

# Haemodynamic changes in man during immersion in water at different temperatures

C. F. M. WESTON, J. P. O'HARE, J. M. EVANS\* AND R. J. M. CORRALL

Department of Medicine and \*Medical Physics, Bristol Royal Infirmary, Bristol, U.K.

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## SUMMARY

1. Stroke volume and cardiac output were measured using the Doppler ultrasound technique in 16 normal subjects immersed to the neck in water at 33°C, 35°C, 37°C and 39°C. A standard aortic diameter was assumed and results were expressed as percentage changes from pre-immersion resting values.

2. Cardiac output rose progressively at higher temperatures, increasing by 30% at 33°C and by 121% at 39°C. At thermoneutral temperatures (33°C and 35°C) this was achieved by an increase in stroke volume of 50% despite a significant decrease in heart rate. There was a further rise in stroke volume and pulse rate at higher temperatures and a mean tachycardia of  $109 \pm 4$  beats/min was noted at 39°C. Calculated peripheral resistance reduced progressively with increasing temperature of immersion.

3. This non-invasive and simple technique may provide a non-exercise-related cardiovascular stress test to study cardiovascular responses in a variety of pathophysiological states.

**Key words:** cardiac output, Doppler ultrasound, haemodynamics, immersion.

## INTRODUCTION

Water immersion to the neck has marked physiological effects on the cardiovascular system through a redistribution of blood from limbs to thorax [1]. In the present study we have examined the cardiovascular response to water immersion at a variety of temperatures. The results indicate that the temperature of immersion has profound effects on the physiological changes observed.

Correspondence: Dr R. J. M. Corral, Department of Medicine, Bristol Royal Infirmary, Bristol BS2 8HW, U.K.

## EXPERIMENTAL

### Subjects and methods

Studies of immersion were performed on a group of healthy individuals comprising nine men and seven women with a mean age of 34 years (range 18-65 years).

Immersion was carried out in a modified jacuzzi-bath (Aquatech Ltd, Newbury, Berks., U.K.) using tap water which was constantly recirculated, filtered and brominated. The temperature of immersion was adjusted rapidly by mixing hot or cold water and then maintained by a thermostatically controlled heater. A temperature change of 2°C could be completed within 5 min. Core and water temperatures were obtained using a thermocouple (Thermalert; Bailey Instruments Ltd, Saddle Brook, NJ, U.S.A.).

After a control period of 15 min in the seated position at a room temperature of 28°C, resting measurements were made outside the tank. Subjects were then immersed in the seated position to the level of the sternal notch in water at 35°C, 37°C, 39°C and 33°C in that order. An acclimatization period of 15 min was allowed at each temperature before recordings were made. At the end of the study post-immersion values seated outside the tank were obtained. Subjects were not allowed to pass urine during the study.

Cardiac output was measured using an ultrasonic probe connected to a 2 MHz pulsed Doppler flowmeter (Vingmed Pedof), placed in the suprasternal notch. The sample volume was placed within the ascending aorta and adjusted to obtain the maximum Doppler shift at approximately 5-6 cm from the body surface. This technique was found to minimize any error due to the unknown angle of isonation to the vessel and to allow the sample volume to be placed in the centre stream of the flow. The mean frequency of the Doppler spectrum was estimated using an analogue circuit. This velocity/time envelope (the angle of isonation being assumed zero) was converted into digital form and displayed on a microcomputer (BBC B). The

criteria for acceptance of adequate signals were a rapid onset and cessation of flow and an absence of diastolic flow as judged by listening to the audible signal. An average from 15 cardiac cycles was obtained and cardiac index was calculated assuming a fixed aortic diameter of 28 mm for men and 27 mm for women. Heart rate and stroke volume were also computed from the signals, and blood pressure at the brachial artery was measured using a mercury sphygmomanometer using phase V as the diastolic blood pressure. Peripheral resistance was calculated from cardiac output and mean blood pressure. Mean blood pressure was taken as the sum of diastolic blood pressure and one-third of the pulse pressure (systolic minus diastolic).

Because of assumptions relating to the aortic cross-sectional area, results are expressed as percentage changes from pre-immersion values rather than in absolute units. If sampling is consistent within subjects then the effect of differences in aortic diameter between patients will thus be effectively eliminated.

### Reproducibility

To avoid variation in technique between observers, one of us (J.M.E.) performed all the Doppler flow sampling in this study. Reproducibility was assessed by obtaining eight cardiac output estimations at 5 min intervals in one normal subject immersed at 35°C and 37°C. The coefficients of variation were 6.4% and 3.5% respectively.

### Statistical analysis

Data are expressed as means  $\pm$  SEM. Statistical analysis was performed using two-way analysis of variance for the effect of temperature change.

## RESULTS

Body-core temperatures in the 16 subjects did not alter significantly on immersion in water at temperatures of 33°C and 35°C, but increased from a pre-immersion mean value of  $36.7 \pm 0.1^\circ\text{C}$  to  $37.2 \pm 0.1^\circ\text{C}$  at 37°C and  $38.3 \pm 0.1^\circ\text{C}$  at 39°C ( $P < 0.001$ ).

Cardiac index rose significantly at each temperature above the pre-immersion estimate of  $5.4 \pm 0.2$  litre/min, to  $6.9 \pm 0.3$  litre/min at 33°C,  $7.1 \pm 0.4$  litre/min at 35°C,  $9.6 \pm 0.4$  litre/min at 37°C and  $11.7 \pm 0.4$  litre/min at 39°C ( $P < 0.001$  for all measurements). These increases may be expressed as percentage increments of 30%, 34%, 80% and 121% respectively (Fig. 1). This was achieved despite a fall in heart rate at 33°C to  $70 \pm 3$  beats/min from  $82 \pm 3$  beats/min pre-immersion ( $P < 0.02$ ). A similar bradycardia was also seen at 35°C when the heart rate was  $73 \pm 3$  beats/min ( $P < 0.05$  vs pre-immersion). At both these water temperatures stroke volume increased by just over 50% ( $51 \pm 6\%$  at 33°C and  $50 \pm 6\%$  at 35°C). A further increment of stroke volume at 37°C and 39°C was noted, by 67% and 64% above pre-immersion values respectively. At these higher temperatures heart rate rose to mean levels of  $87 \pm 3$  beats/min at 37°C and  $109 \pm 4$  beats/min at 39°C ( $P < 0.001$ ).

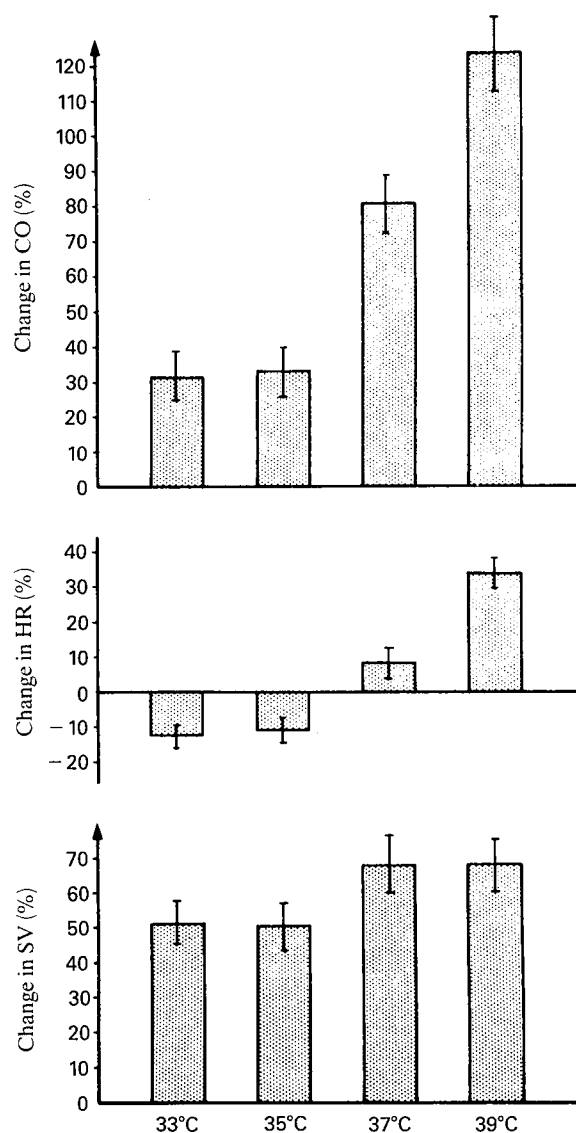


Fig. 1. Percentage change in cardiac output (CO), heart rate (HR) and stroke volume (SV) at different temperatures of water immersion. Bars indicate  $\pm$  SEM.

Systolic blood pressure did not change significantly during the study, yet mean blood pressure altered due to a fall in diastolic pressure by a mean value of 9 mmHg at 33°C and 35°C, 18 mmHg at 37°C and 30 mmHg at 39°C (Fig. 2). Calculated peripheral resistance was therefore reduced progressively at increasing temperatures of water immersion by 31% at 35°C, 53% at 37°C and 65% at 39°C.

There were no significant differences between the pre- and post-immersion results, although there was a tendency towards a diminished stroke volume and cardiac output ( $5.1 \pm 0.2$  litre/min compared with  $5.4 \pm 0.2$  litre/min) and an increased heart rate ( $85.7 \pm 4.1$  beats/min compared with  $81.7 \pm 3.0$  beats/min) in the post-immersion period.

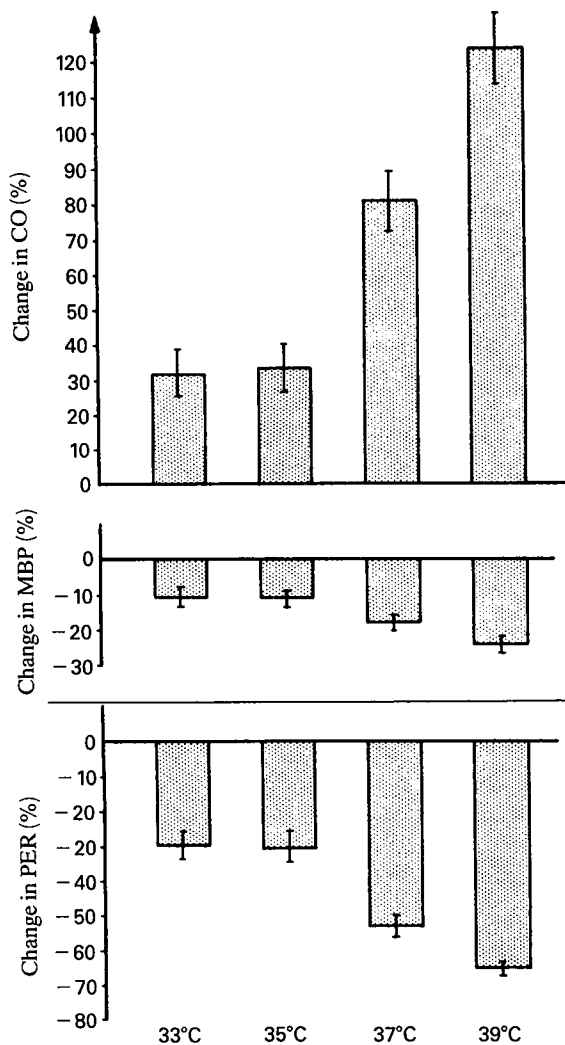


Fig. 2. Percentage change in cardiac output (CO), mean blood pressure (MBP) and peripheral resistance (PER) at different temperatures of water immersion. Bars indicate  $\pm$  SEM.

## DISCUSSION

The Doppler ultrasound technique for assessment of cardiac function has been well documented and validated [2, 3]. It is a simple, non-invasive procedure that may be easily and quickly repeated, allowing measurement of rapid changes in cardiac output and stroke volume. It compares favourably with other methods of cardiac output determination which are cumbersome and may be less reliable [4, 5]. Reproducibility is satisfactory in the present and other studies, with quoted coefficients of variation ranging from 5 to 11% [2, 6, 7].

There are, however, a number of assumptions inherent in the conversion of the Doppler-derived value of ascending aortic blood velocity into an estimate of stroke volume. The velocity profile of the aorta is assumed to be flat; that is the flow is not turbulent and the velocity is constant at all points in a cross-section of the lumen. The angle of isonation (between the direction of the ultra-

sound beam and the blood flow vector) is assumed to be zero. The aorta is assumed to be a cylinder with a perfectly circular cross-section that does not vary with changes in blood pressure. The measurement of aortic cross-sectional diameter by echocardiography is felt to be the main source of error in estimating cardiac output by this technique [3]. More recently one group has attempted to eliminate this source of error by declaring it unnecessary to measure cross-sectional area and have directly linked stroke distance and minute distance to stroke volume and cardiac output [8]. For direct measurement of aortic cross-sectional area it is assumed that Doppler flow sampling takes place at the point where the area is measured. With the present method this cannot be checked when the beam is being directed externally. This potential source of inaccuracy can be avoided by recording only the relative changes in indices measured. In the present study we have elected not to express our results in terms of absolute values and have assigned constant aortic diameters to male and female subjects, recording the results as percentage changes from resting values. The central translocation of blood from the periphery may affect the aortic area, but this effect should be similar at all water temperatures and not influence the measurement obtained.

In future studies it is hoped to minimize some of the errors in flow due to the use of a narrow Doppler beam. Currently under development is a probe which produces a wide beam of ultrasound to completely encompass the vessel of interest, allowing a true mean velocity to be measured [9]. Moreover, using a pulsed Doppler flowmeter, as we have in this study, instead of a continuous wave system, allows the operator to detect blood flow signals from a known sample volume within the ascending aorta at the depth of interest. Signals from valves and other vessels lying along the ultrasound beam axis are hence rejected, allowing a more accurate and reliable flow measurement to be made.

The major documented physiological effects of water immersion are haemodynamic and those renal effects related to volume homeostasis (diuresis, natriuresis, kaliuresis) [10-12]. The haemodynamic changes are mediated through an increased venous return to the thorax induced by a hydrostatic pressure gradient, dependent on the depth of water in which the subjects are immersed. The right atrial pressure has been shown to rise by 18 mmHg and mean pulmonary artery pressure by 12 mmHg during thermoneutral water immersion at 35°C, and central blood volume is increased by about 700 ml [13]. Reported changes in cardiac output have varied between studies, with increases ranging between 30% and 60% at 35°C [13, 14]. The results we present confirm previous work and show that some of the variation in other earlier reports relates to differences of immersion water temperature. By increasing the temperature of water immersion we have been able to show a progressive rise in cardiac output which doubles at 39°C, associated with a fall in diastolic blood pressure and total peripheral resistance. At thermoneutral temperatures this was achieved in spite of a significant reduction in heart rate by increasing the

stroke volume by approximately 50%. At 37°C and 39°C there was a further rise in stroke volume associated with a tachycardia.

The relative bradycardia at lower temperatures has been documented both during physical work and in the resting state [15], and appears to be dependent on the level of immersion. It is thus greatest on immersion to the level of the xiphoid, although it is still significant on immersion to the neck [14]. It is probably mediated through cardiac vagal innervation, being reduced in subjects with diabetic autonomic neuropathy [16].

It is probable that variation in bath temperature produces a complex series of changes under baroreceptor control in response to alterations in pulse pressure, mean blood pressure and peripheral resistance. While variations in baroreceptor response contribute to the tachycardia at higher temperatures, the most important factor is likely to be the increased rate of sinu-arterial node depolarization at higher body-core temperatures [17]. The baroreceptor reflex has only a minor role in the control of myocardial contractility [18]. The increase in contractility is thus probably directly related to a raised body-core temperature, but mediation via the sympathetic activity and humoral factors may also be involved.

There is a reduction in total peripheral resistance at all temperatures of immersion. Under thermoneutral conditions the skin temperature rises towards that of the core [19], leading to cutaneous vasodilatation via local changes of arteriolar autoregulation. There is a diminution of skin-initiated 'cold vasoconstrictor autoregulation'. At higher core temperatures this vasoconstrictor response is completely abolished by a central mechanism, leading to a fall in sympathetic nervous activity referred to as a 'physiological sympathectomy' or the 'sauna phenomenon' [20]. The possible contribution of humoral vasodilating factors, for example atrial natriuretic peptide and kinins, is at present speculative.

Warm-water immersion constitutes a non-exercise-related cardiovascular stress test causing increased cardiac output by an increased stroke volume at 35°C and via both stroke volume and heart rate change at higher water temperatures. The recorded tachycardias were not, however, in the same range as those achieved with treadmill exercise testing. Higher immersion temperatures were attempted but were poorly tolerated by our volunteers. Furthermore we consider it unnecessary to undertake immersion at 33°C; there is little difference in comparison with results obtained at 35°C. Thus the present experimental model of water immersion at 35°C, 37°C and 39°C could be utilized to investigate physiological responses in patients with abnormalities of myocardial contractility, peripheral vasodilatation or autonomic function. It may also be of value in the assessment of pharmacological agents.

## REFERENCES

- O'Hare, J.P., Heywood, A., Summerhayes, C., Lunn, G., Evans, J.M., Walters, G., Corral, R.J.M. & Dieppe, P.A. (1985) Observations on the effects of immersion in Bath spa water. *British Medical Journal*, **291**, 1747-1751.
- Huntsman, L.L., Stewart, D.K., Barnes, S.R., Franklin, S.B., Colonsis, J.S. & Messel, E.A. (1983) Non-invasive Doppler determination of cardiac output in man: clinical validation. *Circulation*, **67**, 593-602.
- Ihlen, H., Amlie, J.P., Dale, J., Forfang, K., Nitter-Hauge, S., Otterstad, J.E., Simonsen, S. & Myhra, E. (1984) Determination of cardiac output by Doppler echocardiography. *British Heart Journal*, **51**, 54-60.
- Distante, A., Moscarelli, E., Rovai, D. & L'Abbate, A. (1980) Monitoring changes in cardiac output by transcutaneous aorto-velography a non-invasive Doppler technique: comparison with thermodilution. *Journal of Nuclear Medicine and Allied Science*, **24**, 171-175.
- Ihlen, H., Mybre, E., Amlie, J.P., Forfang, K. & Larsen, S. (1985) Changes in left ventricular stroke volume measured by Doppler echocardiography. *British Heart Journal*, **54**, 378-383.
- McLennan, F.M., Haites, N.E., Mackenzie, J.D., Daniel, M.K. & Rawle, J.M. (1986) Reproducibility of linear cardiac output measurement by Doppler ultrasound alone. *British Heart Journal*, **55**, 25-31.
- Gardin, J.M., Dabestani, A., Matin, K., Allfie, A., Russell, D. & Henny, W.L. (1984) Reproducibility of Doppler aortic blood flow measurements: studies on intraobserver, interobserver and day-to-day variability in normal subjects. *American Journal of Cardiology*, **54**, 1092-1098.
- Haites, N.E., McLennan, F.M., Mowatt, D.H.R. & Rawles, J.M. (1985) Assessment of cardiac output by the Doppler ultrasound technique alone. *British Heart Journal*, **53**, 123-129.
- Evans, J.M., Skidmore, R. & Wells, P.N.T. (1986) A new technique to measure blood flow using Doppler ultrasound. In: *Physics in Medical Ultrasound*, no. 47, chapter 18, pp. 141-144. Ed. Evans, J.A. Institute of Physical Sciences in Medicine, London.
- Epstein, M. (1978) Renal effects of head-out water immersion in man: implications for an understanding of volume homeostasis. *Physical Reviews*, **58**, 529-581.
- Anderson, J.V., Miller, N., O'Hare, J.P., McKenzie, J.C., Corral, R.J.M. & Bloom, S.R. (1985) Do circulating levels of atrial natriuretic peptide mediate the natriuretic response to water immersion in man? *Clinical Science*, **69** (Suppl. 12), 39.
- O'Hare, J.P., Roland, J., Scott, G., Walters, G. & Corral, R.J.M. (1984) Haemodilution during water immersion in man (Abstract). *Clinical Science*, **66** (Suppl. 10), 47P.
- Arborelius, M., Jr, Ballin, U.I., Lilja, B. & Lundgren, C.E.G. (1972) Haemodynamic changes in man during immersion with the head above water. *Aerospace Medicine*, **43**, 592-598.
- Farhi, L.E. & Linnarsson, D. (1977) Cardiopulmonary readjustments during graded immersion in water at 35°C. *Respiration Physiology*, **30**, 35-50.
- McArdle, W.S., Magel, J.R., Lesmes, G.R. & Fechar, G.S. (1976) Metabolic and cardiovascular adjustments to work in air and water at 18, 25 and 33°C. *Journal of Applied Physiology*, **40**, 85-90.
- O'Hare, J.P., Evans, J.M., Weston, C.F.M., O'Brien, I.A.D., Millar, N. & Corral, R.J.M. (1987) Cardiovascular responses to water immersion in diabetics with autonomic neuropathy (Abstract). *Journal of the Autonomic Nervous System* (In press).
- Ganong, W.F. (1975) The heart: origin of the heart beat and the electrical activity of the heart. In: *Review of Medical Physiology*, pp. 395-411. Lange Medical, California.
- Braunwald, E. (1974) Regulation of the circulation. *New England Journal of Medicine*, **290**, 1124-1129.
- Robinson, S. (1968) Physiological adjustments to heat. In: *Physiology of Heart Regulation and the Science of Clothing*, pp. 193-231. Ed. Newburgh, L.H. Hafner Publishing Co., New York.
- Grayson, J. & Kuehn, L.A. (1979) Heat transfer and heat loss. In: *Body Temperature*, pp. 71-81. Ed. Lomax, P. & Schonbaum, E. Marcel Dekker Inc., New York.