EFFECTS OF ANGIOTENSIN II AND TELMISARTAN ON TRANSIENT OUTWARD POTASSIUM AND L-TYPE CALCIUM CURRENTS IN SPRAGUE–DAWLEY RAT ATRIAL MYOCYTES

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Objective To explore possible electrophysiological mechanisms of single atrial myocyte by evaluating the effects of angiotensin II (AngII) and telmisartan on transient outward potassium currents (Ito) and L-type calcium currents (ICa-L) in Sprague–Dawley rats.

Methods Single atrial myocyte of SD rats was obtained by enzymatic dissociation method. The whole cell patch-clamp recording technique was used to record the change of Ito and ICa-L by intervening of AngII, telmisartan and AngII plus telmisartan, respectively. Experimental groups: (1) AngII group: cells were perfused with bath solution containing AngII (0.1 μmol/l); (2) Telmisartan group: cells were perfused with bath solution containing telmisartan (0.01 μmol/l); (3) Combined group, myocytes were perfused with bath solution containing AngII (0.1 μmol/l) and telmisartan (0.01 μmol/l).

Results Compared with baseline value, AngII (0.1 μmol/l), Telmisartan (0.01 μmol/l) and AngII plus Telmisartan group significantly decreased the peak density of Ito in SD rat atrial myocytes (22.48±2.75 vs 15.71±2.06 pA/pF, p<0.01), (24.16±2.36 vs 16.15±1.82 pA/pF, p<0.01) and (24.41±2.27 vs 21.35±1.46 pA/pF, p<0.05), respectively. AngII (0.1 μmol/l) significantly increased the peak density of ICa-L in SD rat atrial myocytes (−4.51±0.38 vs −5.16±0.29 pA/pF, p<0.01). Telmisartan (0.01 μmol/l) had no significant effect on ICa-L in the rat atrial myocytes (−4.35±0.27 vs −4.29±0.34 pA/pF, p>0.05), but it could antagonise the effects of AngII. In the Ang II combined telmisartan group, the peak density of ICa-L was (−4.08±0.28 vs −4.20±0.31 pA/pF, p>0.05), which was significantly different from that of AngII group (p<0.05).

Conclusion AngII and telmisartan had directly electrophysiological effects on SD rat atrial myocytes as well as telmisartan had antagonist effects on AngII at the level of angiotensin II receptor.