Cardiac Memory and Cortical Memory

To the Editor:

Dr Rosen and colleagues\(^1\) elegantly reviewed similarities between cortical and cardiac memory and presented new perspectives on the evolution of cardiac arrhythmias. We propose that, in addition to ion channels and gap junctions, myocyte size can also change in response to altered activation sequence and behaves like a “memory element.”

In developing cortical memory, neurons form new branches and synapses. The heart responds to altered electrical activation by differential, asymmetric hypertrophy, as observed during chronic ventricular pacing, a model for sustained monomorphic ventricular arrhythmias. Remote from the pacing site, pronounced growth (observed using echocardiography and microscopy [myocyte size]) occurs, but some atrophy is observed near the pacing site.\(^2,3\) These structural alterations are accompanied by an increase in QRS duration, indicating more pronounced asynchrony.\(^2,3\) Cell growth may play a role in this process, because hypertrophy and coinciding ventricular dilatation create a longer path for impulse conduction. Therefore, the cellular growth response would make the abnormal activation pattern more prominent over time. Thus, the hypertrophy response may contribute to cardiac memory.

Another similarity of the cellular growth response to the described properties of memory\(^1\) is that the cellular growth response appears to depend on the rarity of activation sequence. During chronic left ventricular (LV) pacing, the difference in the degree of hypertrophy between early and late activated regions averaged 43%.\(^2\) In contrast, abnormal activation originating from the right ventricle (RV) is associated with considerably less asymmetry (19% in hypertrophying dog hearts during RV pacing\(^3\) and 3% to 10% in patients with left bundle-branch block\(^4\)). These differences in degree of asymmetry occurred despite similar degrees of asynchrony and regional differences in mechanical work during LV and RV pacing.\(^5\) However, because the sequence of ventricular activation during LV pacing is approximately opposite to that during both natural sinus rhythm and RV pacing, it therefore is rarer. This pacing site–dependent growth response appears to match with the higher sensitivity to arrhythmias after left atrial than after right atrial pacing.\(^1\)

Finally, application of the “sensitivity to the rarest” hypothesis to pacemaker therapy (both alternate site and biventricular pacing) implies that the pacing site(s) should not only be chosen to reduce the degree of asynchrony, but also to create a sequence of activation as similar as possible to that during natural sinus rhythm.

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