanation for this result, however, is anything but simple.

Heart rate responses are complex and are highly buffered. It is interesting to note that several opposing factors act together to determine the HR during immersion. Other factors being constant, the rising atrial pressure should trigger a tachycardial response through the Bainbridge reflex (59–61). But concurrent elevation of arterial pressure, by the increasing SV and $\dot{Q}$, opposes the tachycardia via the baroreceptor mechanism. Cutaneous vasodilation, by transferring the subjects at 25°C air to 35°C water, is opposed by the hypertension-baroreceptor mechanism also. When the thermal condition is such that peripheral vasoconstriction is intense (water temperature below 35°C, for example) the baroreflex-induced bradycardia should be moderated by tachycardia induced by the Bainbridge reflex. It is reasonable to predict that if any change at all occurs in the HR during immersion, it will be minor due to these antagonistic actions. Clear-cut bradycardia results only when intense peripheral vasoconstriction occurs during the stimulus of severe cold (34).

HEMODYNAMIC STATES AND BREATH-HOLD DIVES

A breath-hold (BH) dive in open water is necessarily preceded by immersion up to the neck. And, most likely, the divers spend most of their time in this condition. As has been summarized,

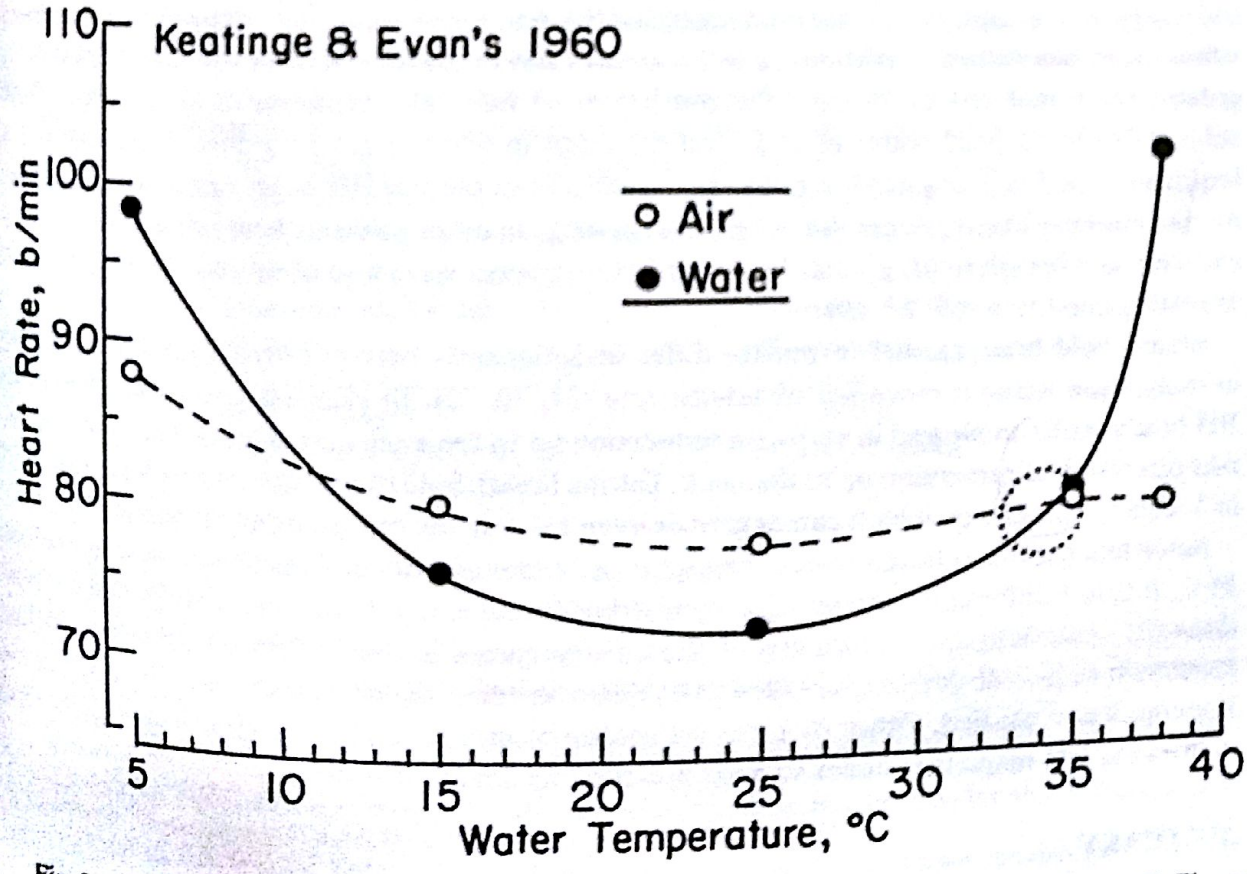

Fig. 3. Heart rate responses to immersion in water when temperature varied from 5°C to 38°C. Figure is plotted using Keatinge and Evans' data (58), the means of 7–12 subjects at each temperature. *Dotted circle*, majority of values summarized in Table 5, where water temperature varied between 33°C and 35°C.

an unusual circulatory condition exists during head-out immersion. Furthermore, the diver requires a metabolic rate above resting condition, either staying afloat in place, replacing thermal loss to the water, or recovering from diving activity. On the other hand much of the cardiopulmonary changes during BH dives are generalized from studies conducted in the laboratory where, in most cases, the face is the only part that makes contact with water, or not even that. In general, breath hold induces various degrees of bradycardia, peripheral vasoconstriction, hypertension, and variable cardiac output (23). Whether the two distinct pre-BH circulatory, respiratory, and metabolic states influence the responses during breath hold has not been systematically examined.

As alluded to previously, pre-BH physiological state in open water involves changes in the circulatory, respiratory, and metabolic systems. Each of these could potentially affect cardiovascular responses to breath hold. Various aspects of physiological stresses that confront humans as working BH divers are summarized by Rahn and Yokoyama (62) for the earlier studies. In the natural settings it is necessary to measure simpler quantities or measurements under severe limitations (63, 64). It is not surprising that in most cases, for cardiovascular responses, only ECG and/or HR are available. In the laboratory a friendlier environment and the availability of instrumentation encourage more sophisticated quantification of physiological variables. But investigators see fit to simulate the natural condition only in part: for example, breath hold only, either in air or with face immersed (for review see Ref. 33), exercise and breath hold (65–69), immersion and breath hold (51, 70–72) and, only rarely, the combination of swimming and breath hold (73). For this reason we still do not have a clear-cut picture of what influences the pre-BH cardiopulmonary and metabolic states (comparing resting in air and in water) have on the circulatory responses to breath hold. What we do have points to the direction that bradycardial response in natural settings (immersion in water below thermoneutral temperature, and exercising) is exaggerated (63, 64). Previous review reveals that exercising subjects, in laboratory conditions, exhibit greater BH bradycardial response than at rest by a greater fractional reduction from the pre-BH heart rate (23). Stromme et al. (73) had their subjects swim in pool water at 25°C and recorded in one subject a cardiac interval of 6.8 s (equivalent to 8.8 beats/min), a reduction of 90% from the pre-BH heart rate. Obviously this marine mammallike response did not persist for long. In other studies where subjects performed exercise and breath hold, greater bradycardial responses were also observed than breath hold at resting condition (65–67, 69).

Breath-hold bradycardial responses differ insignificantly between breath hold on land and in immersion without elevating metabolic rate (51, 70, 72). In Harding's study there was no BH bradycardia in air and in stepwise immersion up to the xiphisternal level, but bradycardia was observed in immersion up to the neck. During breath hold their subjects may have engaged in Valsalva maneuver, which can negate or even reverse the bradycardial responses (72, 74).

Since in a natural setting subjects engage in muscular activity and immersion in water below 35°C, it is not difficult to imagine that sympathoadrenal activity is elevated under such conditions (75, 76). Whether sensitivity of the baroreceptors is thus enhanced deserves further inquiry. It is also desirable that other variables of cardiovascular responses to breath hold be systematically studied, either in a natural setting or in a simulated condition, especially that comparing BH responses under various pre-BH physiological states.

SUMMARY

Current literature provides abundant data demonstrating that an unusual circulatory state exists in humans immersed in water at or near 35°C. This circulatory state is characterized by

a persistent and elevated stroke volume with heart rate that changes little from the preimmersion condition. Cephalad redistribution of blood increases central blood volume, central venous pressure, and heart volume during diastole, and generally a favorable preload prevails. Thus the whole body is under a hyperperfused state without a concurrent rise in metabolic requirements, a condition not conforming to the concept of autoregulation. Current data suggest that the BH-bradycardial response is exaggerated during immersion. It also suggests that systemic comparisons of other circulatory variables between breath hold on land and in water require further attention. Comparison of results during breath hold between the two distinct pre-BH physiological states (i.e., on land and in water) may yield useful information that furthers the understanding of stresses confronting humans as BH divers.

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Lin YC. Fonctions circulatoires durant l'immersion et les plongées en apnée chez l'homme. Undersea Biomed Res 1984; 11(2):123–138.—Un état circulatoire inhabituel existe chez les humains immergés dans l'eau à ou près de 35°C. Cet état circulatoire est caractérisé par une élévation soutenue de l'indice systolique (SI) mais avec peu de changement dans la fréquence cardiaque (FC) et donc une augmentation résultante de l'indice cardiaque (CI). Dans la comparaison des mesures dans l'air et lors de l'immersion dans l'eau jusqu'au cou, lesquelles sont basées sur les moyennes pondérées de 45 sujets dans 8 études, SI augmenta de 29% et CI de 24%, tandis que FC diminua de 6%. L'évidence est présentée pour appuyer la découverte que l'augmentation du volume systolique est le résultat d'un accroissement dans la précharge cardiaque, et que les modifications dans la surcharge et la contractilité exercent un rôle peu important sur la performance cardiaque durant l'immersion jusqu'au cou dans de l'eau thermoneutre. L'état circulatoire représente une surperfusion puisqu'il n'y a pas d'élévation concomitante dans la demande métabolique ou le stress thermique. Jusqu'á quel point cet état circulatoire pré-existant affecte les réponses cardiovaculaires reliées à la plongée en apnée (PA) demeure principalement inconnu. Cependant les réponses bradycardiques à la PA sont plus puissantes dans un milieu naturel que dans un laboratoire où l'apnée est exécutée avec le visage immergé dans l'eau seulement. Au contraire, dans le milieu naturel, les plongeurs exécutent des plongées en apnée lorsqu'ils sont immergés jusqu'au cou dans de l'eau froide (à des températures beaucoup plus basses que 35°C) et au cours de l'exercice. Il serait souhaitable dans des études futures de comparer systématiquement d'autres aspects des réponses circulatoires aux plongées en apnée lorsqu'immergé dans l'eau et durant l'exercice.

immersion dans l'eau
humain
état circulatoire
débit cardiaque
volume systolique
fréquence cardiaque
pré-charge
surcharge
contractilité
plongée en apnée

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