GASTRIC FUNCTION AND HYPOHYDRATION DURING EXERCISE

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There appears to be a relationship between hypohydration during endurance exercise and gastrointestinal (GI) dysfunction. Results of a field study indicate that body weight losses in the range of 4-5% during marathon running are associated with an increased prevalence of GI disorders (Rehm, N.J. et al. Int. J. Sports Med. 10 (suppl. 1): 22-25, 1989). Delayed gastric emptying (GIE) with a mean body weight loss of 5% was observed in a laboratory study by P.D. Neuffer et al. (Eur. J. Appl. Physiol. 58: 493-499, 1989). These findings point to a relationship between exercise, hypohydration, and the concomitant heat stress with GI function. Reports by endurance athletes who complain of GI problems occurring after they drink during long-lasting competition may possibly be explained by these results.

Sixteen endurance trained athletes conducted similar hypohydration and eusthenia experiments at rest and during exercise. Hypohydration was achieved by continuous running (60% VO_{2max}) at 30°C C or by intermittent sauna exposure (100°C) until 4% body weight (BW) was lost or maximally for 2 h. Mean weight loss was 3.7% ± 0.3 (SE). Thereafter, 0.5 kg BW^{-1} of a 7% carbohydrate and electrolyte containing beverage was ingested and GIE was monitored for 40 min. The double sampling technique (Bocken, E.J. et al. Gut 19: 1725-1729, 1998) was used to measure gastric residue and secretion volumes. Dehydration-exercise resulted in slower GIE than in all other treatments (p<0.05). ANOVA revealed significant effects of dehydration and exercise (p<0.05). GI complaints were reported by 37% of the subjects during dehydration-exercise experiments. No GI distress was reported in other tests. Core body temperature was greater after the dehydration-exercise regime (39.1°C) than after the rest-dehydration regime (37.9°C) (p<0.05). It is concluded that dehydration effect is acting to delay GIE and may be related to a greater prevalence of GI disturbance.

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L-PROPYLCARNITINE-INDUCED INCREASE IN POST-ISCHEMIC BLOOD FLOW DOES NOT LEAD TO RECOVERY OF FUNCTION AND ENERGY CHARGE IN ANAESTHETISED PIGS

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In the present investigation the effects of pretreatment with L-propylcarnitine (50 mg/kg, n = 9) or saline (n = 10) were studied in open-chest anaesthetised pigs, in which ischaemia was induced by decreasing left anterior descending coronary artery blood flow to 20% of baseline (baseline values 79 ± 6 and 87 ± 6 ml/min 100 g^{-1}) for the saline- and the L-propylcarnitine-treated animals, respectively. After 60 min of ischaemia the myocardium was reperfused for 2 hours. In both groups, flow reduction abolished contractile function of the affected myocardium and caused similar decreases in ATP (by 55%) and energy charge (decrease from 0.91 to 0.60). Mean arterial blood pressure, the maximum rate of rise in left ventricular pressure and cardiac output decreased from 97 ± 4 to 74 ± 5 mmHg from 2530 ± 130 to 1730 ± 110 mmHg s^{-1} and from 2.3 ± 0.1 to 1.6 ± 0.11 min^{-1}, respectively in the solvent-treated animals and from 87 ± 6 to 75 ± 5 mmHg from 2390 ± 110 mmHg s^{-1} and from 2.0 ± 0.1 to 1.6 ± 0.11 min^{-1}, respectively in the L-propylcarnitine-treated animals. Recovery of contractile function was not observed in either group after two hours of reperfusion. "No-reflow" was attenuated by L-propylcarnitine, since myocardial blood flow returned to 53 ± 8% and 52 ± 6% of baseline in the saline- and the L-propylcarnitine-treated animals, respectively. Cardiac output of the saline-treated animals further decreased (to 52 ± 8% of baseline) and an increase in systemic vascular resistance (from 46 ± 3 to 61 ± 9 mmHg/min 100 g^{-1}) was necessary to maintain arterial blood pressure. In the L-propylcarnitine-treated pigs cardiac output remained at 76 ± 4% of baseline and systemic vascular resistance decreased from 42 ± 3 to 38 ± 4 mmHg/min 100 g^{-1} (p<0.05 versus changes in saline treated animals). In both groups, energy charge, but not the ATP-level of the ischaemia-reperfused myocardium tended to recover, while the CP level showed significantly more recovery in the saline-treated animals. We conclude that L-propylcarnitine partially preserved vascular patency in ischaemia-reperfused porcine myocardium, but this had no immediate effect on "myocardial stunning". Potential markers for long-term recovery (Ca2+ uptake, in vitro phospholamban phosphorylation and post systolic wall thickening) were not affected by L-propylcarnitine.

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MECHANICAL PROPERTIES OF PASSIVE AND ACTIVE ISOLATED SMALL MESENTERIC ARTERIES OF THE RAT.

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Aim of this study was to compare distensibility of passive and activated small mesenteric arteries.

Seven female Wistar rats were anaesthetized with Sodium Pentobarbital (6 mg/100g body weight i.p.). From each rat a mesenteric arcade artery was manually dissected and cannulated at both ends. Mean inner diameter at 80 mmHg transmural pressure and maximal vasodilation was 260 ± 40 μm (mean±SD). Pressure ramps were applied to the vessels with frequency 2-10 min^{-1} and range 10-1200 mmHg. Pressure (P) and cross-sectional area (CSA) were continuously measured, and used to calculate distensibility (D): D = ΔCSA/(CSA × ΔP). Arteries were diluted using Acetylcholine < 3 μM or Papaverine 17 μM. Activation of vessels was induced using Nor epinephrine 0.6 μM.

Distensibility of passive vessels was maximal at P = 27 ± 2 mmHg and equalled (2.5 ± 0.5)×10^{-2} mmHg^{-1} at this pressure. At 120 mmHg, D did not significantly differ from zero. P-CSA loops of active vessels exhibited a large hysteresis. With rising P, distensibility was maximal at P = 30 ± 13 mmHg and equalled (1.9 ± 1.2)×10^{-2} mmHg^{-1}. D subsequently decreased to zero at P = 64 ± 15 mmHg. Upon further pressure elevation, the CSA decreased and varisation appeared, indicating myogenic behavior of these vessels. With decreasing P, the P-CSA curve was flat.

We conclude that submaximal activation of isolated small mesenteric arteries does not affect distensibility at low pressures. At higher pressures, activated vessels show regulatory behavior with asymmetric responses to pressure changes.

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