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The Effect of Different Water Temperatures on the Release of the Atrial Natriuretic Factor (ANF) During "Head Out Water Immersion (HOI)"

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Summary: Head-out water immersion (HOI) results in a redistribution of blood volume with a relative central hypervolemia, which is the cause of increased secretion of the atrionatriuretic factor (ANF). This study was conducted to examine the role of different bath temperatures for the stimulation of ANF secretion. Twelve healthy subjects were immersed in 4 water baths (24, 28, 35 and 39 °C) for 40 min in a randomized fashion. Plasma ANF was determined by radioimmunoassay following extraction. The baths led to a significant increase in plasma ANF. Maximal increments were the highest in hot baths. This data suggests that heat induced changes in circulation may modulate the secretion of ANF. The importance of heart rate is being discussed.

The atrial natriuretic factor (ANF) is a polypeptide which is synthesized and stored in the secretorial granula of the atria (1). It probably also affects the regulation of the extracellular water balance and blood pressure, without a final explanation of its physiological significance. Hypervolemia is one of the most important factors for the release of the hormone (2).

In addition water immersion has effects on blood volume and many other physiological reactions (3). An important consequence of the hydrostatic pressure is a change in the blood volume distribution within the low pressure system with preference on the intrathoracic vascular system. Central blood volume and central venous pressure increase, it is therefore not surprising that HOI results in ANF release into the circulation (4–9).

Water immersion has proved to be a valid model for the study of blood volume regulation and is generally carried out with thermoneutral temperatures (about 34–36 °C). Yet there are varying water temperatures with different forms of therapeutical baths, which can affect significant cardiovascular, hormonal and metabolic reactions and may influence the conditions of ANF-secretion by changing the central haemodynamics. Therefore we investigated the effects of cool and hot baths on plasma concentration of ANF.

Methods

The study included 12 healthy subjects (4 females, 8 males with mean age of 26 ± 2 years). They had no specific nutrition. Informed consent was obtained in respect to the conditions of the declaration of Helsinki.

In a randomized fashion the subjects took four baths (HOI) with a one week interval; temperatures were 24, 28, 35 and 39 °C respectively. A sitting position was kept for 40 minutes before – during – and after the baths. The experiments were carried out in the afternoon. The subjects had to drink 150 ml of water per hour in order to guarantee a standardized hydration.

Venous blood samples were drawn before 10, 20 and 40 minutes after the start as well as 20 and 40 minutes after the end of immersion. The blood samples were immediately transferred into precooled containers (1 mg/ml Na-EDTA, 500 KIV/ml Aprotinin); the plasma was deep-frozen at –80 °C after centrifugation. ANF was determined with a RIA within extracted plasma (10). In addition heart rate, blood pressure and sublingual temperatures were registered.

The paired t-test was used for statistical evaluation of changes in plasma ANF. Values of $p < 0.05$ were considered significant.

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Results

The mean values of ANF measurements for different baths are shown in Figure 1 on a per cent basis. The single values before and after the baths ranged from 1.9–8.4 ($x = 4.7 \pm 0.25$) respectively 2.6–17.2 ($x = 7.1 \pm 0.68$) f mol/ml plasma. In all the series there was an increase in ANF concentration in the blood as a result of water immersion. At the end of the water immersion (40 min) there were very similar values for the groups of 24, 28 and 35 °C. Differences during the time course were not significant.

There was a stronger secretion of ANF in the hyperthermic bath (39 °C). Already after 20 minutes a significantly higher concentration was observed in comparison to the other groups. After 40 minutes 224% of the initial value were achieved, which equals three times the value of the thermoneutral immersion values. The ANF levels quickly decreased after ending the immersion phase, although not achieving the basal values after 40 minutes.

Haemodynamics as well as sublingual temperatures showed the following changes: In cool baths (24 and 28 °C) there was a slight increase in blood pressure and decrease of heart rate as well as a reduction of the sublingual temperatures (1.1 resp. 0.6 °C). The hot baths (39 °C) led to a decrease of the diastolic blood pressure and an elevation of heart rate from 74 up to 103 min–1; the sublingual temperature increased by 1 °C.

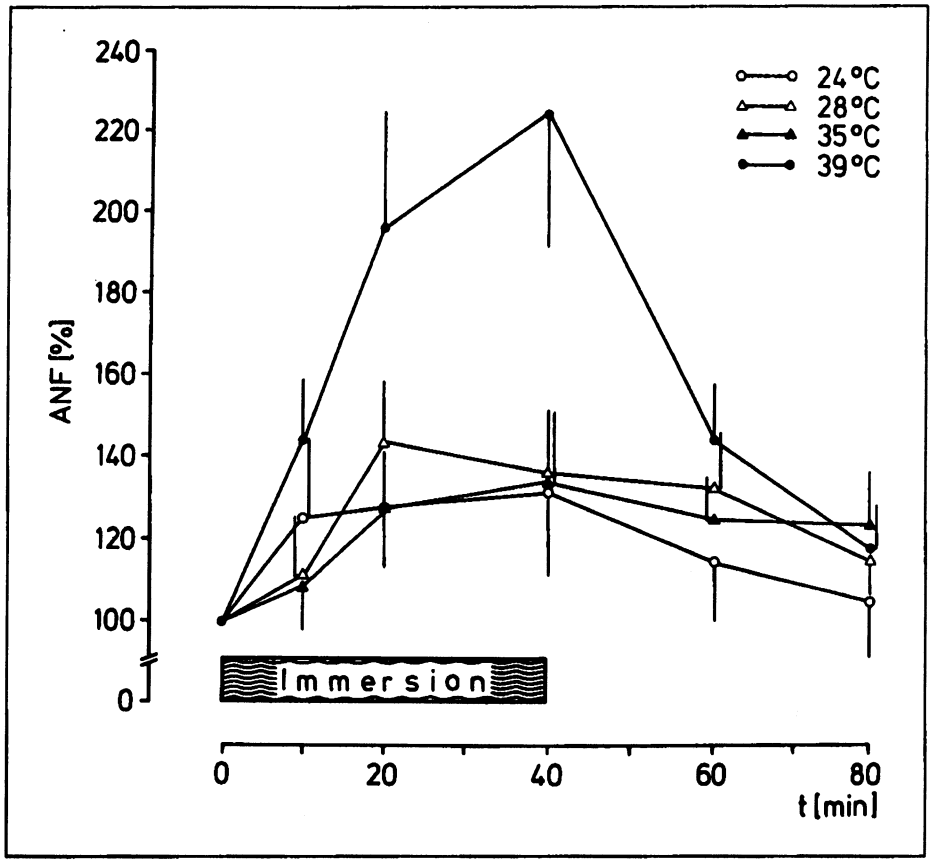

Fig. 1. Changes of the atrial natriuretic factor (ANF) before, during and after baths with different temperatures.

Discussion

The increased hydrostatic pressure during water immersion (HOI) on the peripheral vessels leads to a preferential distribution of the blood volume into the central intrathoracic vessels. This immersion related central hypervolaemia results from an increase of blood volume of about 700 ml of blood and increases the central venous pressure by approximately 15 mm Hg (3). This led to a stimulus in the regulation of the extracellular fluid volume (11). The atrial natriuretic factor (ANF) is known to be a hormone for blood volume control. Experiments have proved the expansion of blood volume with an increase of the right atrial pressure causing a release of ANF into the circulation (2). For this reason tension stimuli of the atrial muscle might be the triggering factor.

In addition heart rate is also considered to be important (12–14). The increase of central venous pressure during water immersion results in an acute volume stress. Under these circumstances a reinforced release of ANF has been proved. An increase in plasma concentration of 27% within 10 minutes was found by *Gerbes* et al. (6, 7). *Anderson* et al. (4), *Epstein* et al. (5), *Ogihara* et al. (8) and *Pendergast* et al. (9) confirmed the effects of water immersion on ANF.

Differences in thermoneutral water temperatures may show various thermophysiological responses depending on intensity and duration of exposition. Hot and cold stimuli especially show an immediate reaction on haemodynamics, metabolic and endocrine functions. The release of ANF could be influenced under these circumstances.

Exposition to cold leads to an increase of mean arterial blood pressure as a consequence of complex circulatory regulation with peripheral vasoconstriction and an increase of vascular total peripheral resistance (TPR). Our data showed similar results under cool water immersion. *Keatinge* and *McCance* (15) assumed that an increase in peripheral venous pressure, as a result of cold exposition, led to a compression of the capacitative vascular system with an increase in central venous pressure. No evident data is available measuring central blood volume and central venous pressure under cool water immersion. Our data shows no difference in the release of ANF concerning 24 °C and 28 °C in comparison to thermoneutral

baths. The increased atrial and venous tonus within cool water might not allow a further centralization of blood volume.

For the immersion under hot conditions, however, our results demonstrate a stronger rise of ANF concentration in the plasma. *Asanuma* et al. (16) have also found relatively high values after baths of 10 minutes (40 °C). We have to consider several mechanisms concerning the thermoregulative characteristics in hot baths. With rising core temperature in the bath we can especially state an increase of heart rate, stroke volume, cardiac output and a reduction of peripheral resistance (17). The regulatory adjustment of the circulation typically leads to an increased skin perfusion with a reduction in the splanchnicus region at the same time.

The mean sublingual temperature increased about 1 °C during baths of 40 minutes (39 °C). Higher core temperature might influence the ANF secretion, since a thermic dependence has been described in the isolated atrial preparation of the rat (18). At the same time the strong sympathicotonic reaction under hot water conditions leads to an increased release of catecholamines. This might be a contributing factor to higher release of ANF, because it is reported a response to the direct effect of adrenergic stimulation (19–23).

Supposing that the atrial pressure is most important for the ANF stimulation, we have to consider that the haemodynamics correspond with an increase in atrial pressures. In contrast to this assumption, there is a reduction in atrial pressures if hot exposure takes place with air as the environmental medium (24). There are fewer results of atrial pressure measurements in warm baths. In warm baths (38 °C, 15 min) there were no alterations of central venous pressures compared to thermoneutral conditions (25). Other experiments, however, have shown a higher cardiac output (17). Obviously, the hydrostatic pressure prevents a peripheral venous pooling and improves the venous reflow. Possibly there is a mobilization of blood volume from the splanchnicus region, where the increase of vascular tonization also involves the venous vessels (24).

Further discussions also stressed the importance of heart rate for the ANF release. Higher plasmatic ANF levels have been observed in patients suffering from paroxysmal tachycardia (12–14), as well as with pacemaker induced tachycardia (26). Furthermore a rise of stimulation rate at the isolated atrial preparation led to an increase of ANF release (27).

However, we have to ask for the interdependance of heart rate and atrial pressure. Patients with supraventricular tachycardia showed an increase of ANF plasma concentration as well as a rise of mean right atrial pressure (28, 29). There are similar effects with pacemaker tachycardia in a rabbit experiment (30). Corresponding with results in patients, an ANF effect has not been achieved by a mere increase in heart rate (31). In a dog experiment an increase of heart rate during low atrial pressures did not correspond with an increased ANF release (32).

In a recent study these authors tried to differentiate the influence of atrial pressure, atrial distension and heart rate. They were able to prove a higher correlation between ANF plasma concentration and the systolic atrial pressure in contrast to mean atrial pressure. The highest concentration coefficient has been found for the heart rate and systolic atrial pressure product. It was concluded that within the determinants for ANF stimulation, the atrial distension is less than the increase in actual wall tension. In these experiments the influence of diastolic atrial filling is also evident. With an elevated diastolic atrial pressure, the atrial systolic pressure is potentiated, and only with high atrial pressure can heart rate play a major role in ANF stimulation.

These findings might explain the relatively high ANF concentration we found under hot bath conditions. It can be hypothezised, that the hydrostatic pressure allows no more thermically dependent venous pooling in the periphery, and thus guarantees a high cardiac preload. Thus the increased heart rate (74 min–1 to 103 min–1 in a 39 °C (bath) in combination with a more rapid rise in atrial systolic pressure could have a substantial effect on ANF secretion.

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Conference Calendar

Date	Place	Name	Office (Information
1991			
September 6 – 8	Copenhagen, Denmark	2nd Scientific meeting of the Scandinavian Medical Society of Paraplegia	Centre for spinal cord injured, Rigshospitalet, TH 2002, DK-2100 Copenhagen, Denmark
October 23 – 27	Düsseldorf, Germany	REHA '91 Exhibition and Congress – Rehabilitation Aids for Handicapped Persons	NOWEA-Rehab '91, Postfach 32 02 03, Stockumer Kirchstraße 61, W-4000 Düsseldorf 30, Germany
November 23	Vienna, Austria	Annual meeting of the Austrian Society PMR	Prim. Dr. Rathkolb, Hanusch-KH, Heinrich-Collinstraße 30, 1140 Vienna, Austria
December 8 – 12	Vienna, Austria	Second Paneuropean Congress of Neurology	Admin. Secr. Wiener Medizinische Akademie für Ärztliche Fortbildung und Forschung, Alser Straße 4, A-1090 Wien/Vienna Austria
1992			
September 16 –21	Bad Gottleuba Germany	World Conference PMR	Prof. Dr. Reinhold, Kliniksanatorium, Ernst Thälmann Straße 89, D-8302 Bad Gottleuba
September 26 –28	Dublin, Ireland	Int. Physiotherapy Conference	School of Physioth., Trinty College, PO Box 814, James's Street, Dublin, Ireland