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Does human epicardial adipose tissue enhance atrial fibrillation induced by beta adrenergic stimulation in human cardiac muscles?

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Background: Atrial fibrillation (AF) is the most common type of cardiac arrhythmia. Beta (β)-adrenergic stimulation is a well-known trigger of AF. Excessive epicardial adipose tissue (EAT) around the heart is also a risk factor for AF. However, whether β-adrenergic activation stimulates EAT to promote AF in human myocardium remains unknown.

Purpose: To determine if human EAT enhances AF induced by β-adrenergic stimulation in human atrial muscles.

Methods: After informed consent, human right atrial appendages were obtained from patients that underwent open chest surgery (n = 20). From the tissue isolated muscles (trabeculae) were mounted in a bath, and the susceptibility of AF was measured based on the development of spontaneous contractions during one-minute rest period that followed one-minute stimulation at 1Hz (60 bpm) (automaticity protocol). The automaticity protocol was then repeated following 30 minutes of β-adrenergic stimulation (isoproterenol (ISO), 10-5M). In a separate group of patients (n = 10), trabeculae and a piece of human EAT (average weight = 53 ± 8 mg) was obtained. The EAT was situated upstream of the muscle in an adjacent bath, so the superfusion fluid of the EAT flows over the trabecula, and the automaticity protocol was performed during β-adrenergic stimulation (ISO, 10-5M) in the absence and presence of EAT in the adjacent bath.

Results: β-adrenergic stimulation increased the percentage of muscles with spontaneous contractions (No ISO: 0%, with ISO: 55%, p < 0.001, Chi-square test) and also the numbers of spontaneous contraction (No ISO: 0 vs. with ISO: 22±7 number of spontaneous contractions, p=0.001, Wilcoxon signed-rank test). In the second group, superfusion of the muscles with EAT alone did not induce spontaneous contractions in the muscles. Interestingly, when the EAT was triggered by β-adrenergic stimulation, 70% of the muscles superfused with EAT developed spontaneous contractions compared to 20% of the muscles without EAT superfusion (p=0.02, Chi-square test). However, there was no significant difference in the numbers of spontaneous contractions between the muscles superfused with or without EAT after β-adrenergic stimulation (No EAT: 12±10 vs. with EAT 14±8, number of spontaneous contractions, p=0.2, Wilcoxon signed-rank test).

Conclusion: The β-adrenergic stimulation-induced increases in the susceptibility for developing spontaneous contractions in human cardiac muscles was enhanced in the presence of human EAT. Thus, human EAT has a functional interaction with the human myocardium that affects the susceptibility for AF, specifically through modulation of the β-adrenergic arrhythmogenic trigger.