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EDITORIAL

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Not so fast

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The relationship between the cardiac conduction system and myocardial function has been subject to decades of elegant research. Initial experiments were performed among various animals exposed to prolonged periods of rapid pacing. Relatively rapid heart rates persisting for several consecutive weeks resulted in reproducible abnormalities in cardiac ion channel function, action potential characteristics and, ultimately, cardiac performance. Altered calcium handling was branded a main culprit in this very complex process [1, 2]. Not only did incessant tachycardia impart a risk of mechanical failure to the ventricle, but it also affected a change in repolarizing potassium currents in a nonuniform manner among the myocytes. Such heterogenous remodeling can impart a risk of tachycardia-induced tachycardia, wherein one nuisance tachycardia sets the stage for another, more lethal one to develop. The most poignant application of this research in today's clinical climate is to the patient with atrial fibrillation. And there is no shortage of such patients. It has long been recognized that atrial fibrillation (AF) is the most common arrhythmia among the general population, with a prevalence that increases dramatically with age. Given the current trend toward an increase in the population's mean age, improved diagnostic tools for the detection of AF (event recorders and implanted loop recorders), the impetus to reduce AF-associated risk of stroke (via anticoagulation) and cardiomyopathy (via rate control), and the affinity of electrophysiologists to further develop left atrial ablation as a therapy, it is no surprise that the prevalence of diagnosed AF is rising exponentially. Keeping AF patients free from rapid ventricular rates, which can be asymptomatic until insidious onset of tachycardia-induced heart failure, is a worthy challenge. Indeed, attempts at maintaining sinus rhythm have thus far shown no advantage over a carefully monitored rate-control and anticoagulation strategy [3, 4].

In this issue of Cardiology Journal, Duszanska et al. [5] look beyond the usual culprits in tachycardia-induced cardiomyopathy to search for a relationship between atrioventricular reentrant tachycardia (AVRT), AV nodal reentrant tachycardia (AVNRT), and ventricular dysfunction. Given the known risk of cardiomyopathy associated with persistent junctional reentrant tachycardia (a misnomer for incessant tachycardia conducting anterograde via the AV node and using a slowly conducting accessory pathway as the retrograde limb) this question has some historical foundation. Indeed, ablation of the accessory pathway tends to resolve ventricular dysfunction in patients with persistent junctional reciprocating tachycardia [6–9]. To duly acknowledge the elephant in the room, however, AVRT and AVNRT tend to be neither incessant nor associated with symptoms of congestive heart failure. In fact, the typical patient with AVNRT tends to be a young woman who is otherwise healthy, aside from the symptoms that accompany episodes of tachycardia, which tend to persist for minutes to hours. Likewise for AVRT patients, except that they tend to be male. To be fair, evidence does exist that remodeling can occur even with nonsustained tachycardia. This has mainly been documented in terms of ventricular repolarization changes, and can be transient [10] or persistent [11], manifesting as T-wave abnormalities on ECG after termination of tachycardia. Recent evidence has suggested that tachycardia is not even required, as frequent ventricular ectopy from the region of the right ventricular

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outflow tract has proven to impair left ventricular function, with improved contractility after ablation of the ectopic focus [12, 13]. Along similar lines, Duszanska et al. [5] search for altered systolic and diastolic ventricular function in association with supraventricular tachycardia. After exclusions of numerous subjects deemed ineligible, transesophageal electrocardiogram (TEE) findings were compared among the remaining patients — 25 with AVNRT, 65 with AVRT and, somewhat remarkably, 50 healthy volunteers. Paired comparisons are then made for several baseline, systolic, and diastolic measures — in place of the more conventional ANOVA or Kruskal-Wallis test with subsequent correction for multiple pairwise comparisons. This culminates in a perceived difference in end-systolic diameter, end-systolic volume, ejection fraction, and fractional shortening, all of which favour the control group in terms of systolic function. A similar multitude of paired comparisons among diastolic parameters suggests reduced diastolic function with tachycardia compared to controls, but no difference between AVNRT and AVRT. Further exploration reveals no convincing relationship between systolic function and episode frequency, time since diagnosis, or heart rate. An apparent relationship between frequency of AVRT and ejection fraction is no longer evident after removal of one subject with outlying data. Frequency of tachycardia episodes do, however, seem to correlate with severity of diastolic dysfunction as measured by flow across the mitral valve and right upper pulmonary vein. Surprisingly, the actual frequency of episodes, along with time since diagnosis, and mean heart rate during episodes, is omitted from the manuscript. We are therefore left with the claim that frequent episodes cause incremental diastolic dysfunction, but no concept of 'how frequent'. Nonetheless, Duszanska et al. [5] have raised an important issue, and extended the scope of tachycardia-induced cardiomyopathy beyond the usual suspects of atrial fibrillation, persistent junctional reciprocating tachycardia, and frequent ventricular ectopy. Their manuscript raises the hypothesis that recurrent episodes of supraventricular tachycardia, even if brief, can impair ventricular function. If this hypothesis is confirmed in future studies, the clinical implication will be diligent suppression of tachycardia even if asymptomatic, or elimination of its substrate by ablation.

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