Prevention of Systemic Hemodynamic Changes with Taurine

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Intravenous infusion of taurine prevents a decrease in cardiac pump function caused by electric stimulation of the aortic arch and promotes recovery of systemic blood flow and total peripheral resistance.

Key Words: *taurine; stress; electric stimulation*

The role of nonprotein sulfur-containing amino acid taurine in the regulation of cardiovascular functions is now well established. Taurine (2-aminoethanesulfonic acid) exerts vasodilatory and hypotensive effects, stabilizes cardiac rhythm, and improves heart contractions [2]. Taking into account the positive effects of taurine on the cardiovascular system, of particular interest is the possibility of using this amino acid for prevention of stress-induced hemodynamic disturbances. Stress was modeled by electric stimulation of the aortic arch. This procedure is routinely used for modeling of neurogenic damage to the myocardium [1,3].

MATERIALS AND METHODS

Experiments were carried out on male Chinchilla rabbits weighing 2.5-3 kg. The animals were narcotized and an electrode was introduced through the common carotid artery into the aortic arch, while its proximal end went out thought the ear skin. Another needle electrode was stuck into the left paw skin. The electrode positioned in the aorta was connected to a generator and stimulation with 10-msec square pulses was performed (50 Hz, 5-7 V) for 3 h as described previously [3]. The animals were then sacrificed via air embolization and the location of the stimulating electrode was verified.

Taurine (50 μmol/kg/h) or physiological saline (0.1 ml/min) were infused to experimental and control rabbits, respectively, throughout the stimulation period. Cardiac output (CO) was measured by thermodilution [5]. Mean blood pressure (BP) in the femoral vein was measured with a manometer, heart rate (HR) was recorded with an electrocardiograph. Total peripheral resistance (TPR), stroke volume and stroke index of the left ventricle were calculated. The data were processed statistically using Student's *t* test.

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RESULTS

Electric stimulation caused hypodynamic shifts in systemic hemodynamic (Fig. 1), in particular, a drop of BP, stroke volume, and HR, and a gradual decrease in cardiac output and stroke index of the left ventricle, and an increase in TPR. These changes resulted from impaired pump function of the heart due to exhaustion of energy resources in the myocardium and spasm of peripheral vessels due to sharp increase in blood catecholamine concentration [1,3].

Taurine normalized systemic hemodynamic parameters (Fig. 1). BP after a transient decrease returned to the initial level 2.5 h after the start of infusion. TPR also underwent biphasic changes: after considerable reduction by 30% this parameter returned to the initial level 2 h after the start of the experiment. Biphasic changes in BP and TPR were also reported by other investigators [7]. Stroke volume slightly increased during the first hour and then returned to baseline value, while CO remained practically unchanged during the experiment. The dynamic of stroke index was similar to that of BP and TPR, while the dynamic of HR was practically the same in the control and experimental groups.

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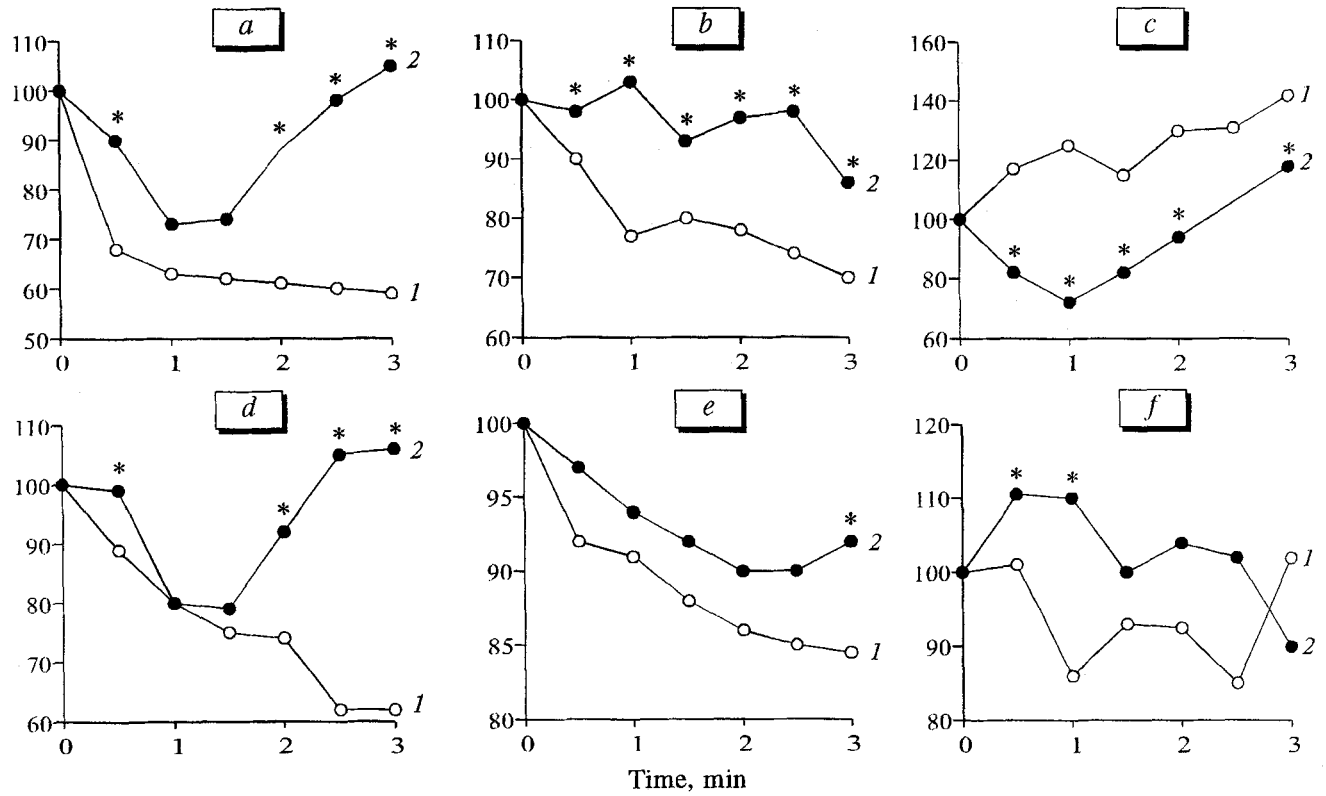

Fig. 1. Effect of taurine on systemic hemodynamics during electric stimulation of rabbit aortic arch (% of initial level taken as 100%). *a*) blood pressure; *b*) cardiac output; *c*) total peripheral resistance; *d*) stroke index; *e*) heart rate; *f*) stroke volume in control (*1*) and experimental groups (*2*). *$p<0.05$ compared with the control.

Thus, taurine infused against the background of electric stimulation prevented the decrease in CO and promoted normalization of BP and TPR. The positive dynamics of stroke index indicates recovery of cardiac function by the end of the experiment. Parallel shifts of BP and TPR with unchanged CO suggest that changes in systemic hemodynamic are underlain by vascular rather than cardiac mechanisms. Our experiments showed that taurine can be used for preventing systemic hemodynamic disturbances caused by stress.

The mechanism underlying the positive effect of taurine on cardiovascular functions in stress calls for further investigation. Previous studies attribute the protective effect of taurine in stress to its interaction with the peripheral nervous system, in particular, to modulation of the sympathetic system [6]. Activation of the sympathetic nervous system underlies stress-induced overload of the heart. Under these conditions taurine prevents calcium overload and reduces epinephrine release from nerve terminals. Moreover, taurine reduces spontaneous epinephrine release and accelerates its catabolism, inhibits norepinephrine utilization in the myocardium, and decreases its mobilization from adrenergic granules in the adrenals [4]. These processes prevent the drop of myocardial ATP and impairment of the cardiac pump function.

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