GW23-e1391 **THE INFLUENCE OF ACH TO ISOLATED GUINEA PIG CARDIAC ATRIUM MYOCYTES IKACH**

doi:10.1136/heartjnl-2012-302920a.260

Ren Yanfeng, Wang Li, Wang Peng, Zhang Fan, Fei Yu. *Department of Cardiology, The Second Hospital Attached to Jilin University*

Objectives Atrial frillation (AF) is one of the common clinical arrhythmia and its pathogenesis is extremely complex. In recent years, scholars have observed that stimulating vagus nerve can induce AF. Acethlcholhe (Ach) is transmitter of vagus nerve and it gives its biological role through combination with M receptor. Acetylcholine sensitive potassium current (IKAch) is the current which induces AF. However, it is not clear about the relation between Ach and IKAch. So in this paper the change of isolated guinea pig cardiac atrium myocytes under the intervention of Ach is observed and it aims to further discuss the relation between vagus nerve and AF and provide a new thinking for the research of AF pathogenesis and better treatment.

Methods Take a healthy adult guinea pig. Beat the head to make it dizzy and then take out of the heart quickly. Under the temperature of 37°C and condition of oxygen, do Langendorff perfusion. Infuse by tyrode with calcium for 0.5 min to wash the remaining blood in the heart. Infuse by tyrode without calcium for 6 min to make the heart stop beating. Get atrium muscle along with atrioventricular groove and cut it. Infuse with by tyrode without calcium containing collagenase to digest atrium muscle. Put it into stock solution and shake it

for 10 min. Take supernatant liquor for centrifugation about 1 min and then throw away the supernatant liquor. Add stock solution and then put it into the fridge under the temperature of 4°C to preserve for standby application. Divide prepared cardiac atrium myocytes into 4 groups including control group, 0.01 μmol/l Ach, 0.1 μmol/l Ach and 1 μmol/l Ach. Use whole-cell patch clamp technique to record guinea pig cardiac atrium myocytes IKAch. During the operation, add $CaCl_2$ (200 μmol/l) into extracellular fluid in order to block calcium current and chlorine current of calcium activation. Use Glyburide (10 μmol/l) and Mg-ATP (5 mmol/l) in electrode inside fluid to block ATP sensitive potassium channel. Use chromanol 293B (20 μmol/l) and E-4031 (5 μmol/l) to respectively block Iks and Ikr channels; 4-AP excludes intervention of Ito and TTX excludes intervention of INa+.

Results

1. IKAch of control group is (9.3 ± 0.70) pA/pF; in 0.01 μmol/l Ach group IKAch is made to increase to (10.05 ± 0.72) pA/pF. But there is no substantial difference between the two groups ($p>0.05$); In 0.1 μmol/l Ach group IKAch is made to increase to (11.69±2.13) pA/pF. Compared with control group there is relatively remarkable difference ($p<0.05$); in 1 μmol/l group IKAch is made to increase to (13.36±2.91) pA/pF and compared with control group there is very remarkable difference ($p<0.01$). It is concentration dependent.
2. Respectively measure IKAch value corresponding to Ach under the voltage of +40 mV (−120 mV). After function of Ach, the value of IKAch increases. The computer draws its I–V curve and it can be seen that I–V curve moves up and the form of curve is not changed, that is to say, it has the function of promotion to IKAch and within a certain concentration range, the promotion to IKAch increases with the increase of concentration.

Conclusions Ach can increase amplitude of IKAch of cardiac atrium myocytes and promote IKAch of cardiac atrium myocytes to shorten action potential time interval and effective refractory period. Then foldback excitation is caused and finally it leads to the production of AF. So IKAch may be effect target of vagus nerve inducing AF.