Surgery makes patients happy

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As recently as 2 decades ago, most of us believed that the substrate for atrial fibrillation (AF) was thousands of stretched (and therefore ischemic) atrial cardiomyocytes firing randomly and completely independently. We also assumed that this essentially continuous electrical chaos left most of the atria refractory most of the time. The conceptual breakthrough occurred when Jim Cox¹ postulated the intuitively impossible (at least to me) idea that a pathway-seeking electrical impulse could continuously discover circuitous pathways of excitable (nonrefractory) atrial myocytes and successfully negotiate them to sustain a reentrant rhythm. Thus, AF is a reentrant rhythm.

For reentry to be sustained it obligates (1) slow conduction, (2) rapid refractoriness, and (3) unidirectional block, and it is facilitated by micro-anatomic heterogeneity. An impulse must take long enough traveling over a circuitous path for the origin to have had time to repolarize upon its return. Now, Jimmy reasoned that the larger the mass of myocardium, the more likely a wayward impulse would be able to find a sufficiently distant and time-consuming trajectory for the origin to regain excitability. So he suggested that we cut the atria up into strips, and any wayward impulse would be forced to stay local and arrive home too fast for reentry to be sustained. The good news is that Jimmy was right. The bad news was that with the early Cox-Maze procedures, after atrial slicing, the operative field looked like a lawnmower accident. So, surgeons and cardiologists began cutting corners with fewer incisions. The procedures were technically easier, but, the results were not as good.²

So, why not add ganglion plexus ablation to a limited Cox-Maze? Perhaps this would boost the results of this easier procedure. This strategy enjoyed conceptually intuitive promise. Efferent sympathetic preganglionic neurons that control cardiac function exit via the ventral rami of segments T1-T4 to the sympathetic chain and synapse on axons within the stellate ganglion at spinal levels T2-T4.³ Sympathetic denervation of the heart has been appended to therapies for many disparate diseases ranging from myocardial ischemia to thyrotoxicosis.

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Central Message

Making patients happy while recognizing and repairing reentry.

See Article page 972.

The accompanying important article⁴ is based on the Atrial Fibrillation Ablation and Autonomic Modulation via Thoracoscopic Surgery study,⁵ in which 240 patients underwent a thoracoscopic pulmonary vein isolation (a mini Cox-Maze procedure) and were preoperatively randomized to an additional ganglion plexus ablation or no additional therapy.

Unfortunately, the additional sympathetic neurectomy did not seem to help.⁵ For those of us in search of less-challenging surgical or interventional cardiologic procedures, the news is also bad: The old-fashioned cut-and-sew Cox-Maze III continues to offer significantly greater freedom from atrial fibrillation off medications than any other combination of lesion sets.²

Interestingly, the accompanying study⁴ does conclude that patients feel better (by Short Form-36) after thoracoscopic pulmonary vein isolation even if they have experienced a single recurrence. Not surprisingly, however, when patients experience multiple recurrences—especially if they experience complications—they are not happy. Most of us know this firsthand.

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