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EFFECTS OF ATORVASTATIN ON TRANSIENT SODIUM CURRENTS IN RAT NORMAL/SIMULATED ISCHEMIA/REPERFUSION VENTRICULAR CELL

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Background Some clinical trials have shown statins have anti-arrhythmic effects and can improve clinical results. But its mechanism is unclear.

Objective observing the effects of atorvastatin on transient sodium currents in rat normal/simulated ischemia/reperfusion ventricular cell.

Methods Taking whole-cell patch clamp method to record I_{Na} and measuring the expression level of SCN5A by western blot technique of simulated ventricular ischemia /reperfusion cell.

Results The short-time effects of atorvastatin on the rat normal and simulated ischemia ventricular peak I_{Na} were inhibited about 25% ($p<0.05$), and after elution, inhibition disappeared. However 15 min after simulated ischemia atorvastatin inhibited the I_{Na} decreasing progress. In simulated reperfusion status, I_{Na} reduced and atorvastatin inhibited the reduction degree, while I_{Na} of the atorvastatin and wortmannin combination group had no difference with which of reperfusion group ($p>0.05$). The expression level of SCN5A had the almost same changes with I_{Na}.

Conclusion (1) The short time (3 min) effect of Atorvastatin in I_{Na} of the normal and simulated ischemia rat ventricular myocytes is inhibition, similar to sodium channel blockers. (2) Atorvastatin can protect the decrease of I_{Na} in the status of simulated long-time (>15 min) ischemia/reperfusion. (3) Effects of Atorvastatin in the status of simulated ischemic/reperfusion can be partly overcome by Wortmannin, which means atorvastatin can affect I_{Na} and the expression level of SCN5A through the way of RISK signal pathway especially of PI3K.