Neuromodulation for Ventricular Tachycardia and Atrial Fibrillation

A Clinical Scenario-Based Review

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Neuromodulatory therapies targeting sympathetic and parasympathetic pathways have been applied at multiple levels of the cardiac autonomic nervous system to treat ventricular tachycardia (VT) and atrial fibrillation (AF). Afferent nerve fibers are omitted for simplicity. CSD = cardiac sympathetic denervation; GP = ganglionated plexi; RDN = renal artery denervation; SCS = spinal cord stimulation; SGB = stellate ganglion block; TEA = thoracic epidural anesthesia; TNS = tragus nerve stimulation; VNS = vagus nerve stimulation.

The role of Marshall bundle epicardial connections in atrial tachycardias after atrial fibrillation ablation

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MB reentrant ATs accounted for up to 30.2% of the left ATs after AF ablation. Ablation of the MB-LA or CS-MB connections or ethanol infusion inside the VOM is required to treat these arrhythmias.

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Figure 4    Of the 31 Marshall bundle (MB)-dependent macroreentrant atrial tachycardias (MATs), 17 (54.8%) terminated at the MB-left atrial (LA) junction, 5 (16.1%) terminated at the MB-coronary sinus (CS) junction, and 7 (22.6%) were terminated with alcohol infusion inside the vein of Marshall (VOM). We were unable to terminate 2 MATs (6.5%) with radiofrequency energy, either endocardially at the MB-LA junction or epicardially at the MB-CS junction. We were further unable to identify or cannulate the VOM and thus unable to perform ethanol infusion. Of the 29 MB-dependent localized reentry circuits, 27 (93.1%) were terminated at the MB-LA junction, none were terminated at the MB-CS junction, and 2 (6.9%) were terminated after ethanol injection.

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Voltage during atrial fibrillation is superior to voltage during sinus rhythm in localizing areas of delayed enhancement on magnetic resonance imaging: An assessment of the posterior left atrium in patients with persistent atrial fibrillation

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CONCLUSION The correlation between low-voltage and posterior LA MRI-DE is significantly improved when acquired during AF vs sinus rhythm. With adequate sampling, mean AF voltage is a reproducible marker reflecting the functional response to the underlying persistent AF substrate.

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CONTEMPORARY REVIEW

Systematic review of biological therapies for atrial fibrillation

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Figure 1  Schematic outline of the biological therapies evaluated to date targeting the atrial substrate that perpetuates atrial fibrillation. Ach = acetylcholine; 
I_{Ca,L} = L-type calcium current; I_{Kr} = rapid delayed rectifier potassium current; TGF-β = transforming growth factor beta.
Atrial Tachycardia With Atrial Activation Duration Exceeding the Tachycardia Cycle Length

Mechanisms and Prevalence

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ATs with Atrial Activation Duration lasting longer than the TCL were present in approximately 10% of the ATs referred for ablation, mostly in ATs caused by localized re-entry. Ultra-high-density mapping allows detection of these complex patterns of activation.
FIGURE 1 Schematic of a Focal AT With AAD Longer Than the TCL

Activation breaks before the full activation from the previous one was terminated. AAD = atrial activation duration; AT = atrial tachycardia; TCL = tachycardia cycle length.

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Full cycle length activation along a localized circuit is shown from site 1 (reference point) to site 6 (yellow arrows). Sites 7 and 8 were lately activated from an anterior site, turning around the previous ablation lines, and then invading a gap before ascending into the PV antrum (red arrow). Black dots represent areas of block. AAD is calculated from site 1 (onset of activation) to site 8 (termination of activation) and then compared to the TCL. Note that this site is delayed compared to the next activation at site 1 (reference point) (dashed red line) (See Online Video 2). Abbreviations as in Figure 5.