ture. The enthalpy of micellization is defined by the equation

$$\Delta H^\circ = -(2 - \beta)\, RT^2\, [\partial \ln (\text{c.m.c.})/\partial T]$$

and the entropy contribution of micellization can be calculated as follows:

$$\Delta S^\circ = (\Delta H^\circ - \Delta G^\circ)/T\,.$$

ΔG°, ΔH°, ΔS° values are presented in the Table.
Based on the presented results it can be generalized that

- ΔG° values are negative and slightly decline with temperature
- Depreciations of standard molar enthalpy ΔH° are more significant at more negative values. It means that micellization process becomes more exothermic at increasing temperature.
- $T\,\Delta S^\circ$ values are positive and decline at increasing temperature.

Experimental

Heptacaine was prepared by a literature method [1, 2]. NaCl (Lachema, s.p., Brno) was used to prepare the stock solution with a concentration of 0.1 mol · l^{-1}. NaCl solution was used to prepare the local anaesthetics solution with pH ≈ 4.5–5 at 25–45 °C.
KNO_3 and KCl were analytically pure (Lachema, s.p., Brno) as filling-out solutions for electrodes. KNO_3 solution was used with the concentration 0.1 mol · l^{-1} and formed the external solution of the electrode. Further, we prepared 100 ml saturated KCl solution formed the internal solution of the electrode.
A membrane with the tetracaine-SDS (dodecylsulphatsodium) complex was prepared [5, 6]. A little wheel of 5 mm diameter was cut out from the formed membrane of about 0.1 mm thickness in a Petri dish and glued via a hole to the bottom of a plastic PVC tube using tetrahydrofurane.
The electrodes was constructed as follows: Ag/AgCl/saturated KCl/0.1 mol · l^{-1} KNO_3/standard heptacaine solution/PVC membrane/solution with a sample/0.1 mol · l^{-1} KNO_3/saturated KCl/AgCl, Ag. The electromotoric force was measured with OP 208/1 pH meter (Radelkis, Hungary).
The concentration change of heptacaine homologues was measured using the automatic burette (Radelkis, Hungary) and controlled by a computer.
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Effect of captopril on the contraction of the aorta after chronic volume overload in rabbits

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There is ample evidence that the functional state of peripheral or coronary arteries is impaired in cardiac haemodynamic overload [1, 2]. Most studies on the vascular function deal with models of pressure overloaded circulation, while corresponding data in volume overload are extremely rare. We therefore studied the responsiveness of the aorta to vasoconstrictor stimuli after long term volume overload provoked by insufficiency of aortic valves in rabbits. Further, we investigated the abiltity of the angiotension converting enzyme (ACE) inhibitor captopril to reverse the potential alterations of aortic contractility.
Four groups of rabbits were studied: control(c) (n = 11) − sham operation + four months without any drug + 5 week placebo treatment, control + captopril (cC) (n = 11) − sham operation + 4 months without any drug + 5 week captopril treatment (twice daily intramuscularly 10 mg/kg), aortic insufficiency (AI) (n = 8) − operation + 4 months without any drug + 5 week placebo treatment, aortic insufficiency + captopril (AIC) (n = 8) − operation + 4 months without any drug + 5 week captopril treatment as above. The rabbits were male, Chinchilla species, with an average body weight of about 3000 g. Aortic insufficiency was induced according to Fizel and Fizelova [3] and the period of developed hypertrophy was investigated [4]. Contractility of aorta was tested with two different vasoactive drugs: prostaglandin (PGF) F2α (10^{-5} mol · l^{-1}) and potassium chloride (KCl) (50 mmol · l^{-1}). Eight rabbits were used for contractility investigation in each group. After killing the animals, a six centimeter long segment of the thoracic aorta was dissected free, and placed in an ice-cold Krebs solution, cleaned of connective tissue and cut into segments about 4 mm long. The individual segments were attached between an isometric force transducer (Sanborn FT 10) and a holder under a tension of 20 mN in a 20 ml organ bath containing Krebs solution. After a resting period of 90 min the contraction of the aorta was observed 30 min after administration of the vasoactive substance [5, 6].
Aortic pressures were measured by a catheter with an electric transducer (Statham DB P23, GB) introduced into the aorta through the left carotid artery and recorded on an oscillographic recorder Mark VII, type WR 3101 (Graphtec Corp., USA). The measurements were performed under thiopental anaesthesia [7].
Hypertrophy was determined by weighing the left ventricle and the ratios of its weight to the body weight are calculated for each animal. The results are expressed as means ± S.E.M. Differences between the groups were assessed by one way ANOVA test with $p < 0.05$ taken as significant.
Hypertrophy (weight increase by 62%) of the LV in the AI group was not reversed by five week captopril treatment. In the AI group the reactivity of the aorta was decreased to KCl (by 44%), and to PGF2α (by 51%). Five week captopril treatment improved the reactivity of the aorta to KCl but not to PGF2α.

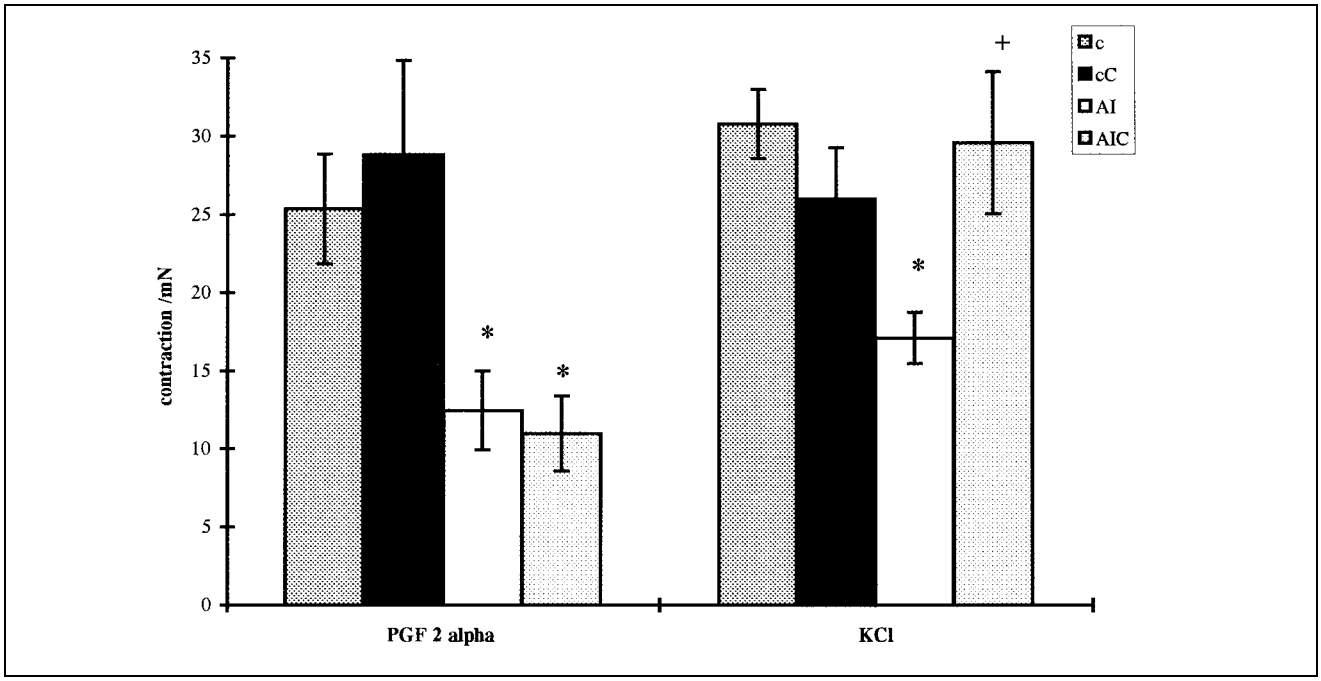

Fig.: Changes in the contractility of aorta to prostaglandin (PG) F2 alpha and potassium chloride (KCl); values are means ± S.E.M.; * – $P < 0.05$ compared to the control; + – $P < 0.05$ compared to previous group; $N = 8$ in each experimental group

Table: Changes in the relative weight of the left ventricle (LV) and in aortic pressure

	Control	Control + captopril	Aortic insufficiency	Aortic insufficiency + captopril
LV weight/b.w. (g/kg)	0.808 ± 0.032	0.790 ± 0.029	$1.308 \pm 0.065^{*}$	$1.281 \pm 0.083^{*}$
Systolic pressure (mm Hg)	111.2 ± 3.8	$100.4 \pm 3.0^{*}$	123.2 ± 6.6	118.4 ± 5.8
Diastolic pressure (mm Hg)	89.2 ± 3.3	$77.6 \pm 3.2^{*}$	85.0 ± 5.2	$71.2 \pm 2.7^{*+}$
Pulse pressure (mm Hg)	22.0 ± 1.2	21.8 ± 1.9	$38.2 \pm 2.7^{*}$	$47.2 \pm 4.5^{*}$

Values are means ± S.E.M.; * $P < 0.05$ compared to control; + $P < 0.05$ compared to previous group; $n_c = 11$, n_{cC} 11, $n_{AI} = 8$, $n_{AIC} = 8$.

The fact that the contractile response of the aorta to substantially different stimuli was decreased indicates that weakening of contraction can hardly reside in the receptor apparatus. Volume overload should be considered as the essential stimulus eliciting structural and functional alterations. The high pulse pressure in aortic insufficiency implies a higher pulse stretch and deformation of the aorta [6]. It stimulates proteosynthesis in the aorta, as we recently observed in one month lasting aortic insufficiency in this particular model [8], resulting expectedly in hypertrophy of the aortic wall. This potential structural remodelling may be an important factor associated with deterioration of aortic contractions.

Five week ACE inhibition by captopril did not induce regression of left ventricular hypertrophy, and analogically regression of potential structural alteration of the aorta should not be anticipated. In accordance with this consideration, captopril did not restore the contractile answer of the aorta to prostaglandin F2α. The fact that captopril improved aortic contractility to KCl suggests that impairment of aortic contraction associated with membrane depolarisation is more subtle and can thus be more easily restored by ACE inhibition than changes linked with alteration of the smooth muscle contractile apparatus [9].

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