Pacemaker Pro-Arrhythmia
Beyond Spike-on-T and Endless Loop Tachycardia*
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There is a longstanding love–hate relationship between pacing and tachycardias. In patients with profound bradycardia, pacing at physiologic rates is effective at preventing bradycardia-mediated ventricular tachycardia (VT) or ventricular fibrillation (VF). “Anti-tachycardia pacing” (1) has progressed from simple elimination of bradycardia by suppression by pacing at slightly faster rates (now somewhat controversial) to “preventive” algorithms and a host of stimulation sequences designed to terminate tachycardias, with most of the latter now a standard feature of modern implantable cardioverter-defibrillators (ICDs).

The pro-arrhythmic side of pacing was also recognized early. The pacing equivalent of the “R on T” is the “spike on T” that can precipitate VT/VF. (2) Perhaps the most common form of pacemaker pro-arrhythmia is the type of pacemaker-mediated tachycardia (PMT) known as endless loop tachycardia (ELT) (3). Endless loop tachycardia occurs when a premature atrial beat, either spontaneous or conducted retrograde by a ventricular premature depolarization (VPD), is sensed and generates a ventricular stimulus that produces a retrograde atrial beat. The sequence repeats itself, establishing the ELT.

Many patients with pacemakers (or ICDs) have tachycardias where the relationship to the pacemaker stimulus is less clear. In this issue of the Journal, Sweeney et al. (4) analyze the onset of tachycardia events in patients from 2 ICD trials (5,6) in an effort to categorize the various relationships between the pacer stimuli and subsequent tachycardias. The many subcategories and variations included in their exhaustive analysis make for difficult reading. This editorial seeks to clarify by adding a kind of annotation or “Cliff Notes” guide.

Classification
Non-pacing associated. This is very straightforward: no pacing within 5 cycles of the onset of VT/VF (Fig. 1). In the article by Sweeney et al. (4), this was the most common mode of VT/VF onset and was relatively evenly distributed among the various pacing modes.

Pacing associated. Pacing was present within 5 cycles before VT/VF initiation, with no short-long-short (S-L-S) sequence (Fig. 2A). This accounted for the next largest number of VT/VF episodes but is not equally distributed. It was rare with the Managed Ventricular Pacing (MVP) pacing algorithm, uncommon with VVI, and most common with DDD.

Pacing permitted. In this classification, VT/VF is initiated by an S-L-S sequence not caused by pacing stimuli but passively allowed by the device mode and lower rate (Fig. 2B). In the examples in the article by Sweeney et al. (4) (their Figures 1 and 3), there is no pacing. The distinction between “pacing permitted” and “non-pacing associated” seems to boil down to whether there is an S-L-S sequence. This mode of onset was fairly equally distributed but least common with DDD pacing.

Pacing facilitated. Single ventricular pacing stimuli initiate or terminate pauses prematurely, producing an S-L-S sequence followed by VT/VF (Fig. 3). This sequence was most common with DDD, less so with VVI, and least with MVP.

Comments
Sweeney et al. (4) have worked mightily to parse and classify the various types of VT/VF onsets recorded in 2 ICD trials. They have added several new terms to the lexicon that are likely to endure. One challenge to the general adoption of the formulations of Sweeney et al. (4) lies in the complexity and thoroughness of the analysis. The condensed version in this editorial might serve to whet the appetite for the details in the article by Sweeney et al. (4).

The role of S-L-S sequences is an interesting finding and raises some thought-provoking questions.

VT/VF onset and pacing. The S-L-S sequences were unusual in “non-pacing associated” and “pacing-associated” events, whereas S-L-S sequences were the norm in “pacing permitted” and “pacing facilitated” events. The MVP mode often produces S-L-S sequences and yet had the lowest incidence of pacing facilitated VT/VF. Of the 1,356 VT/VF episodes, 356 (26%) were “pacing permitted” or “pacing facilitated” (gleaned from their Table 2 and text). This is
actually a rather disturbingly high number. However, the reader should keep in mind that the terminology is descriptive; we do not know with certainty that the VT/VF was caused by “pacing permitted” or “pacing facilitated” sequences. **Torsade de pointes.** Torsade de pointes typically begins with S-L-S sequences, but most of the episodes induced by S-L-S sequences seem to have been monomorphic VT.

**Atrial fibrillation and S-L-S.** Neither of the trials excluded patients with atrial fibrillation. Patients with atrial fibrillation have many S-L-S sequences that can induce VT/VF (7) but usually do not.

**Other arrhythmias and pacing modes.** The article by Sweeney et al. (4) relates to VT/VF; however, similar sequences can be seen to induce supraventricular tachycardia (SVT) (Fig. 4). “Orthorhythmic” pacing (8) is a “preventive” technique that operates with algorithms that can produce S-L-S intervals as part of the preventive strategy. In contrast, “rate smoothing” pacer algorithms might be effective in preventing some S-L-S sequence-related VT/VF episodes (9).

The work of Sweeney et al. (4) will inspire many of us to rethink and reconsider the whole topic of pacemaker proarhythmia. Good job!
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REFERENCES