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RADIOFREQUENCY ABLATION OF CARDIAC NERVE ATTENUATES BEZOLD–JARISCH REFLEX IN CANINE

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Background The Bezold–Jarisch reflex (BJR) is one of the main causes for vasovagal syncope (VVS). Blocking the efferent pathways of the vagus ganglia in the heart may attenuate the BJR.

Objective To observe the influence of radiofrequency catheter ablation of the interventricular sulcus fat and endocardial nerve potential on Bezold–Jarisch reflex (BJR) induced by veratridine in canine.

Methods Six mongrel dogs were anaesthetised and their chest were opened through a right thoracotomy to expose the Interventricular sulcus fat. Veratridine was injected into the left ventricle at bolus doses of 15 µg/kg through a catheter to induce BJR. The sinus cycle length (SCL), systolic arterial pressure (SAR), diastolic arterial pressure (DAP), mean arterial pressure (MAP), left ventricle systolic pressure (LVSP), left ventricle diastolic pressure (LVDP) and mean left ventricle pressure (MLVP) were measured under the baseline and after ablation of fat pad; the changes of the above parameters (SCL,

SAP, DAP, MAP, LVSP, LVDP and MLVP) were calculated. Then the same dose of veratridine was injected under mapping along the mitral annulus, and potential nerve ablation and the changes of the above parameters were observed.

Results BJR was successfully induced in the 6 animals; the animals had prolonged sinus cycle length (SCL) and decreased MAP and MLVP. After ablation, the prolongation of SCL after injection of veratridine was significantly reduced (229.2 ± 92.3 ms vs 39.3 ± 14.1), $p < 0.01$). All the pressures were decreased after injection of veratridine, with significant decrease found only for DAP and MAP (both $p < 0.05$).

Conclusion Ablation of the interventricular sulcus fat and endocardial nerve potential fat can effectively attenuate the decrement of heart rate and the decrement of DAP and MAP during the BJR induced by veratridine, which provides a basis for the treatment of VVS