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Research Article

Reaction Features of the Mature Rats' Inotropic Heart Function Subjected to Different Modes of Motor Activity Under Adreno and M-Choline Stimulants

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Abstract

The research has been carried out for the first time to study the mice stroke volume (SV) response to the insertion of β, α1, α2-adrenergic and M- cholinergic stimulants. The largest increase in the SV response was revealed during animals muscle training with the insertion of β and α1-adrenergic stimulants and a simultaneous decrease in the SV response to the insertion of an M-cholinergic stimulant can be noted. A group of animals subjected to limited motor activity, i.e. hypokinesia was seen to have a decrease in the SV response to the insertion of β-, α1-, α2-adrenergic stimulants. Consequently, it can be argued that while animals subject to a regime of limited motor activity, the sympathetic regulatory influence remains at a low level.

Keywords: laboratory animals, muscle training, different modes of motor activity, enhanced motor activity (EMA), limited motor activity (LMA), hypokinesia, unlimited motor activity (UMA), β, α1, α2-AR, M-ChR, stroke volume blood response (SV).

Introduction

The regulation of heart activity is known to be carried out by the interaction of the sympathetic and parasympathetic parts of the autonomic nervous system, which exert its influence through adrenoreceptors and cholinergic cardiac cells receptors [1-11].

According to some researchers, β-adrenergic receptors are the most common in the heart. Their stimulation increases the force of myocardial contraction, the conductivity and sensibility of the cardiac muscle. Through specific G proteins, β- ARs are able to modulate the activity of various intracellular signaling systems.

Most clinical and experimental studies have focused on the effect of β-AR blockade. Recently, there has been a renewed growth of interest in studying the participation of α-adrenergic receptors in the regulation of cardiac functions [12]. Despite the low density of α1-ARs compared to β-ARs, they play an important role in the regulation of cardiac functions [13]. α1-ARs are known to be present in the heart and are considered to be identical in different animal species. The representation of α1-AR in the human heart has been demonstrated at the molecular level [12]. However, the significance of α2-AR in the heart has not been studied properly. Previously, it was believed that α2-AR in the mammal hearts modulates regulatory influences, being located presynaptically and inhibiting the release of norepinephrine [12]. There is another opinion that α2-AR is responsible for the regulation of myocardial contractility. Thus, at present, researchers do not have a consensus on the participation of β and α-AR in the regulation of inotropic heart function. Moreover, the role of different AR subtypes in the

regulation of the pumping heart function of animals with different modes of physical activity remains practically unstudied. The role of M-ChR in the regulation of inotropic heart function of animals exposed to various types of physical activity also requires research.

The aim of our study is to find out the role of alpha-, beta-adrenoreceptors and M-cholinergic receptors in the regulation of inotropic heart function of mature animals undergoing various modes of motor activity.

Materials and Methods

White mongrel rats aged from 120 to 150 days were used for experiments. 3-4 same-sex rodents were placed in standard plastic cages for keeping and breeding.

To study the role of different subtypes of AP and M-ChP in the regulation of the pumping heart function of animals subject to different modes of motor activity, the corresponding agonists euphillin (β), meditin (a2), phenylephrine (a1) and carbacholine (M-XP) were inserted [14].

The muscle training of the animals was carried out by daily swimming, increasing in time and intensity. The restriction of motor activity, i.e. hypokinesia for laboratory rats was created by keeping them in special pencil cases.

Tetrapolar thoracic rheography was used to determine the stroke volume of blood (SV) and heart rate [15]. A differentiated rheogram was recorded in dynamics in anesthetized animals with natural respiration using an RPG– 204 device.

To assess the reliability of the differences, standard values of the Student's t- test were used.

The Results of our Own Research

As our studies have shown, the highest reaction to the beta-agonist injection is observed in animals with enhanced motor activity (EMA), whereas in animals subject to limited motor activity (LMA), this reaction was significantly lower.

During the first week of being intensively trained mature rats after the α1- adrenostimulator-phenylephrine injection were observed to have the increase of SV by 0.132 ml compared with the baseline data and amounted to 0.349 ± 0.007 ml ($P \leq 0.05$) (Table 1). During the next three weeks of keeping the animals under unlimited motor activity (UMA) the reaction of SV to the insertion of an α1- adrenostimulator increased by about 12-18 ml weekly ($P \leq 0.05$). The difference between the initial SV reactions to the injection of an α1-agonist and those registered at the fourth week of UMA group animals was 0.062 ml ($P < 0.05$). Consequently, UMA group animals kept in the mode of unrestricted motor activity for four weeks showed a significant increase in the response of the SV to the insertion of an α1- adrenostimulator.

	β (agonist)	α1 (agonist)	α2 (agonist)	M-ChP (agonist)
n (number of animals)	11	10	12	11
initial	0.211± 0.007	0.217± 0.005	0.219± 0.008	0.210± 0.007
after injection	0.297± 0.008*	0.327± 0.009*	0.297± 0.009*	0.195±0.003*
1 week	0.299± 0.005	0.349± 0.007*	0.299± 0.007	0.193± 0.005
2 weeks	0.307± 0.009*	0.367± 0.008*	0.287± 0.004*	0.185±0.003*
3 weeks	0.327± 0.004*	0.377± 0.005*	0.277± 0.005*	0.174±0.004*
4 weeks	0.359± 0.007*	0.389± 0.009*	0.269± 0.006	0.165± 0.005
*- the difference ($P<0.05$) compared with the previous value				

Table 1: Changes in the SV reactions of UMA group mature rats to the injection of β, α1, α2-adrenostimulants and M-ChP stimulator

Animals subjected to systematic muscle training (the EMA group) were also observed to have a significant increase in the reaction of the SV to the injection of the α1-agonist- phenylephrine in the first week (Table 2). In contrast, rats exposed to systematic muscle training had a better reaction of SV to the injection of an α1- agonist starting from the second week of systematic muscle training. Consequently, during four weeks systematic muscle training, animals of the experimental group were seen to have significant increase in the SV reaction to the injection of an α1- adrenostimulator.

	β (agonist)	α1 (agonist)	α2 (agonist)	M-ChP (agonist)
n(num of aminals)	12	14	15	14
initial	0.217± 0.005	0.215± 0.007	0.214± 0.009	0.217± 0.009
After injection	0.307±0.007*	0.315± 0.008*	0.277± 0.004*	0.197±0.004*
1week of training	0.319± 0.004*	0.359± 0.004	0.269± 0.005	0.160± 0.005
2 weeks of training	0.327± 0.006	0.377± 0.003*	0.235± 0.008*	0.155±0.008*
3 weeks of training	0.347± 0.009*	0.389± 0.008*	0.247± 0.007*	0.141±0.007*
4 weeks of training	0.379± 0.007*	0.417± 0.007*	0.249± 0.006	0.130± 0.006
*- the difference (P<0.05) compared with the previous value				

Table 2: Changes in the SV reactions of the EMA group mature rats to the introduction of β, α1 and α2-adrenostimulants and M-ChP stimulator

The least reaction of the SV to the injection of an α1-agonist was found out in the group of animals subject to a regime of lmited activity, i.e. hypokinesia (LMA group) (Table 3). This group of animals showed lower reaction of SV to the introduction of an α1-adrenostimulator in the first week of experiments compared to the indicators of the UMA group animals. This difference was 0.027 (P<0.05). The low reaction of the SV to the insertion of an α1-adrenostimulator in this group of animals remained during the next three weeks of activity restriction. By the end of the fourth week of hypokinesia, the reaction of the SV to the injection of the α1- agonist was approximately the same as of the initial point. Consequently, a group of mature animals with hypokinesia showed significantly low reaction of SV to the injection of an α1-agonist. Subsequently, the low reaction of the SV remained the same during the next four weeks of hypokinesia. As our studies have shown, slow motor activity (hypokinesia) causes a decrease in the response of the SV to the injection of an α1-adrenostimulator.

	β (agonist)	α1 (agonist)	α2 (agonist)	M-ChP (agonist)
n(number of animals)	11	15	14	15
initial	0.214± 0.005	0.218± 0.008	0.215± 0.009	0.210± 0.007
After injection	0.291± 0.007*	0.317± 0.009*	0.189± 0.004*	0.195±0.002*
1 week of hypokinesia	0.298± 0.009	0.322± 0.007	0.179± 0.005*	0.197± 0.005
2 weeks of hypokinesia	0.277 ± 0.005*	0.317± 0.008	0.167± 0.007*	0.188±0.003*
3 weeks of hypokinesia	0.258± 0.004*	0.299 ± 0.005*	0.157± 0.006*	0.179±0.005*
4 weeks of hypokinesia	0.249± 0.008*	0.309± 0.009	0.139± 0.009*	0.175± 0.005
*- the difference (P<0,05) compared with the previous value				

Table 3: Changes in the SV reactions of the LMA group mature rats to the introduction of β, α1 and α2-adrenostimulators

Analyzing the features of the SV to the injection of an α1-adrenostimulator, we found that the group of animals subjected to systematic muscle training demonstrate the significant increase of the SV reaction by the end of the fourth week experiments, whereas the animals having hypokinesia, on the contrary, show significant decrease.

While experimenting with intact rats kept in the mode of intensive motor activity we observe the SV increase by 0.080 ml during the first week of experiments after the injection of the α2-adrenostimulator- meditin. During the next three weeks of keeping the same animals in the UMA mode, the reaction of the SV to the injection of an α2-adrenostimulator decreased weekly by about 0.010 ml ($P \leq 0.05$). The difference between the initial SV reactions to the insertion of the a2-agonist and those registered at the fourth week of the UMA was 0.050 ml ($P < 0.05$). Consequently, the intact animals kept in the UMA mode for four weeks showed a significant decrease in the response of the SV to the injections of an α2- adrenostimulator. After the insertion of an α2-agonist systematically trained animals (the EMA group) illustrated less reaction of the SV in the first week of experiments (lower to 0.030 ml) compared with the indicators of the same age lab animals ($P \leq 0.05$). Moreover, the second week showed a further decrease in the SV response of systematically trained animals to the injections of an α2-agonist. During the third and fourth weeks of training, the SV response of trained rats remained stable. In the fourth week the EMA group demonstrated the reaction of the SV to the injection of an α2-agonist 0.028 ml lower than the initial value. Consequently, animals subjected to systematic muscle training demonstrated decrease in the response of the SV to the injection of an α2-adrenostimulator. The difference between the reactions of the control group and the EMA group to the insertion of an α2-adrenostimulator by the end of the fourth week of experiments was more than 0.020ml ($P < 0.05$).

As can be seen the group of animals with hypokinesia was observed to have a decrease in the response of the SV to the injection of an α2-adrenostimulator during the first week. At the same time, it should be noted that the reaction of the SV to the injection of an α2-adrenostimulator to animals subjected to hypokinesia in the first week of experiments was 0.012 ml more than the reaction of animals exposed to muscle training ($P \leq 0.05$). During the next four weeks of activity restriction, the reaction of the SV gradually decreased. The difference between the initial SV reactions to the injection of the a2-agonist and the reactions obtained at the end of the fourth week of slow active group animals was 0.050 ml ($P < 0.05$). Consequently, the regime of limited motor activity (hypokinesia) causes a significant decrease in the response of the SV to the injections of an α2-adrenostimulator.

Analyzing the features of the SV reaction to the introduction of an α2- adrenostimulator, we found that in all studied groups of animals (EMA, LMA and UMA), the reaction of the SV decreases significantly by the end of the fourth week of experiments. At the same time, a significant SV reduction to the injections of an α2-adrenostimulator occurs to the animals subjected to a regime of limited motor activity.

Under ordinary conditions rats kept in the UMA regime for four weeks demonstrated a significant decrease in the response of the SV to the injection of the M-choline stimulator. Animals subjected to systematic muscle training under the M-holino agonist, had the lower reaction of the SV in the first week of experiments (by 0.033 ml) compared with the indicators of the same age control group animals ($P < 0.05$). During the fourth week of systematic muscle training, the EMA group animals demonstrated the reaction of the SV to the injection of an M-choline agonist 0.067 ml lower than the initial value. Consequently, animals subjected to systematic muscle training, showed a decrease in the response of the SV to the injection of an M-choline-adrenostimulator. The difference between the reactions of the control group and the EMA group to the insertion of the M-choline stimulator by the end of the fourth week of experiments was more than 0.035ml ($P < 0.05$).

The group of animals exposed to the hypokinesia, as the previous studied groups showed, was seen to have a decrease in the response of the SV to the injection of the M-choline stimulator during the first week of the experiment. At the same time, we noted that the reaction of the SV to the injection of an M-choline stimulator to the animals subjected to hypokinesia in the first week of experiments was 0.037 ml more, compared with the reaction of animals exposed to muscle training ($P < 0.05$). During the next four weeks of motor restriction the animals SV reaction gradually decreased. The difference between the initial SV reactions to the injections of M-choline agonist and the reactions obtained at the end of the fourth week of hypokinesia in this group of animals was 0.020 ml ($P < 0.05$). Consequently, the limited motor active regime (hypokinesia) slightly reduces the reaction of the SV to the introduction of an M-choline stimulator.

Thus, analyzing the features of SV reaction to the introduction of M- cholinostimulants, we found that in all studied groups of animals (EMA, LMA and UMA), the reaction of the SV goes down significantly by the end of the fourth week of experiments. At the same time, greater decrease in the SV reaction to the introduction of M-cholinostimulants occurs to the animals subjected to a regime of increased motor activity (EMA group).

Conclusion

Analyzing the features of the SV reaction to the injections of an α2- adrenostimulator, we found that in all the studied groups of animals (EMA, LMA and UMA), the SV reaction decreases significantly by the end of the fourth week of experiments. At the same time, more decrease in the SV reaction to the injection of an α2-adrenostimulator was seen to the animals subjected to a regime of limited motor activity (LMA group).

EMA group animals demonstrated a high level of SV reaction to the injection of β and α1–adreno stimulators, the SV reaction to the injection of an M-holino stimulator is seen to be significantly reduced. LMA group animals showed a significant decrease in the SV response to the injection of β, α1 and α2-adrenostimulants. The

SV to the injection of the M-choline stimulator remains at a high level.

According to our data, in the process of muscle training, the greater increase in the SV reaction occurs during the α1-adreno stimulator injection. Apparently, this is due to the fact that in the adult body, with systematic muscle training, myocardial hypertrophy is observed, with the participation of these receptors.

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