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Effect of prolonged physical exercise on fluid regulating hormones

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Summary. Sixteen well-trained young men performed a test marathon to study the behaviour of atrial natriuretic peptide (ANP) and its second messenger cyclic guanosine monophosphate (cGMP) in relation to changes in plasma volume (PV) and plasma proteins, arginine vasopressin (AVP), renin, aldosterone, potassium and sodium. Blood samples were drawn under standardized conditions before and immediately after the run, as well as 3 h and 31 h after the run. Directly after the run, a two-and-a-half fold increase of plasma ANP and a twofold increase of plasma cGMP level were found, whereas PV decreased significantly by 7.4%. At this time renin-, aldosterone- and AVP-secretion were much stimulated. Thirty-one hours after the run, PV was markedly greater (10%) than before the race, whereas plasma proteins had returned to pre-exercise values. The ANP and cGMP were not significantly altered compared to the pre-race values. We have concluded that ANP and the other volume-regulating hormones may play an important role during and immediately after prolonged physical exercise but not in the longer recovery period. It seems that an influx of plasma proteins into the vascular space is responsible for the increased PV at this time.

Key words: Prolonged physical exercise - Atrial natriuretic peptide - 3'-5'-guanosine monophosphate - Vasopressin - Renin - Aldosterone

Introduction

Prolonged physical exercise is known to have strong influences on the homeostasis of body water and electrolytes (Pivarnik et al. 1984; Refsum et al. 1973; Röcker et al. 1989; Saltin and Stenberg 1964). From previous studies it is known that the renin-angiotensin-aldosterone-system and arginine vasopressin (AVP) is deeply involved in the regulation of fluid homeostasis [especially in the homeostasis of plasma volume (PV)] during and after prolonged physical exercise (Convertino et al. 1980; Newnmark et al. 1976; Röcker et al. 1989; Wade et al. 1981). Some years ago a peptide secreted from the atria was discovered with strong diuretic, natriuretic and vasodilating functions, which was named atrial natriuretic peptide (ANP) (De Bold et al. 1981). A large body of evidence suggests that this peptide plays a primary role in the regulation of water and electrolyte balance (Müller et al. 1986).

However, knowledge concerning the role of ANP and its second messenger, guanosine 3',5'-cyclic monophosphate (cGMP), with respect to prolonged physical exercise is scant (Lijnen et al. 1987). The aim of this investigation was to study simultaneously both the role of ANP and other volume-regulating hormones and plasma proteins for the homeostasis of PV after prolonged physical exercise. Preliminary results of this study were presented at the 31st Deutscher Sportärztekongreß in Hannover (Schultes et al. 1988).

Methods

Subjects. Sixteen healthy male marathon runners aged 23–40 (mean 29) years with a height of 173–196 (mean 180) cm and a mass of 61.5–77.5 (mean 67.5) kg agreed to participate in the study after being given a detailed description of the procedure and potential complications. All of them had been involved in regular running for several years.

Protocol. The participants were asked not to take any medication, to keep to their normal dietary habits for a period of 2 weeks before the test marathon and to abstain from physical exercise or strenuous physical work in the 24-h period before the run.

The subjects arrived in the laboratory at 8 a.m. The specially organized test marathon races (38.4 and 37 km, respectively) took place in Berlin over 2 days commencing at 9 a.m. The temperature was −4° C on the 1st and −5° C on the 2nd day, the relative humidity was 63% and 70%, respectively. The subjects were asked to run within 90%–95% of their best performance. All the runners completed the races in average times of 2 h 46 min (range: 2 h 40 min–2 h 53 min) and 2 h 38 min (range: 2 h 24 min–2 h 50 min), respectively.

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During and at the end of the run fluid intake was recorded and related to the body fluid loss. Five runners did not ingest any fluid or electrolytes during the run, 1 runner ingested 20 ml, 3 runners 50 ml, 6 runners 200 ml each and 1 runner 300 ml of tea without electrolytes. Fluid and food intake were not controlled during the recovery period.

Blood samples were collected by repetitive venipuncture with a butterfly needle from different cubital veins 30-min before the run and within 1 min after finishing the run, and 3 h and 31 h later. Blood was taken without stasis and serum or plasma was obtained from whole blood in a refrigerated centrifuge and stored in aliquots at −80° C until tested.

Analyses. Concentrations of plasma AVP were determined after extraction by radio-immunoassay according to Möhring's technique (personal communication). The intra-assay coefficient of variation (CV) for this method was 15% (1–5 pg·ml^{-1}) and 10% (5–10 pg·ml^{-1}), respectively, for ten seperately extracted samples. Extraction was performed with acetone and petroleum benzene. The overall recovery of (I^{125})-AVP was 70%. The antisera cross-reaction with lysine vasopressin was less than 1%. The limit of detection for this assay was about 1 pg·ml^{-1}.

Serum concentrations of aldosterone were analysed twice by radio-immunoassay technique using a commercial kit obtained from Diagnostic Product Corporation, Los Angeles, USA. The intra-assay CV was less than 5%, the sensitivity being less than 15 pg·ml^{-1}. Cross-reaction with other corticosteroids was less than 0.06%.

The ANP was measured by radio immunoassay after extraction of ethylenediaminetetraacetate (EDTA) plasma with C_{18}-cartridges (Lang et al. 1985; Weil et al. 1986). The cGMP was determined in ethanol extracted EDTA plasma samples by radio-immunoassay as previously described (Heim et al. 1988). Renin was analysed by a immunoradiometric technique, by Pasteur, for active renin. The intra-assay CV was less than 10.5%, the sensitivity being about 6 pg·ml^{-1}.

Serum sodium and potassium concentrations were measured by flame photometry, haemoglobin was determined spectrophotometrically and packed cell volume by using a microcapillary technique. The CV was less than 1% ($n=20$) for these methods. The percentage changes in plasma volume (%ΔPV) were calculated from resting and post-exercise haemoglobin and packed cell volume measurements (Straus et al. 1951). The plasma protein concentration was determined by the Biuret method. This method had a CV of less than 3%.

Statistics. Statistical calculations were carried out with the non-parametric Wilcoxon test for matched pairs because it was not known whether the data had a gaussian distribution. For that reason the results are expressed as medians rather than arithmetic means. With respect to the variation, percentile values (P_{25}–P_{75}) have been given rather than mean and standard deviation. The zero hypothesis was rejected when $P<0.05$.

Results

The results of this study are presented in the Table 1.

Body mass

A mean body mass loss of 3.0 kg was observed at the end of the run.

Table 1. Plasma volume, concentrations of plasma proteins, electrolytes (sodium, potassium), and fluid-regulating hormones before (t_0), immediately after (t_1), and 3 h (t_2) and 31 h (t_3) after a test-marathon run. The values given are medians ($\tilde{x}$) or differences between medians ($\Delta\tilde{x}$) the 25th and 75th percentiles. The first row gives the percentage changes in plasma volume related to t_0.
Significance levels of differences related to t_0: * $0.05>P\geq0.01$; ** $0.01>P>0.001$; *** $0.001>P$

	t_0 pre-exercise			(t_1–t_0) immediately after exercise			(t_2–t_0) 3 h after exercise			(t_3–t_0) 31 h after exercise		
	$\tilde{x}$	P_{25}	P_{75}	$\tilde{x}$	P_{25}	P_{75}	$\tilde{x}$	P_{25}	P_{75}	$\tilde{x}$	P_{25}	P_{75}
%Δ, Plasma volume				−7.4***	−10.3	−2.3	+2.5	−0.3	+6.2	+10.0***	+2.6	+14.0
Plasma proteins (g·dl^{-1})	7.82	7.38	8.19	+0.85***	+0.23	+0.97	+0.05	−0.12	+1.19	−0.10	−0.58	+0.11
Sodium (mmol·l^{-1})	142	139	142	+5.5***	+3.3	+7.0	+1.5**	+0.3	+4.0	+0.5*	−1.0	+2.0
Potassium (mmol·l^{-1})	4.13	4.0	4.4	+0.32**	−0.27	+0.47	+0.09	+0.07	+0.45	−0.05	+0.28	+0.29
Osmolality (mosmol·kg^{-1}) ($n=9$)	287	281	288	+10***	+7	+14	+4*	0.0	+7	+1	−3	+4.5
Arginine vasopressin (pg·ml^{-1})	1.35	0.65	1.60	+5.53***	+3.79	+7.71	+0.80**	+0.40	+1.25	+0.35*	0.0	+0.60
Renin (pg·ml^{-1})	12.8	7.98	17.5	+67.8***	+32.5	+91.3	+16.6***	+9.38	+29.1	−0.09	−5.40	+0.97
Aldosterone (pg·ml^{-1})	98.5	71.8	163.8	+502***	+267	+806	+16.0*	+4	+87	−23.0***	−59	−2
Atrial natriuretic peptide (pg·ml^{-1})	51.0	37.5	53.5	+67.0***	+57.8	+116.3	−10.0	−20.0	+6.0	+7.5	−11.8	+33.0
Cyclic guanosine monophosphate (pmol·ml^{-1})	4.59	4.18	5.01	+4.17***	+3.02	+7.17	−0.67**	−1.15	−0.14	+0.36	−0.39	+0.64

Heart rate

Mean resting heart rate (f_c) of 60 beats·min^{-1} increased significantly ($P<0.001$) to 90 beats·min^{-1} at the end of the run.

Systolic blood pressure and diastolic blood pressure

Systolic blood pressure (BP_S) had increased significantly ($P<0.01$) at the end of the run by a mean of +10 mm Hg compared to resting values of 125 mm Hg. Diastolic blood pressure had increased by an average of +7.5 mm Hg but the difference was not significant.

Percentage changes of PV

The PV had decreased by 7.4% after the race. No significant difference was found 3 h later, whereas 31 h after the run an increase of 10.0% was observed.

Percentage changes of plasma protein concentration

Plasma protein concentration had increased by 9.8% immediately after the race. No significant changes were found 3 h (+0.6%) and 31 h (−2.6%) after the run.

Electrolytes

Sodium and potassium concentrations had increased at the end of the run (+5.5 mmol·l^{-1}, and +0.32 mmol·l^{-1}, respectively). Three hours later the sodium concentration was still elevated (+1.5 mmol·l^{-1}) whereas potassium showed no significant difference to the control value. Thirty-one hours after the race sodium concentration was still elevated by 0.5 mmol·l^{-1} compared to the resting value, whereas potassium had returned to the pre-exercise value.

Arginine vasopressin

The AVP showed a marked increase (+5.53 pg·ml^{-1}) immediately after the race. Three hours and 31 h later increases of +0.80 pg·ml^{-1} and 0.35 pg·ml^{-1}, respectively, were still observed.

Renin

Renin concentration had increased by +67.8 pg·ml^{-1} immediately, and by +16.6 pg·ml^{-1} 3 h after the race. Thirty-one hours after the race the renin concentration had returned to the pre-exercise value.

Aldosterone

Aldosterone had increased by +502 pg·ml^{-1} immediately after the race, remained elevated 3 h after the race (+16.0 pg·ml^{-1}) and had decreased by −23.0 pg·ml^{-1} 31 h after the run.

Atrial natriuretic peptide

The ANP concentration was markedly increased immediately after the run (+67 pg·ml^{-1}) and showed no significant changes 3 h and 31 h after the race.

Cyclic guanosine monophosphate

The cGMP concentration had increased by 4.17 pmol·ml^{-1} immediately after the race. Three hours and 31 h after the run no difference was found compared to the pre-exercise value.

Discussion

The results of the present study showed a two-and-a-halffold increase in plasma ANP concentration and a twofold increase in plasma cGMP concentration directly after the race. This result is in accordance with data from others (Lijnen et al. 1987). After short maximal exercise performed on a cycle ergometer, it has also been demonstrated that plasma ANP concentrations increased fivefold (Bollerslev et al. 1987) or three-and-a-halffold (Somers et al. 1986). This increase in plasma ANP concentrations correlated with the heavy workload performed on the cycle ergometer, with the increase in f_c, BP_s and the plasma noradrenaline concentration (Saito et al. 1987; Tanaka et al. 1986).

Examinations have also shown that the increase in f_c caused by atrial pacing or paroxysmal atrial tachycardia is sufficient to increase ANP (Espiner et al. 1985; Schiffrin et al. 1985). However, this result could not confirmed by Haufe et al. (1987). It is debatable whether the increase of atrial pressure be or head-down tilt also are stimuli for the secretion of ANP. Garcia et al. (1986) reported that chronic subcutaneous infusion of isoproterenol produced a threefold increase in plasma ANP levels and an increased natriuresis in rats. Therefore, the exercise-induced increase of catecholamines (Hartley et al. 1972) may also contribute to the increase in ANP plasma levels. Since strenuous physical exercise causes changes in multiple parameters of the cardiovascular system, e.g. increase in f_c and arterial and atrial pressure, it is difficult to identify the main reason for ANP secretion.

It is remarkable that the cardiovascular load in marathon racing is more important than the volume load in releasing ANP. The marked decrease of PV after the race would be expected to depress a hormone that has a potent diuretic action. Under different circumstances the marked decrease of PV usually leads to the suppression of potent diuretic hormones. Since urinary secretion decreases during a marathon race, the marked increase of aldosterone and AVP measured directly after

the race (Table 1) would reduce the diuretic effects of ANP.

Sodium concentration is increased after the run, and hypernatraemia caused by saline infusion is known to increase ANP (Rascher et al. 1985; Sagnella et al. 1985; Tikanen et al. 1985). Sodium-replete normal men have higher plasma ANP concentrations and a greater increase of ANP after maximal treadmill exercise than sodium-depleted subjects (Richards et al. 1987). Hypernatraemia is possibly an additional factor for the increase of ANP, though it would not be efficient to secrete a hormone that causes natriuresis during conditions of sodium loss. However, high levels of aldosterone under different circumstances probably prevent additional urinary sodium losses in long distance runners.

Considering the ineffective diuretic effect of ANP under the conditions of a marathon run, it remains unclear whether high plasma ANP concentrations have any particular physiological function, or if ANP is only released by the strenuous work of the heart without specific purpose.

High levels of renin and aldosterone (Table 1) prevent the long distance runner from losing additional fluid. But the activation of the renin-angiotensin-aldosterone system also has a strong vasoconstricting effect. Vasoconstriction decreases blood flow, which probably reduces the physical capacity. It is known that ANP inhibits the vasoconstricting effect of angiotensin II (Kleinert et al. 1984; Anderson et al. 1986). Furthermore, it has been suggested that the atrial secretion of a natriuretic hormone might be increased by circulating angiotensin II (Manning et al. 1985).

Exercise on the cycle ergometer at 20% of maximal oxygen uptake ($\dot{V}O_{2max}$) leads to a slight increase of ANP while renin activity is unchanged. A significant increase of renin activity was also observed in this study during a second exercise at $\dot{V}O_{2max}$ 40% and higher plasma ANP concentrations (Saito et al. 1987). The heart workload should be the primarily stimulus for ANP secretion during physical exercise but activation of the renin system may be an additional stimulating factor. The ANP reduces renin secretion, blocks aldosterone secretion and hinders the sodium retaining action of aldosterone (Laragh 1986) but these mechanisms are apparently insignificant under conditions of a maximally stimulated renin-aldosterone system, as caused by PV losses in our study.

Manning et al. (1985) showed that administration of pharmacological doses of AVP induced a significant release of ANP. The ANP antagonized the vasoconstricting effect of a hormone that is primarily secreted to prevent the runner from fluid losses. It has been suggested that the diuretic action of ANP might be related to an inhibition of vasopressin renal activity (Sagnella et al. 1987).

Mean increases in plasma cGMP concentrations, measured directly after the run, were significantly ($P<0.05$) lower than those of the increases in mean plasma ANP concentrations. Since cGMP is extruded from cells after interaction with ANP, it is a possible marker for ANP. Moreover, because of the longer half-life in plasma of cGMP (15 min) compared with the shorter half-life in plasma of ANP (1–2 min) cGMP can be regarded as a more sensitive marker for ANP release than ANP itself in many situations (Gerzer et al. 1985, 1986).

Under conditions of water loading before the infusion of 2 l of physiological NaCl, during the next 30 min ANP levels may be almost unaltered, whereas cGMP increases about twofold (Gerzer et al. 1986). When contemplating the longer half-life of cGMP, it is unclear why the increase in cGMP after marathon running is smaller than the increase of ANP. One day after the run, PV was shown to be markedly elevated but plasma concentrations of ANP and cGMP were not significantly altered compared to the pre-race values.

However Lijnen et al. (1987) demonstrated marked elevations of plasma ANP and cGMP at two longer periods 36 h and 7 days after a marathon run and attributed this to plasma volume changes. In our study ANP had decreased immediately after the race with no further significant alterations at later times. According to Follenius and Brandenberger (1988) ANP-levels increased rapidly during 30 min of exercise (75% $\dot{V}O_{2max}$) but decreased immediately during the recovery period to control values within 30 min. It seems that ANP had no influence on the PV increase in the longer recovery period. The same holds for the other fluid regulating hormones (renin, aldosterone and AVP).

As previously described (Röcker et al. 1989), the influx of plasma proteins into the intravascular space may be responsible for the post-exercise haemodilution. This suggestion is supported by the PV increase of 10%, whereas the plasma protein concentration remained unchanged ($-0.10\ g \cdot dl^{-1}$) 31 h after the race (Table 1). Similar results were reported by Maron et al. (1975) who calculated an increase of 27 g of plasma proteins in the vascular space after marathon running.

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References

Anderson JV, Struthers AD, Payne NN, Slater JDH, Bloom SR (1986) Atrial natriuretic peptide inhibits the aldosterone response to angiotensin II in man. Clin Sci 70:507–512

Bollerslev J, Svanegard J, Blaabjerg O, Pindborg T (1987) Atrial natriuretic peptide in relation to physical exercise. Scand J Clin Invest 47:681–683

Convertino VA, Brock PJ, Keil LC, Bernauer EM, Greanleaf JE (1980) Exercise training-induced hypervolemia: role of plasma albumin, renin, and vasopressin. J Appl Physiol 48:665–669

De Bold AJ, Borenstein HB, Veress AT, Sonnenberg H (1981) A rapid and potent natriuretic response to intravenous injection of atrial myocardial extract in rats. Life Sci 28:89–94

Espiner EA, Crozier IG, Nicholls MG, Cuneo R, Yandle TG, Ikram H (1985) Cardiac secretion of atrial natriuretic peptide. Lancet II:398–399

Follenius M, Brandenberger G (1988) Increase in natriuretic peptide in response to physical exercise. Eur J Appl Physiol 57:159–162

Garcia R, Lachance D, Thibault G, Cantin M, Gutkowska J (1986) Mechanisms of release of atrial natriuretic factor. II. Effect of chronic administration of α- and β-adrenergic and cholinergic agonists on plasma atrial ANF in the rat. Biochem Biophys Res Commun 136:510–520

Gerzer R, Witogall H, Tremblay J, Gutkowsky J (1985) Rapid increase in plasma and urinary cGMP after bolus injection of atrial natriuretic factor in man. J Clin Endocrinol Metab 61:1217–1219

Gerzer R, Weil J, Strom T, Müller T (1986) Mechanisms of action of atrial natriuretic factor: clinical consequences. Klin Wochenschr 64 [Suppl VI]:21–26

Hartley LH, Mason JW, Hogan RP, Jones LG, Kotchen TA, Mougey EH, Wherry FE, Pennington LL, Ricketts PT (1972) Multiple hormonal responses to prolonged exercise in relation to physical training. J Appl Physiol 33:607–610

Haufe MC, Weil J, Nafziger K, Gerzer R, Bidlingmeier F, Theisen K (1987) Acute increase in right atrial pressure by intracardiac stimulation releases atrial natriuretic peptide. Eur Heart J 8:277–281

Heim J-M, Gottmann K, Weil J, Haufe MC, Gerzer R (1988) Is cyclic cGMP a clinically useful marker for ANF action? Z Kardiol 77 [Suppl 2]:41–46

Kleinert HD, Maack T, Atlas SA, Januszewicz A, Sealey JE, Laragh JH (1984) Atrial natriuretic factor inhibits angiotensin-, norepinephrine, and potassium-induced vascular contractility. Hypertension 6 [Suppl I]:I-143–I-147

Lang RE, Thölken H, Ganten D, Luft FC, Ruskoaho H, Unger Th (1985) Atrial natriuretic factor - a circulating hormone stimulated by volume loading. Nature 314:264–266

Laragh J (1986) The endocrine control of blood volume, blood pressure and sodium balance: atrial hormone and renin system interactions. J Hypertens 4 [Suppl 2]:S 143–S 156

Lijnen P, Hespel P, M'Buyamba-Kabangu JR, Goris M, Lysens R, Vanden Eynde E, Fagard R, Amery A (1987) Plasma atrial natriuretic peptide and cyclic nucleotide levels before and after a marathon. J Appl Physiol 63 (3):1180–1184

Manning PT, Schwartz D, Katsube NC, Holmberg SW, Needleman P (1985) Vasopressin-stimulated release of atriopeptin: endocrine antagonists in fluid homeostasis. Science 229:395–397

Maron MB, Horvath SM, Wilkerson JE (1975) Acute blood biochemical alterations in response to marathon running. Eur J Appl Physiol 34:173–181

Müller FB, Erne P, Raine AEG, Bolli P, Linder L, Resink TJ, Cottier C, Bühler FR (1986) Antrial antipressor natriuretic peptide: release mechanisms and vascular action in man. J Hypertens 4 [Suppl 2]:S 109–S 114

Newnmark SR, Himathongkam T, Martin RP, Cooper KH, Rose LI (1976) Adrenocortical response to marathon running. J Clin Endocrinol Metab 42:393–394

Pivarnik JM, Leeds EM, Wilkerson JE (1984) Effects of endurance exercise on metabolic water production and plasma volume. J Appl Physiol Respir Environ Exerc Physiol 56:613–618

Rascher W, Tulassay T, Lang RE (1985) Atrial natriuretic peptide in plasma of volume-overloaded children with chronic renal failure. Lancet II:303–305

Refsum HE, Tveit B, Meen HD, Strömme SB (1973) Serum electrolyte, fluid and acid-base balance after prolonged heavy exercise at low environmental temperature. Scand J Clin Lab Invest 32:117–122

Richards AM, Tonolo G, Cleland JGF, McIntyre GD, Leckie BJ, Dargie HJ, Ball SG, Robertson JIS (1987) Plasma atrial natriuretic peptide concentrations during exercise in sodium replete and deplete normal man. Clin Sci 72:159–164

Röcker L, Kirsch KA, Heyduck B, Altenkirch H-U (1989) Influence of prolonged physical exercise on plasma volume, plasma proteins, electrolytes, and fluid-regulating hormones. Int J Sports Med 10:270–274

Sagnella GA, Markandu ND, Shore AC, MacGregor GA (1985) Effects of changes in dietary sodium intake and saline infusion on immunoreactive atrial natriuretic peptide in human plasma. Lancet II:1208–1210

Sagnella GA, Markandu ND, Shore AC, Forsling ML, MacGregor GA (1987) Plasma atrial natriuretic peptide: its relationship to changes in sodium intake, plasma renin activity and aldosterone in man. Clin Sci 72:25–30

Saito Y, Nakao K, Sugawara A, Nishimura K, Sakamoto M, Morii N, Yamada T, Itoh H, Shiono S, Kuriyama T, Hirai M, Ohi M, Ban T, Imura H (1987) Atrial natriuretic polypeptide during exercise in healthy men. Acta Endocrinol (Copenh) 116:59–65

Saltin B, Stenberg J (1964) Circulatory response to prolonged severe exercise. J Appl Physiol 19 (5):833–838

Schiffrin EL, Gutkowska J, Kuchel O, Cantin M, Genest J (1985) Plasma concentration of trial natriuretic factor in a patient with paroxysmal atrial tachycardia. New Engl J Med 312:1196

Schultes I, Gerzer R, Altenkirch H-U, Kirsch KA, Heyduck B, Röcker L (1988) Das Verhalten des Atrialen Natriuretischen Faktors, der klassischen volumenregulierenden Hormone, der Plasmaproteine und des Plasmavolumens bei einem Testmarathon. 31st Deutscher Sportärztekongreß Hannover, 27th–30th October, 1988

Somers VK, Anderson JV, Conway J, Sleight P, Bloom SR (1986) Atrial natriuretic peptide is released by dynamic exercise in man. Horm Metab Res 18:871–872

Strauss MB, Davis RK, Rosenbaum JD, Rossmeisl EC (1951) Water diuresis produced during recumbency by the intravenous infusion of isotonic saline solution. J Clin Invest 30:862–868

Tanaka H, Shindo M, Gutkowska J, Kinoshita A, Urata H, Ikeda M, Arakawa K (1986) Effect of acute exercise on plasma immunoreactive atrial natriuretic factor. Life Sci 39:1685–1693

Tikkanen I, Fyhrquist F, Metsärinne K, Leidenius R (1985) Plasma atrial natriuretic peptide in cardiac disease and during infusion in healthy volunteers. Lancet II:66–69

Wade CE, Dressendorfer RH, O'Brien JC, Claybauch JR (1981) Renal function, aldosterone, and vasopressin excretion following repeated long-distance running. J Appl Physiol Respir Environ Exerc Physiol 50:709–712

Weil J, Bidlingmaier F, Döhlemann C, Kuhnle U, Strom T, Lang RE (1986) Comparison of plasma atrial natriuretic peptide levels in healthy children from birth to adolescence and in children with cardiac diseases. Pediatr Res 20:1328–1331