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Evolving understanding of stress cardiomyopathy in critical care settings

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Stress cardiomyopathy (SC) is increasingly encountered in various settings. Originally it was considered as an acute coronary syndrome mimic presenting to the cardiologists with normal coronaries at the time of emergent catheterization. The increasing awareness, frequency of echocardiography and prolonged complicated intensive care course of older population all contribute to the ever increasing diagnosis of SC. Intensivists, surgeons, neurologists and hospitalizts are encountering SC as a possible/probable diagnosis during routine echocardiography. The characteristic apical ballooning variant conitnues to be the most prevalent manifestion in about 2/3 of the patients. Critical coronary occlusion in the left anterior descending artery could give rise to an identical wall motion abnormality (RWMA) in some instances. Either catherization to exclude disease or demonstartion of complete recovery of RWMA after 5-10 days is needed to confirm the diagnosis of SC. Unique patterns of RWMA are encountered in the form of mid or basal symmetrical akinesia in about a third of the SC patients. This RWMA is not consistent with any coronary distribution. The clincial suspicion based on cardiac signs (like ectopy, arrhythmias, ST segment changes, hypotension) and symptoms (like angina, dyspnea, orthopnea) combined with echocardiographic RWMA can confirm the diagnosis of SC at presentation. These basal and mid ventricular variants do not need catheterization or delay of 5-10 days for the repeat echocardiogram to make this important diagnosis. Specific heart failure and supportive management along with addressing the underlying medical issues will significantly improve outcomes without resorting to more cardiac procedures.

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Effect of low-level electrical stimulation of the arotic root ventricular ganglionated plexi on electrical and structural remodling in dogs with heart failure

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Background: Low-level electrical stimulation (LL-ES) of automomic nerve was reported to suppress atrial fibrillation (AF) by inhibiting the intrinsic cardiac autonomic nervous system and bring both anti- arrhythmia and anti-inflammation effect. However, it was still unknown whether LL-ES could reverse the structural remodeling of myocardial fibrosis and atrial enlargement following heart failure (HF)

Aim: This study was designed to investigate the anti-cardiac remodeling effect of LL-ES of the aortic root ventricular GP.

Method: Twenty dogs were randomly divided into control group and LL-ES group after rapid right ventricle pacing was performed to establish heart failure model. Following a week of LL-ES of the aortic root ventricular GP, bioactive factors for HF including angiotensin II, TGF-β, mitogen-activated protein kinase (MAPK), matrix metalloproteinase (MMP) was assessed. Furthermore, ventricle size, cardiac fibrosis as well as left ventricular ejection fraction were also determined.

Results: Compared by control group, expression of angiotensin II, TGF-β, MAPK, and MMP were significantly down-regulated in LL-ES group (P<0.05). Moreover, the volume of left ventricle and cardiac fibrosis were markedly decreased, and LVEF in LL-ES group was significantly increased compared with those in control group (P<0.05).

Conclusion: Long term LL-ES of the aortic root ventricular GP improved rapid pacing induced cardiac structural and functional remodeling by attenuating the sympathetic tone.

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