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Autonomic Reflexes in Ventricular Extrasystoles as Possible Cause of Cardiomyopathy

Salavatian S, Yamaguchi N, Hoang J, Lin N, Patel S, Ardell JL, Armour JA, Vaseghi M. Premature ventricular contractions activate vagal afferents and alter autonomic tone: implications for premature ventricular contraction-induced cardiomyopathy. **Am J Physiol Heart Circ Physiol.** 2019 Sep 1;317(3):H607-H616. doi: 10.1152/ajpheart.00286.2019

Dysautonomias, characterized by sympathetic hyperactivity and reduced parasympathetic activity are usually associated with increased risk of myocardial infarction, heart failure, arrhythmias and sudden death. One of the most prevalent arrhythmias are premature extrasystoles. Several recent studies have demonstrated that frequent premature extrasystoles can activate the sympathetic nervous system, evidenced by changes in stellate ganglion neural activity, increased muscular sympathetic response, greater catecholamine release in coronary vessels and abnormal heart rate variability. In vivo experimental studies have also reported altered electrical activity of the intrinsic neurons of the heart. However, the mechanisms participating in parasympathetic reflex activity are unknown. The physiological changes produced in the heart are registered beat to beat by parasympathetic afferent sensitive neurons of the vagal nodose ganglion, activating reflexes which integrate in the central nervous system and modulate the sympathetic and parasympathetic efferent response. It is therefore logical to assume that these afferent pathways are modified by extrasystoles, and are thus implicated in the pathophysiology of the cardiomyopathy produced by high frequency ectopic ventricular contractions.

In this interesting work, Salavatian et al. studied the adverse influence of these arrhythmias in cardiac autonomic regulation in an experimental pig model with pacemaker-induced ventricular extrasystoles. They specifically demonstrated an alteration in the afferent neurons of the vagal nodose ganglion and the subsequent reduction in efferent parasympathetic

pathways. Surprisingly, they observed that extrasystoles induced at a frequency of one ectopic beat per five normal beats during one minute modulate vagal afferent pathways with a higher intensity than that of myocardial ischemia of equivalent duration. Another interesting finding is that afferent neurons which change their electrical activity may be activated both by mechanical and chemical stimuli. This multimodal activation could explain the highly intense nervous response in such a brief time interval. Although the experimental arrhythmogenic stimulus only lasts one minute, electrical recordings showed that nervous ganglion depolarizations persist for a longer period of time. Also, after an initial experimental period, neurons do not respond again with the same initial characteristics. These last two findings demonstrate the fast adaptation and memory generation that can be involved in the pathophysiology of arrhythmia chronicity.

Extrasystoles are commonly found in cardiac consultation, with a very variable prevalence depending on patient age and the existence or not of structural cardiac diseases. Even in patients without underlying disease, frequent ventricular extrasystoles are associated with increased risk of generating heart failure and cardiomyopathies which reverse with arrhythmia treatment. The precise mechanism by which these arrhythmias may lead to cardiomyopathy is not well known. Several studies suggest altered ventricular synchrony, intracellular calcium management or oxygen consumption as possible causes of cardiomyopathy. Other works have also suggested the contribution of sympathetic hyperactivity in the pathophysiology, given the known damaging effect of catecholamines. Salavatian et al. reinforce this last concept with a well-designed experimental study, involving the other component of the dual pathologic autonomic regulation: the reduction in the efferent parasympathetic response. Although these experiments were performed during very brief periods of time and in anesthetized animals, the persistence of reflex autonomic alterations after the interruption of extrasystoles suggests their possible participation as cause of structural heart changes.