the LVOT, RVOT, RVA with or without vagal stimulation (VS) respectively.

Results 90% of MAP duration (MAPD90) under VS was significantly shorter than baseline ($p>0.05$). With or without VS, the MAPD90 of RVA were significantly shorter than RVOT and LVOT ($p<0.05$), while there was no difference of MAPD90 between RVOT and LVOT. With VS, the abbreviation of MAPD90 at LVOT and RVOT was greater significantly than RVA (MAPD90: 12.1±3.9 at RVOT, 14.8±5.5 at LVOT vs 8.3±4.1 at RVA, $p<0.05$), while there was no difference of MAPD between LVOT and RVOT ($p>0.05$).

Conclusions VS could reduce MAPD significantly. With VS, the abbreviation of MAPD90 at LVOT and RVOT was greater significantly than RVA. It suggested that outflow tract may be sensible to vagal modulation, which might be related to the occurrence of IVT.

Conclusions VS could reduce MAPD significantly. With VS, the abbreviation of MAPD90 at LVOT and RVOT was greater significantly than RVA. It suggested that outflow tract may be sensible to vagal modulation, which might be related to the occurrence of IVT.

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EFFECT OF VAGAL NERVE ON THE MONOPHASIC ACTION POTENTIAL OF VENTRICULAR OUTFLOW TRACT

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Objectives Vagal nerve may be related with idiopathetic ventricular tachycardia (IVT). The present study was aimed to investigate the effect of vagal nerve on the monophasic action potential (MAP) of ventricular outflow tract.

Methods Eight adult mongrel dogs were involved. Bilateral vagosympathetic tunks were decentralised for stimulation. Metoprolol was given to block sympathetic effects. MAP was recorded at