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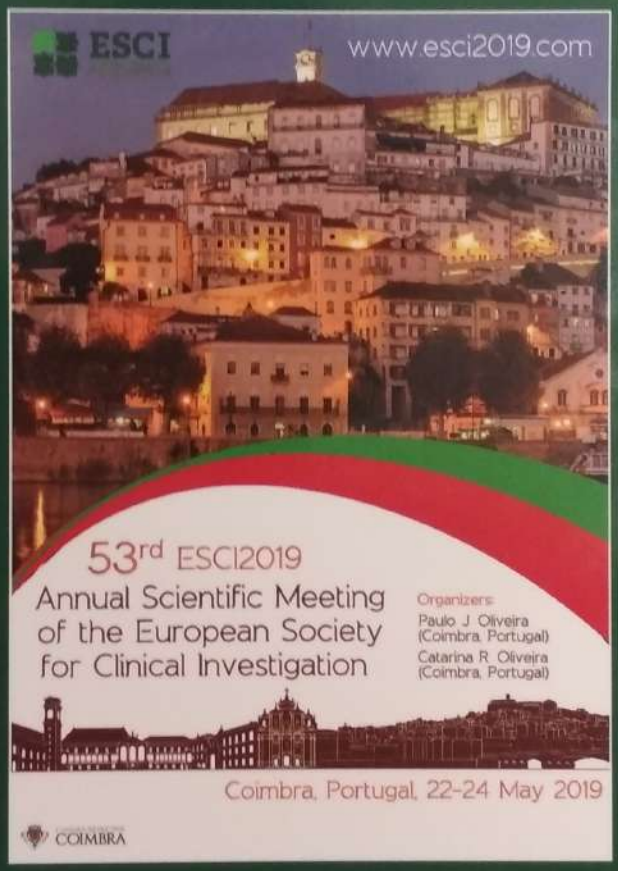

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ABSTRACT BOOK

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Pinacidil produces concentration-dependent vasorelaxation of HSV and HIMA obtained from diabetic patients. 4-aminopyridine (4-AP, 1 and 3 mmol/L), a nonselective blocker of Kv channels antagonize the effect of pinacidil on HIMA obtained from patients with T2DM ($P < 0.05$ both). The same concentrations of 4-AP did not antagonize the effect of pinacidil on HSV obtained from patients with T2DM ($P > 0.05$ both). Margatoxin, highly selective blocker of Kv1.3 channels (30 nmol/L), did not change relaxation effects of pinacidil on both types of grafts obtained from patients with T2DM ($P > 0.05$).

It seems that 4-aminopyridine-sensitive Kv channels are involved in the vasodilatation of HIMA induced by pinacidil. The Kv1.3 channels do not contribute to the relaxation effects of pinacidil on bypass grafts obtained from the patients with T2DM.

P102-T | Effect of dopamine on action potential generation in the newborns rats right atrium cardiomyocytes

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Background: Dopamine is known as the major neurotransmitter in CNS. Dopamine was also found in the sympathetic ganglia, nerves and heart. The intensity of dopamine secretion is 10-20 times higher than that of epinephrine and norepinephrine. The effect of dopamine on the heart in low concentration is mediated via dopamine receptors and in high concentration via α- and β-adrenoceptors. The purpose of this study is to investigate dose-dependent dopamine effects on the parameters of right atrial preparations electrical activity in newborns rats.

Methods: The study was carried out on 7-day white outbred laboratory rats. Membrane potential (MP) and action potential (AP) were recorded using glass microelectrodes (tip diameter <1 μ, resistance 30-80 MΩ).

Results: Dopamine caused concentration-dependent changes in the electrical activity. Dopamine at a concentration of 10^{-7} mol/L did not cause significant changes in MP and PD parameters of animals. Dopamine at a concentration of 10^{-6} mol/L did not cause changes in MP, AP amplitude and the duration of depolarization. But the duration of repolarization increased by 12% ($P < 0.05$). Dopamine at a concentration of 10^{-5} mol/L did not cause significant changes in MP, AP amplitude and the duration of depolarization. The duration of the repolarization increased by 22% ($P < 0.05$).

Conclusions: Our studies indicate that dopamine receptors are involved in regulating of the AP repolarization phase duration. The increase of the repolarization phase duration is possibly associated with a change in the kinetics of K+ channels and total K-current decrease. Further studies will determine the subtypes of dopamine receptors involved in the regulation of the cardiomyocytes electrical activity.

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P103-T | The influence of clonidine hydrochloride on the myocardium electrical activity

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The presence and functional role of α2-AR in the humans and animals heart was extensively studied. This work assessed changes of the action potential (AP) parameters of rat atrial cardiomyocytes induced by a α2-adrenoreceptor agonist clonidine hydrochloride in concentrations 10^{-9} to 10^{-5} mol/L. The experiments on intracellular recording of electrical activities in the working myocardium, were carried out on random-bred albino rats. Isolated right atrial wall from the right auricle exhibiting no pacemaker activity was placed in a 3-mL chamber and superfused with Tyrode solution at 38°C at a rate of 10 mL/min. Intracellular AP was recorded via glass microelectrodes with resistance of 40-80 MΩ. The signals were digitized with an E14-140 converter (L-Card). The data were processed with Elph 3.0, Microsoft Excel software and Student's *t* test.

Clonidine hydrochloride in concentrations of 10^{-9} to 10^{-5} mol/L increased the duration of the action potential and reduced the frequency of action potential generation. The maximum concentration of the substance caused the maximum effect. None of the tested concentrations of the clonidine hydrochloride produced significant effect on resting potential or upstroke velocity of action potential.

Stimulation of α2-AR clonidine hydrochloride affects the electrical activity of the right atrial cardiomyocytes in adult rats and the duration of the action potential, repolarization phase continuance and the frequency of action potential generation.

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