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ORIGINAL ARTICLE

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Human physiological responses to immersion into water of different temperatures

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Abstract To differentiate between the effect of cold and hydrostatic pressure on hormone and cardiovascular functions of man, a group of young men was examined during 1-h head-out immersions in water of different temperatures (32°C, 20°C and 14°C). Immersion in water at 32°C did not change rectal temperature and metabolic rate, but lowered heart rate (by 15%) and systolic and diastolic blood pressures (by 11%, or 12%, respectively), compared to controls at ambient air temperature. Plasma renin activity, plasma cortisol and aldosterone concentrations were also lowered (by 46%, 34%, and 17%, respectively), while diuresis was increased by 107%. Immersion at 20°C induced a similar decrease in plasma renin activity, heart rate and systolic and diastolic blood pressures as immersion at thermoneutrality, in spite of lowered rectal temperature and an increased metabolic rate by 93%. Plasma cortisol concentrations tended to decrease, while plasma aldosterone concentration was unchanged. Diuresis was increased by 89%. No significant differences in changes in diuresis, plasma renin activity and aldosterone concentration compared to subjects immersed to 32°C were observed. Cold water immersion (14°C) lowered rectal temperature and increased metabolic rate (by 350%), heart rate and systolic and diastolic blood pressure (by 5%, 7%, and 8%, respectively). Plasma noradrenaline and dopamine concentrations were increased by 530% and by 250% respectively, while diuresis increased by 163% (more than at 32°C). Plasma aldosterone concentrations increased by 23%. Plasma renin activity was reduced as during immersion in water at the highest temperature. Cortisol concentrations tended to decrease. Plasma adrenaline concentrations remained unchanged. Changes in plasma renin activity were not related to changes in aldosterone concentrations. Immersion in water of different temperatures did not increase blood concentrations of cortisol. There was no correlation between changes in rectal temperature and changes in hormone production. Our data supported the hypothesis that physiological changes induced by water immersion are mediated by humoral control mechanisms, while responses induced by cold are mainly due to increased activity of the sympathetic nervous system.

Key words Cold water immersion · Cardiovascular functions · Catecholamines · Plasma renin activity · Cortisol

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Introduction

Immersion in cold water may exert its influence on the human body by both stimulation of baro- and cold receptors and by activation of the sympathetic nervous system and endocrine function. These responses may in turn influence mechanisms controlling cardiovascular function and water balance.

It has been shown repeatedly that immersion into thermoneutral water increases central blood volume (Arborelius et al. 1972; Epstein et al. 1981), mean stroke volume and, consequently, mean cardiac output, whereas systemic vascular resistance has been shown to decrease (Arborelius et al. 1972; Echt et al. 1974). Changes in blood pressure induced by immersion are not uniform. Most investigators have failed to observe

changes in mean arterial pressure, while others (Coruzzi et al. 1985; Myers et al. 1988) have observed a decline in arterial pressure.

It can be presumed that the increased cardiac output would increase splanchnic flow, the result having been shown to be increased diuresis, diminution of plasma renin activity (PRA, Epstein 1978; Epstein and Saruta 1971; Greenleaf et al. 1980) and aldosterone production (Epstein et al. 1975; Epstein and Saruta 1971). An examination of the relationships between PRA and plasma aldosterone concentration has disclosed a striking parallel. It has been suggested that stimulation of the left atrial baroreceptors may also modulate renal output, also that left atrial distension and stimulation of cardiopulmonary baroreceptors during thermoneutral water immersion (Henry – Gauer reflex) may induce a decrease of activity in the sympathetic nervous system, which in part may also contribute to the increased diuresis and natriuresis (Behn et al. 1969; Kaiser et al. 1969; Galbo et al. 1979).

Epstein et al. (1983) have shown that water immersion fails to alter plasma noradrenaline and adrenaline concentrations, while Krishna et al. (1983) have demonstrated a decrease in plasma noradrenaline concentration. Furthermore, Coruzzi et al. (1986) have concluded that the dopaminergic system constitutes an important determinant of the natriuretic response to water immersion. Various aspects of physiological responses to immersion in water at thermoneutral temperatures have been reviewed recently (Epstein 1992).

All the above mentioned experiments were performed on humans immersed in water at thermoneutral temperatures. Only few data are available on humans immersed in cold water. Keatinge and Evans (1961) have reported changes in activity of the cardiovascular system and Johnson et al. (1977) and Janský et al. (1996) have examined changes in catecholamine production. No attempt has been made to distinguish between responses induced by activation of baro-, or cold receptors. Therefore, this study was performed to investigate cardiovascular and hormone changes during 1-h head-out immersion of young men in water at temperatures of 32°, 20° and 14°C.

Methods

Subjects

Ten young men [mean age 22.2 (SD 2.4) years, body mass 81.1 (SD 8.2) kg, body fat content, assessed from skinfold measurement 10.2 (SD 2.0)%, maximal oxygen uptake, 56.2 (SD 6.9) $ml \cdot min^{-1} \cdot kg^{-1}$] underwent 1-h head-out water immersions, sitting on a chair in water of different temperatures. The water was stirred manually every 2 min. During the immersions the subjects wore only swimming trunks. Prior to and after immersion they rested on a bed under blankets. The air temperature of the experiment room was 25°C. Control measurements of plasma cortisol and aldosterone concentrations were made in the subjects resting in thermoneutral conditions for 60–120 min. The subjects were then immersed in water at 14, 20 and 32°C in a random sequence with an interval of 1 week between each exposure. Experiments were performed in October and November from 9 a.m. to 11 a.m.

All the subjects passed physical examinations before and after the experiments. The study was approved by the Ethics Committee at the Faculty of Physical Education and Sports, Charles University, Prague.

Protocol

Heart rate was measured using a Sport tester Polar Electro 300, systolic and diastolic blood pressures were measured using a sphygmomanometer. Rectal temperature was measured using a copper constant thermocouple and metabolic rate using a O_2 Analyser Ergo Oxyscreen (Jaeger). All parameters were followed at 5-min intervals before, during and after immersion.

Blood samples were taken using an indwelling cannula from an antecubital vein immediately before immersion (0 min), after 30 min of immersion (30 min), immediately after immersion (60 min), and after 60 min of recovery (120 min). The cannula was inserted 30 min before taking the first blood sample. Blood, drawn into ice chilled tubes containing anticoagulants, was centrifuged (4°C, 1.500 *g* for 15 min). The plasma was kept frozen at −25°C. Assays were performed within 3 weeks. Plasma catecholamine concentrations were measured using radioenzymatic kits (Catechola, no. 1709, Immunotech – UVVVR, Prague, Czech Republic). Plasma aldosterone, cortisol concentrations and PRA were measured using a radioimmunoassay method (Immunotech – UVVVR kits nos. 1664, 1841, 1682, Prague, Czech Republic).

A 10-h urine collection was made during the night before immersion. The second urine sample was collected after immersion and the third 3 h afterwards during rewarming. The average rate of urine flow was calculated by dividing the total urine volume collected by the total time of the collection period.

Statistics

Preimmersion values were compared with values obtained at given times, using Student's paired *t*-test. Statistical significance was set at the $P < 0.05$ level.

Results

Immersions into 32°C water did not change rectal temperature and metabolic rate significantly (Fig. 1). Heart rate decreased by 9 beats $\cdot$ min^{-1}, systolic pressure by 12 mmHg and diastolic pressure by 8 mmHg (Fig. 2). The PRA was lowered from 1.06 (SD 0.57) to 0.52 (SD 0.27) $ng \cdot ml^{-1} \cdot h^{-1}$ ($P = 0.01$) and diuresis increased from 1.05 (SD 0.32) to 2.17 (SD 1.02) $ml \cdot min^{-1}$ ($P = 0.009$). Aldosterone concentration was lowered from 90.5 (SD 36.9) to 75.1 (SD 23.9) $pg \cdot ml^{-1}$ ($P = 0.05$; Fig. 3) and cortisol from 185.1 (SD 49.4) to 122.1 (SD 27.1) $nmol \cdot l^{-1}$ ($P = 0.005$; Fig. 4).

Immersion at 20°C lowered heart rate ($P = 0.001$) and both systolic and diastolic blood pressures, as during immersion at thermoneutrality, in spite of a lowering of rectal body temperature [from 37.1 (SD 0.2) to 36.1 (SD 0.48)°C, $P = 0.001$] and a stimulation of metabolic rate by about 93% ($P = 0.01$). The PRA decreased from 1.07 (SD 0.87) to 0.54 (SD 0.27) $ng \cdot ml^{-1} \cdot h^{-1}$ and diuresis increased from 1.31 (SD 0.47) to 2.47 (SD 1.04) $ml \cdot min^{-1}$ ($P = 0.04$). Plasma aldosterone concentration was unchanged [99.6 (SD

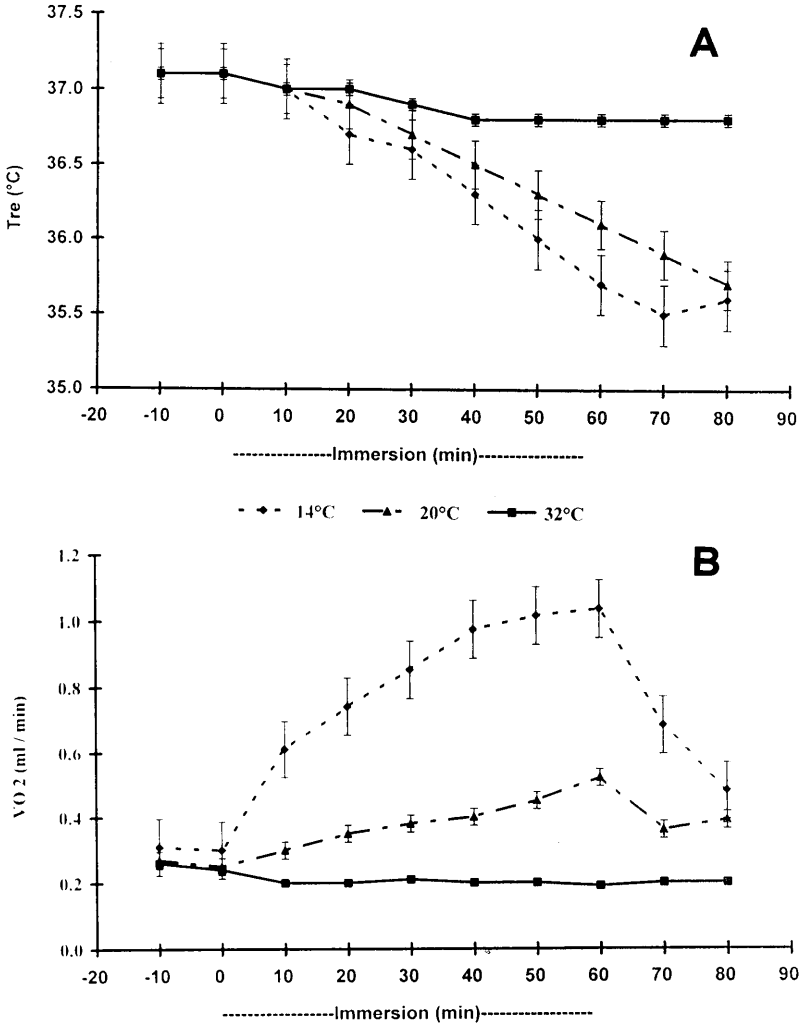

Fig. 1 Changes in rectal temperature (T_{re}; **A**) and metabolic rate ($\dot{V}O_2$; **B**) in subjects immersed for 1 h in water of different temperatures (32°C, 20°C and 14°C). Means and SEM

48.7) compared to 92.8 (SD 31.7) pg · ml^{-1}]. Cortisol concentration decreased from 163.1 (SD 44.0) to 143.7 (SD 49.9 nmol · l^{-1} ($P = 0.05$).

Cold water immersion (14°C) lowered rectal temperature from 37.3 (SD 0.32) to 35.6 (SD 0.19) °C and increased metabolic rate by about 350%. Heart rate was increased by 3 beats · min^{-1}, systolic pressure from 113 (SD 7.9) to 121 (SD 4.9) mmHg ($P = 0.01$) and diastolic pressure from 77 (SD 4.7) to 83 (SD 5.0) mmHg ($P = 0.03$). Plasma noradrenaline concentration increased from 1.17 (SD 0.49) to 6.20 (SD 4.42) pmol · ml^{-1} ($P = 0.003$). Plasma concentration of dopamine also increased from 0.16 (SD 0.03) to 0.73 (SD 0.06) pmol · ml^{-1}, while adrenaline concentration was unchanged (Fig. 5). Diuresis increased markedly by 163% ($P = 0.001$), while PRA was lowered from 0.85 (SD 0.62) to 0.46 (SD 0.32) ng · ml^{-1} · h^{-1} ($P = 0.015$). Plasma aldosterone concentrations increased from 77.0 (SD 30.1) to 95.6 (SD 33.2) pg · ml^{-1} ($P = 0.014$). Cortisol concentrations decreased slightly as at higher temperatures.

It was evident that a 1-h head-out immersion in thermoneutral water lowered heart rate and systolic and diastolic pressures, while leaving rectal temperature and metabolic rate unchanged. Vasomotor changes occurred within the first 10 min of immersion and then became stabilised during the remainder of the immersion. In contrast to immersion in thermoneutral water, immersion in 20°C water lowered rectal temperature, raised metabolic rate slightly, but maintained lowered heart rate and blood pressures.

The data also indicated that 1-h immersion in cold water (14°C) decreased body temperature by about 1.7°C, which induced a stimulation of thermogenesis and activation of the sympathetic nervous system, as evident from the increased noradrenaline concentrations. In accordance with the increased production of noradrenaline, heart rate and blood pressured increased, when compared with subjects immersed in thermoneutral water.

Water immersion was always accompanied by an increased diuresis, which was potentiated by cold. This was reflected in a decreased PRA. A decrease in PRA was accompanied by a decrease in aldosterone concentration at thermoneutral temperatures only. Cortisol production was not influenced either by water immer-

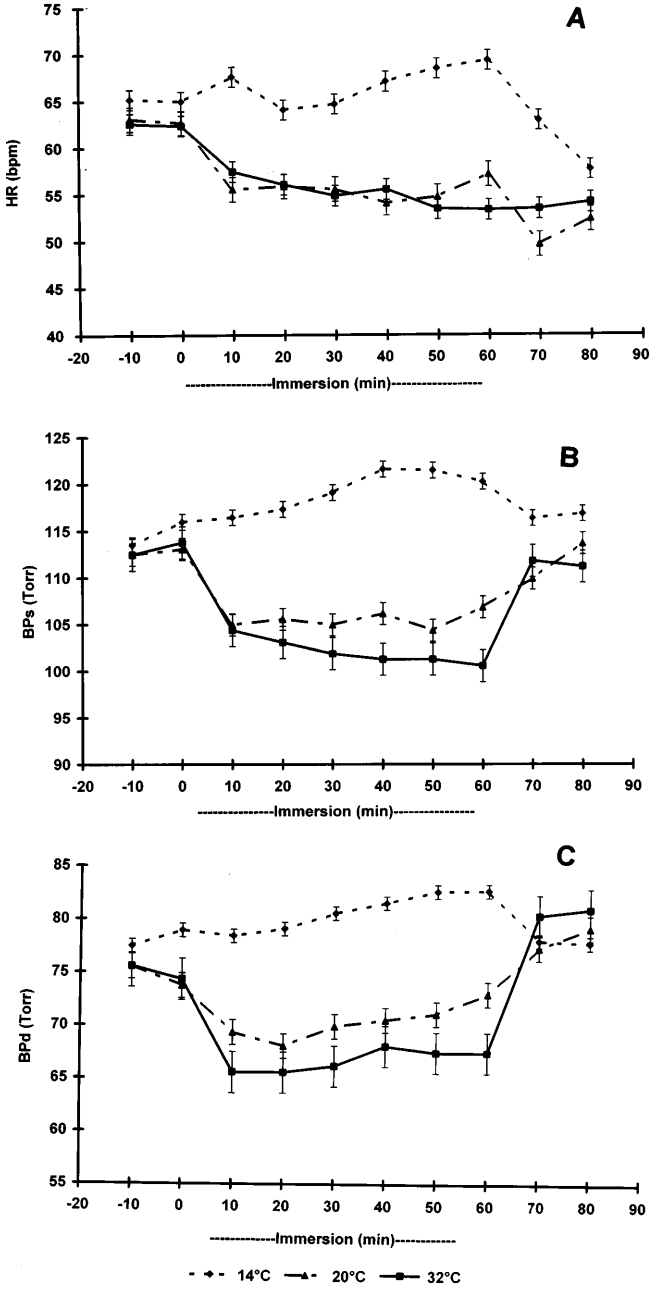

Fig. 2 Changes in heart rate (*HR*; **A**), systolic blood pressure (BP_s; **B**) and diastolic blood pressure (BP_d; **C**) in subjects immersed for 1 h in water of different temperatures (32°C, 20°C, 14°C). Means and SEM

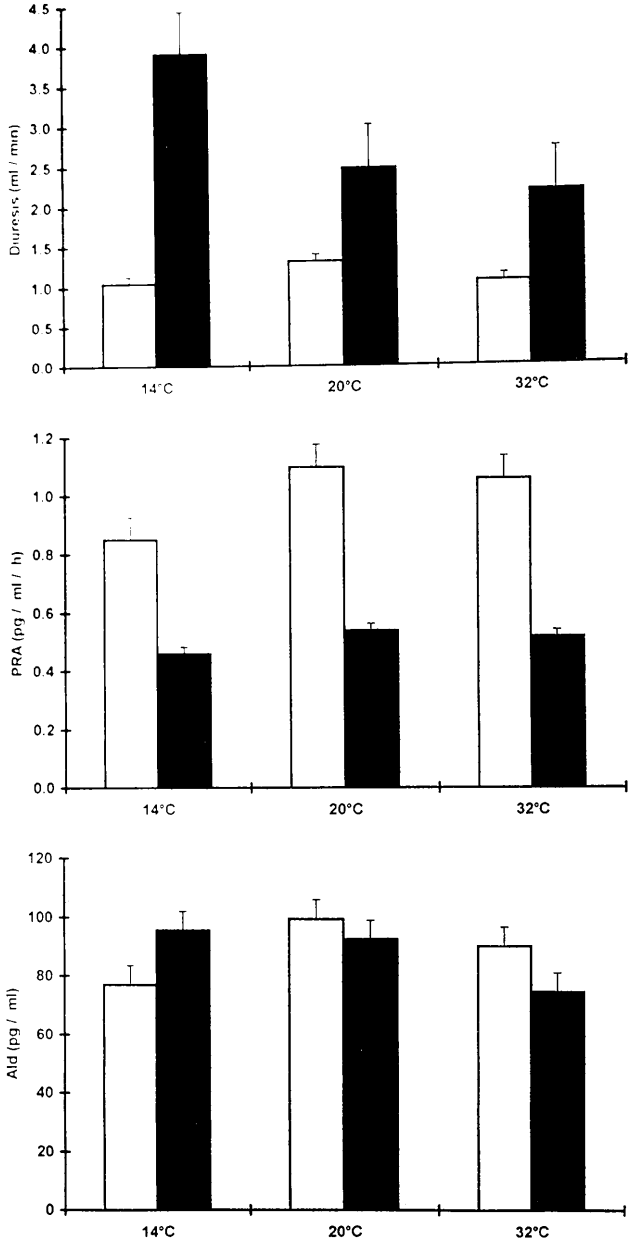

Fig. 3 Diuresis (**A**), plasma renin activity (*PRA*; **B**) and plasma aldosterone (*Ald*) concentrations (**C**) in subjects prior to and after 1-h immersion in water of different temperatures (32°C, 20°C, 14°C). Means and SEM

sion at neutral temperatures or by cold, but rather tended to decrease during all exposures.

The changes in diuresis and PRA were probably due to the effect of hydrostatic pressure during immersion rather than to the effect of cold. Metabolic rate and activity of the sympathetic nervous system were stimulated by cold rather than by water immersion. Cardiovascular functions were affected both by cold and water immersion, but in opposing ways. Intense cold stimuli increased heart rate and blood pressure, while thermoneutral water immersion induced bradycardia and decreased blood pressure. Part of the increase in heart rate may however, have been due to shivering rather than due to cold per se.

Discussion

To our knowledge no previous attempt has been made to compare the effect of immersion in differing water temperatures on cardiovascular responses and hormone production of humans at rest. It should be taken into

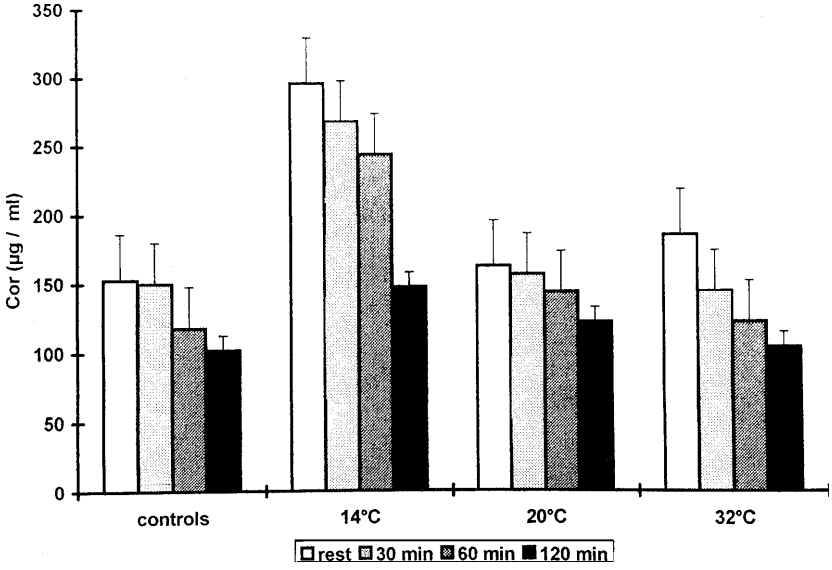

Fig. 4 Plasma cortisol concentrations (*Cor*) in subjects prior to and after 1-h immersion in water of different temperatures (32°C, 20°C, 14°C). *Controls* Subjects in air at thermoneutral conditions prior to and during the course of the experiment. Means and SEM

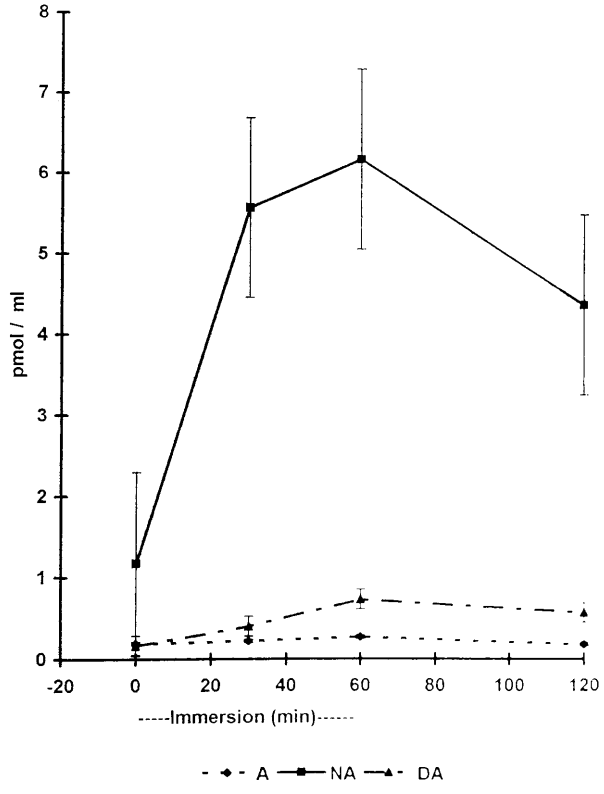

Fig. 5 Changes in plasma catecholamine concentrations during and after immersion into cold water (14°C). [*A*] Adrenaline (●) [*NA*] noradrenaline (■) [*DA*] dopamine concentrations (▲)

consideration, however, that immersion in 14°C water induced shivering, which could be considered as light exercise. Several experiments have been performed on subjects exercising in water at different temperatures (Christie et al. 1990; Connelly et al. 1990; Galbo et al. 1979).

Data presented in our paper would suggest that water immersion at neutral temperatures, which would stimulate mainly baroreceptors, and cold exposure, which would stimulate mainly thermoreceptors, activate different regulatory systems and different effector mechanisms. Thermoneutral water immersion induced bradycardia and decreased blood pressure, while strong cold stimuli increased heart rate and blood pressure. Since an increased concentration of noradrenaline in cold immersed subjects was also observed in this study, it is tempting to conclude that immersion in thermoneutral water relatively strengthened the influence of the parasympathetic nervous system, while immersion in cold water overcame this effect by increasing the sympathetic tone. These findings have been supported by data in the literature documenting unaltered (Epstein et al. 1983; Weihl et al. 1981), or even decreased (Norsk et al. 1990) plasma noradrenaline concentrations during immersion at thermoneutrality and increased concentrations of this substance during cold water immersions (Buhring and Spes 1979; Hiramatsu et al. 1984; Janský et al. 1996; Johnson et al. 1977; Weiss et al. 1988).

Data in the literature have also indicated that plasma adrenaline concentrations were lowered, while plasma dopamine concentrations were increased during cold exposure (Keatinge and Evans 1961; Weiss et al. 1988). We also found that plasma concentration of dopamine was significantly elevated during cold water immersion, while the adrenaline concentration was unchanged. Thus, cardiovascular changes and a part of the metabolic increase in the cold (nonshivering thermogenesis Janský et al. 1997) contributes to the increased production of noradrenaline and dopamine. Changes in cardiovascular functions due to cold water immersion have been discussed in detail in an earlier paper (Janský et al. 1996). It was concluded that an increase in stroke volume may be the reason for the relatively small increase in heart rate in cold immersed subjects.

In accordance with data in the literature (Epstein et al. 1975; Epstein and Saruta 1971; Nakamitsu et al. 1994), the present study also showed that thermoneutral water immersion decreased PRA and increases diuresis. This may be due to an activation of renal baroreceptors by increased splanchnic blood flow as a consequence of an increase in hydrostatic pressure. The renal baroreceptor mechanism has been suggested to involve stretch-sensitive cells in the arterial wall, which may inhibit renin release when renal perfusion pressure increases (Sköott and Jenssen 1993).

Diuresis was further potentiated by cold, while the decrease in PRA was independent of water temperature. The observed decline in PRA does not fully agree with data of Wittert et al. (1992), who have found an increase in PRA 30 min after exposure to cold air (4°). No change in lowered PRA, observed in our study on cold exposed subjects, is in contrast with the known effect of sympathetic tone on renin release (see Gordon et al. 1967). These findings are difficult to interpret. The explanation could be that activation of baroreceptors by an increased splanchnic volume is a very strong stimulus which is able to prevent the action of catecholamines on PRA release. Low PRA during cold immersion may also be due to an increased plasma concentration of dopamine. It has been found that the proximal tubules in the kidney can decarboxylate circulating L-DOPA, producing dopamine, and that activation of dopamine production reduces PRA in humans (Keeton and Cambell 1980). Also it has been shown that an increase of atrial natriuretic peptide can inhibit renin secretion (Sköott and Jenssen 1993). This suggestion has been supported by our finding of a twofold increase in natriuresis during cold immersion (Šrámek et al. 1993).

In accordance with data in the literature, our results would indicate that aldosterone concentrations were slightly decreased in water at 32°C, unchanged in water at 20°C, but slightly increased in water at 14°C. Hiramatsu et al. (1984) have also found increased aldosterone and cortisol concentrations in subjects exposed to moderate cold (4°C for 10 min) or severe cold (immersion of hands to 0°C for 10 min).

The reasons why the lowered PRA did not inhibit aldosterone production and, vice versa, why increased aldosterone production in cold water was not reflected by changes in PRA, are not clear. Evidently, there exist several mechanisms controlling aldosterore production, besides the renin–angiotensin system. One possibility is that the increase in plasma aldosterone concentrations in the cold may have been due to stress-induced adrenocoticotropic hormone (ACTH) secretion as has been suggested by Himathongham et al. (1975). Wittert et al. (1992), however, have observed no significant increase in plasma ACTH concentration in response to cold air (4°C for 30 min) and a significant fall in plasma cortisol concentrations during the first 15 min of exposure. Our unpublished results have indicated that ACTH production is lowered in cold immersed subjects (Šrámek, unpublished).

In our previous study (Šrámek et al. 1993) we have observed a significant increase in plasma potassium concentration during cold water immersion, which may also have contributed to the increased aldosterone production as suggested by Himathongham et al. (1975). A possible explanation of this effect may come from the observation of Thulesius and Yousif (1991), in which cold-induced vasoconstriction in cutaneous veins has been associated with inhibition of Na^+, K^+- ATPase, (adenosinetriphosphatase). Inhibition of the Na^+, K^+-ATPase and the subsequent shift of extracellular water into cells may result in haemoconcentration, as has been observed in our previous study (Šrámek et al. 1993).

Our observation that cortisol concentration decreased both during immersion in thermoneutral and in cold water seems to be in contrast to the common view that environmental stresses stimulate activity of the pituitary – adrenal axis. This finding is in agreement with data of Leppäluoto et al. (1988), however, who have also demonstrated a significant fall in serum cortisol concentration in subjects exposed to cold air (10°C) for 2 h. The observed small decrease of plasma cortisol concentration was likely to have been due to the normal diurnal rhythm in cortisol production, because at no time was there any significant difference between values at rest and those after immersion in either cold or thermoneutral water. On the other hand, Wilson et al. (1970) have not found a diurnal decrease of plasma cortisol concentrations after a 3-h cold exposure to air temperatures of −5 to +2°C. Their experiment was performed in the late afternoon, while we exposed out subjects to cold water in the morning. The reason why the values of cortisol concentrations in subjects exposed to 14°C water are slightly higher than in those exposed to other temperatures is not clear.

In conclusion, as was evident from the cortisol concentrations, water immersions and cold did not induce activation of the hypothalamus-pituitary-corticoadrenal axis, which typically occurs in subjects under stress conditions. Immersions inhibited PRA, while increasing aldosterone concentration. On the other hand, cold activated the sympathetic nervous system, enhancing dopamine and noradrenaline concentration. This was reflected in changes in vasomotor activity, heat production, diuresis and in distribution of body fluids. We would suggest that cold lowers aldosterone concentrations, but does not influence PRA.

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