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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 angiotensinII (AngII) and telmisartan on transient outward potassium currents (I_{to}) and L-type calcium currents (I_{Ca-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 I_{to} and I_{Ca-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 $\mu\text{mol/l}$); (2) Telmisartan group: cells were perfused with bath solution containing telmisartan (0.01 $\mu\text{mol/l}$); (3) Combined group, myocytes were perfused with bath solution containing AngII (0.1 $\mu\text{mol/l}$) and telmisartan (0.01 $\mu\text{mol/l}$).

Results Compared with baseline value, AngII (0.1 $\mu\text{mol/l}$), Telmisartan (0.01 $\mu\text{mol/l}$) and AngII plus Telmisartan group significantly decreased the peak density of I_{to} 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 $\mu\text{mol/l}$) significantly increased the peak density of I_{Ca-L} in SD rat atrial myocytes (-4.51 ± 0.38 vs -5.16 ± 0.29 pA/pF, $p < 0.01$). Telmisartan (0.01 $\mu\text{mol/l}$) had no significant effect on I_{Ca-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 IIcombined telmisartan group, the peak density of I_{Ca-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 receptor.