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Myocardial electrophysiological effects mediated by KATP channels: controversial aspects about their involvement in the protection by chronic exercise

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Background

It is widely accepted that physical exercise protects against cardiac arrhythmias such as ventricular fibrillation (VF). Most of the authors have proposed activation of KATP channels during ischemia as the mechanism by which training would confer protection to the ischemic myocardium. However, it has also been reported that the opening of these channels leads to reductions in action potential duration and dispersion of repolarization with a proarrhythmic effect.

Purpose

We have investigated the effects of either KATP channel activation and blockade on VF inducibility (normoxia conditions) along with the incidence of spontaneous defibrillation and asystole as events related to arrhythmogenesis and electrophysiological deterioration during global ischemic conditions, in order to support or question the most extended idea that it is the opening of these channels what exerts myocardial protection during ischemia.

Methods

44 NZW male rabbits were assigned to 4 groups (control,n=13; trained,n=12; pinacidil,n=9 and glibenclamide, n=11); the hearts excised and placed in a Langendorff system. After a stabilization period the hearts were immersed in a thermostatic bath and pacing and recording electrodes were placed on the ventricular epicardium. In order to determine the inducibility of VF, prior to global ischemia, the ventricular extrastimulus test (VEET) was applied. If the effective refractory period was reached before inducing VF, the arrhythmia was triggered in all hearts by pacing at increasing frequencies. VF signal evolution was analyzed during 10 min of global ischemia to determine the incidence of spontaneous defibrillation and electrical asystole. A chi-square test was used; significance when p<0.05.

Results

Under normoxic conditionsVF was induced in 4 out of 9 (44.44%) hearts treated with pinacidil during VEET application. In 2 cases from control (15,38%), and in 1 from glibenclamide (9,10%) and trained (8,33%) groups the arrhythmia was triggered (²(3)=5.729, p=0.126). After the analysis of ventricular fibrillation signal evolution during global ischemia, we observed that in every (n=9) heart treated with pinacidil there was a total loss of electrical activity (asystole) after cessation of coronary perfusion while only two hearts belonging to glibenclamide and one from control and trained groups went through this circumstance (²(2)=28,079, p<0.001).
Conclusion

Activation of IKATP exerts arrhythmogenic and deleterious electrophysiological effects during normoxia and ischemic conditions. Blockade of KATP channels during ischemia has shown antiarrhythmic properties. The electrophysiological effects of training on the events analyzed do not seem to be mediated by KATP opening. Further research must be taken in order to clarify the role of KATP channels in the protective mechanisms elicited by physical training.