Changes in Stroke Volume in Rat Pups during Adaptation to Various Regimens of Motor Activity

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Experiments on rat pups showed that regular exercise for 4 weeks starting from day 14 after birth considerably increased stroke volume. Similar dynamics of stroke volume was observed in subsequent period of restraint locomotion. Experimental hypokinesia starting from day 14 after birth significantly limited the age-dependent increase in SV. However, subsequent exercise training promoted the increase in stroke volume in these rats. In trained rats the regulation of stroke volume during hypokinesia remained unchanged, while in restrained rats subsequent training was associated with considerable reduction of sympathetic regulation of stroke volume.

Key Words: stroke volume; different regimens of physical activity; regulatory mechanisms; blockade

Effects of different regimens of physical activity on cardiac function and its regulation in postnatal ontogeny are described elsewhere [1-3]. Most studies were devoted to peculiarities of cardiac chronotropic function in developing rats, while regulation of stroke volume (SV) during transitions from one mode of activity to another is poorly studied. Our aim was to study the dynamics of SV and mechanisms of SV control in immature rats during transitions from regular exercise training to hypokinesia and vice versa.

MATERIALS AND METHODS

Experiments were performed on random-bred albino rat pups. Freely moving animals served as the control. Group 1 rats aged 14-42 days were subjected to forced swimming test. The intensity of exercises increased stepwise. At the age of 43-70 days, the animals were placed into a narrow chamber for 23 h daily to simulate hypokinesia. In group 2, rat pups were first exposed to hypokinesia (days 14-42) and on days 43-70 to exercise of increasing intensity, similar to that in

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group 1. SV was determined by thoracic tetrapolar impedance cardiography. Differentiated impedance cardiogram was continuously recorded in spontaneously breathing animals under Nembutal narcosis (40 mg/kg) with an RPG-204 rheoplethysmograph. SV was calculated by Kubichek's formula.

The intensity of sympathetic and parasympathetic influences were evaluated by SV shifts during pharmacological blockade of adrenergic and muscarinic cholinergic receptors with propranolol (0.8 mg/100 g, intraperitoneally) and atropine (0.3 mg/100 g, intraperitoneally).

RESULTS

In control rats, SV gradually increased after birth and by day 70 it attained 0.213 ml. In group 1, this SV (0.212 ml) was observed on day 43 after birth. This parameter continued to grow during restraint stress and attained 0.287 ml by day 70 (by 0.074 ml higher than in control; p<0.05). Thus, regular training for 4 weeks starting from day 14 significantly increased SV, which continued to grow during the subsequent hypokinesia. We hypothesized that exercise training during the early ontogeny promotes myocardial hypertrophy.

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In group 2 animals, SV was only 0.105 ml on day 43, i.e. by 0.107 ml lower than in age-matched trained rats (p<0.01). This group manifested a considerable increase in SV after regular exercise training. By day 70, SV increased 2-fold (to 0.219 ml). Thus, early hypokinesia significantly enhanced the effect of regular exercise on SV growth.

High values of SV in exercise-trained rats and further increase in SV during subsequent hypokinesia can be explained by long-term effects of regular exercise training at early age. It should be noted that experiments were performed on the developing rats, which normally exhibit age-related increase in SV.

The rise of SV during transition from hypokinesia to exercise training can result from progressive and gradual adaptation of the rat to hypokinesia.

Blockade of parasympathetic influences increased SV in 14-day-old controls by 0.014 ml, while sympathetic blockade decreased it by 0.01 ml. These changes in SV slightly decreased by day 43, and increased by day 70.

In group 1, atropine injected to 43-day-old rats increased SV by 0.05 ml, while propranolol decreased it by 0.04 ml, which attested to weakening of both sympathetic and parasympathetic regulation of SV during regular exercise training until day 43. Subsequent immobilization led to only moderate enhancement of the parasympathetic regulation of SV without changes in sympathetic control.

In group 2, both parasympathetic and, especially, sympathetic regulation of SV were considerably stren-

gthened on day 43. Subsequent training was accompanied by weakening of sympathetic influences. For instance, SV response to propranolol in 43-day-old rats subjected to hypokinesia was 40.9%, while the subsequent exercise training reduced this value to 27.3%.

Thus, physical exercises of increasing duration and intensity promote the decrease in both sympathetic and parasympathetic influences on SV. Under these conditions, the extracardial regulatory influences at rest are persistently reduced, and cardiac autoregulation prevails [4]. This effect persists even during subsequent hypokinesia.

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