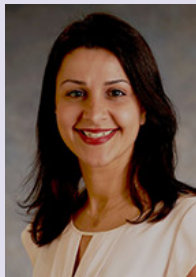


Neural Prosthesis Seminar

Parasympathetic dysfunction in cardiac disease and the role of vagal nerve stimulation in treatment of ventricular arrhythmias

Wednesday, August 23 • 3:00 PM

Wolstein Research Building, Room 1413
Case Western Reserve University



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Abstract

Myocardial infarction causes sympathetic activation and parasympathetic dysfunction, which act in concert to increase the risk of sudden death due to ventricular arrhythmias. Although blockade of the sympathetic nervous system has been invaluable in the treatment of patients with heart failure and ventricular arrhythmias, little is known about the mechanisms underlying parasympathetic dysfunction. New data is delineating consequences of myocardial infarction on parasympathetic myocardial neurotransmitter levels and on the function of parasympathetic cardiac and extra-cardiac ganglia, pointing to a decrease central parasympathetic drive as the reason behind the parasympathetic abnormalities observed in heart failure. Increasing parasympathetic drive with vagal nerve stimulation and elucidating its electrophysiological effects in the ventricle in the setting of chronic myocardial infarction could present an important avenue for treatment of patients heart failure and ventricular arrhythmias. However, the degree and frequency of vagal nerve stimulation has yet to be elucidated, particularly given the controversial results of this therapy in clinical trials of heart failure patients. Any effects of vagal nerve stimulation, however, on treatment of ventricular tachycardia after myocardial infarction could serve as important option for patients with cardiomyopathy, who present with VT storm and recurrent defibrillator shocks, despite standard medical therapy and ablation procedures.

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