[gw22-e0700]

## EFFECTS OF CYCLOVIROBUXIN-D ON INTRACELLULAR CA2+ LEVELS IN ISOLATED VENTRICULAR MYOCYTES OF DIABETIC RATS

Zhang qiang, Chen JiangxiProvincial People's Hospital, 300 Guangzhou Road, Nanjing, Jiangsu, China

10.1136/heartjnl-2011-300867.105

**Objective** To explore the effect of cyclovirobuxin-D (CVB-D) on intracellular calcium in electrically stimulated ventricular myocytes of diabetic rats.

**Methods** Diabetes was induced in male SD rats, using a single injection of alloxan into tail vein. Untreated age-matched animals were used as controls. The ventricular myocytes were isolated with collagenase (type 1) and the spectrofluorometric method was used to measure the intracellular calcium. Fura-2/AM was used as calcium fluorescence probe.

**Results** There was no difference of end-diastolic calcium and electrically stimulated intracellular calcium transient as well as peak transient between sixth week diabetic rats and control ($p>0.05$) CVB-D (0.1~1 μmol/l) showed no significant action on electrically stimulated intracellular calcium transient both of sixth week diabetic rats and control. However, CVB-D (2~5 μmol/l) increased the electrically stimulated intracellular calcium transient amplitude from control rats ($p<0.05$), whilst significantly decreased electrically stimulated intracellular calcium transient amplitude from sixth week diabetic rats ($p<0.01$) CVB-D (0.1~5 μmol/l) showed no significant action on end-diastolic calcium level from diabetic and control rats. Additionally, CVB-D 2 μmol/l, at perfusing 15 min, appeared potentiation on the electrically stimulated intracellular calcium transient from control rats and attenuation on that of diabetes. Finally, there appeared significant prolongation of time to peak and time to relaxation of calcium transient from diabetic rats ($p<0.01$). CVB-D prolonged time to peak from diabetic rats and control, but there was no difference between the two groups. While CVB-D showed no significant action on the time to relaxation of diabetic rats and control.

**Conclusion** The prolongation of calcium transient time probably was responsive for decline of diabetic heart function. CVB-D attenuated the amplitude of calcium transient and prolonged the time to peak of calcium transient from diabetic rats. This was probably associated with CVB-D-depressing myocardium contractile function from sixth week diabetic rats.