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Extended Abstract
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Heart Congress 2017- The Role of Autonomic Nervous System on Right Ventricular Outflow Tract Tachycardia- Hung-Yu Chang- Cheng Hsin General Hospital, Taipei, Taiwan

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Introduction

Ventricular tachycardia (VT) arising from the right ventricular outflow tract (RVOT) is one of the most common types of idiopathic ventricular arrhythmias . RVOT-VTs exhibit the following characteristics [2,3]: they usually occur in patients without overt structural heart disease, (2) the surface 12-lead ECG typically shows lei bundle branch block morphology and inferior axis, and (3) the tachycardia typically arises from a discrete area of the myocardium in the RVOT area. Although pathogenic mechanisms in the genesis of RVOT-VT remained largely unknown, activation of sympathetic tone has been shown to play an important role in provoking as well as maintaining these arrhythmias. He frequency of ventricular arrhythmias is oien increased during periods of wakefulness and activity, and they frequently disappear entirely during sleep. They are sensitive to catecholamine infusion, and typically terminate in response to betablockers, calcium channel blockers, and adenosine [5,6]. Heart rate variability studies showed activation of sympathetic tone prior to the occurrence of these ventricular arrhythmias [7-9]. Sympathetic fibers of the ventromedial cardiac nerve (VMCN) and branches of ventrolateral cardiac nerve (VLCN) innervate the myocardium within the proximal pulmonary artery (PA) and the RVOT [10]. Recently, an animal model has been described for RVOT tachycardia by high-frequency stimulation (HFS) of the extravascular sympathetic nerves within the PA innervating the RVOT [11]. Similarly, HFS in the lei pulmonary artery successfully induced RVOT-ventricular premature complexes (VPCs) and/or VT in a human model. However, in both models, the mechanism whereby a discrete area of myocardium in the RVOT becomes arrhythmogenic is yet to be clarified. Thus, the aim of this study was to investigate the neural mechanism of RVOT ventricular arrhythmias by using HFS within the PA in adult mongrel canine model

Methods

Twelve mongrel dogs (13.7 ± 1.3 Kg, 5 male dogs) were studied through midline thoracotomies. Highfrequency stimulation (HFS) was applied to the proximal pulmonary artery (PA) to induce RVOT VT/VPC. An EnSite Array and a mapping catheter were used for electroanatomical mapping. The RVOT and PA were surgically excised for immunohistochemistry studies, including tyrosine hydroxylase (TH) stain for sympathetic nerves and choline acetyltransferase (ChAT) stain for parasympathetic nerves.

Conclusion

HFS of the proximal PA could induce RVOT VT/VPC. The sympathetic nerves were densely innervated to the origin of RVOT VT/VPC, indicating the critical role of sympathetic hyperactivity in the initiation and perpetuation of RVOT VT/VPC.

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