Overcoming the Defects of a Virtue

Dual-Chamber Versus Single-Chamber Detection Enhancements for Implantable Defibrillator Rhythm Diagnosis: The Detect Supraventricular Tachycardia Study

Michael O. Sweeney, MD

“Mission critical” is a uniquely American cliché generally held to mean an action or series of actions essential for the success of an important endeavor. Randomized clinical trials have conclusively demonstrated that implantable cardioverter-defibrillators (ICDs) reduce mortality among appropriately selected patients who have survived an episode of life-threatening ventricular arrhythmia or are at risk for ventricular arrhythmia. The mission critical aspect of ICD operation is accurate recognition and termination of ventricular arrhythmia. Reproducibly reliable delivery of this critical series of actions is sufficient to guarantee mortal benefit.

Recognition of ventricular arrhythmias is fundamentally a sequential 2-step process. The first step, sensing, requires continuous real-time recording of cardiac electrical activity that is processed and filtered to reduce noise and to exclude physiological signals that are not of interest. Practically speaking, only ventricular electrical activity is necessary for this step and could be obtained from several different recording sites (endocardium, epicardium, subcutaneous tissue). The second step, detection, requires characterization of sensed signals to inform the need for therapy. ICDs rely on the rate of sensed ventricular signals for detection and hierarchical prioritization of specific therapies. Rate-based detection alone has proved robust for accurate recognition of ventricular fibrillation (VF) because, except for preexcited atrial fibrillation (AF), virtually no other cardiac rhythm disturbance is capable of achieving similarly high ventricular rates. On the other hand, although rate-based detection alone is similarly reliable for relatively slower ventricular tachycardia (VT), it is critically flawed because (1) VT occurs across a broad range of rates, and within this range rate alone does not specify rhythm, (2) neither fast and lethal nor slow and harmless are synonymous, and (3) many supraventricular tachycardias (SVTs) reliably achieve ventricular rates overlapping with VT. Thus, the flaw of rate-based detection for VT is a defect of its virtues.

Consequently, this sequential 2-step process is modified to include detection enhancements. The purpose of detection enhancements is to selectively identify true VT for appropriate therapy and to selectively reject SVT with overlapping ventricular rates to avoid indiscriminate therapy. Detection enhancements are applied after rate-based criteria are satisfied, and they are updated continuously until the ventricular rate slows spontaneously or a therapy is delivered. The simplest detection enhancement is time. A specified waiting period (in units of time or ventricular intervals) before a therapy is delivered after rate-based detection has been satisfied can effectively reduce premature therapies for true VT and inappropriate therapies for SVT because either may spontaneously slow or terminate. Time duration alone is incapable of distinguishing between VT and SVT and has obvious practical limitations related to patient safety; therefore, much effort has been spent developing techniques for rhythm discrimination.

The basis for discrimination is identifying and exploiting distinguishing characteristics of VT compared with SVT. The process is similar to the systematic approach to diagnosing VT using surface ECG in which more information, generally speaking, increases the probability of a correct diagnosis. Such information includes ventricular rate and regularity, onset and offset characteristics, atrial rhythm, atrial and ventricular timing relationships, electrical axis, and distinguishing features of the QRS complex (shape, notching, etc) relative to sinus rhythm. However, the availability of information for analysis in ICDs is influenced by the hardware system. The ventricular signal for sensing and detection is recorded from a single site (usually the right ventricular apex) and is common to all ICD systems. This is sufficient for calculating tachycardia onset and regularity, which could be useful for discrimination because sinus tachycardia is usually of gradual onset whereas VT starts suddenly and because AF is characterized by irregular ventricular intervals whereas VT is typically regular (or stable).

Accordingly, use of onset and stability alone may yield specificity as high as 96% for rejecting sinus tachycardia and AF at ventricular rates <180 to 190 bpm in single-chamber ICDs. The specificity for AF using ventricular interval stability alone declines significantly at higher rates (>190 bpm) because of regularization. In either situation, the penalty for the improved specificity using this simple approach to inappropriate therapy inhibition is a reduction in sensitivity for detection of true VT to 80% to 90%, which is fundamentally unacceptable. The addition of a therapy inhibi-
itor override (high-rate timeout), after which therapy is delivered regardless of rhythm discrimination if ventricular rate criteria are still satisfied, guarantees 100% detection sensitivity for true VT but erodes detection specificity.1

Because VT is characterized by a change in ventricular activation sequence, morphological features of the ventricular electrogram (EGM) also could be useful in diagnosing VT. This can be achieved with single-chamber or dual-chamber ICDs using various ventricular EGM sources. Atrial rhythm analysis and AV timing relationships also might enhance rhythm discrimination but require a dual-chamber ICD. The hybridization of such advanced detection enhancements to simple techniques such as onset and stability would be reasonably expected to increase ventricular rate–based detection specificity without compromising the goal of 100% sensitivity.

Interestingly, this has been difficult to prove in clinical investigation. Regardless of increasingly sophisticated detection enhancements, the specificity for ventricular rate-based detection resides around 70% to 90%.3–7 Likewise, small prospective randomized studies have not demonstrated a consistent advantage of dual-chamber over single-chamber detection enhancements.8–10 Further complicating matters is that all forms of detection enhancement are nominally off in most ICD systems, and physician awareness of the value of such enhancements is inadequate. This exasperating set of conditions recently led to an experiment wherein patients were randomized to purely rate-based detection versus one specific approach to dual-chamber detection merely to demonstrate that “any” detection enhancements are superior to none whatsoever.11

The Detect SVT study12 reported in this issue of *Circulation* randomized 400 patients who received dual-chamber ICDs for conventional indications to single- or dual-chamber detection. The primary end point was the proportion of expertly adjudicated SVT episodes that met ventricular rate detection criteria and were inappropriately classified as VT. Slightly more than 30% to 40% of patients overall had SVTs during follow-up, which accounted for a disturbingly large proportion of all ventricular detections in either treatment group (42% single chamber versus 69% dual chamber). After correction for multiple episodes within the same patient, the inappropriate detection rates were 46.5% versus 32.3% for the single- versus dual-chamber groups, respectively. Thus, dual-chamber enhancements reduced overall inappropriate detections by nearly 50% (odds ratio, 0.53; 95% CI, 0.30 to 0.94) compared with single-chamber detection. The clinical consequence of improved rejection of VT episodes that met ventricular rate-based detection was a 46% reduction in the odds of inappropriate therapies in the dual- versus single-chamber group (adjusted rates, 38.3% versus 26.1%, respectively). However, the inability of this study to demonstrate a reduction in inappropriate shocks in the dual-chamber group despite a reduction in misclassification of SVTs as VT is a huge disappointment. This is a direct result of failing to specify standardized tachycardia therapy programming between treatment groups. Serendipitously, a greater use of antitachycardia pacing (ATP) in the single-chamber group reduced overall shocks despite a higher rate of misclassification of SVT that received inappropriate ventricular therapies. It is well recognized that ATP may prevent shocks for inappropriate detections by >1 mechanism such as terminating AV node–dependent SVTs, delaying shocks long enough to permit spontaneous slowing or termination of VT, and slowing of SVTs by concealed retrograde penetration of the AV node. Furthermore, the success of ATP when broadly applied to detection rates as high as 250 bpm results in most shocks being delivered for misclassified VT.13

Several other observations merit emphasis. The prescribed exclusion of a high-rate timeout is a notable departure. The authors justified this choice on the basis that therapy inhibitor overrides inevitably degrade detection specificity. Whether a therapy inhibitor is more likely to interrupt an appropriate therapy withhold for VT that meets ventricular rate detection criteria or to interrupt an inappropriate therapy withhold for true VT has not been carefully studied and probably varies by patient, arrhythmia mechanism, discrimination technique, and other factors. The clinical consequence of this decision in the Detect SVT study was that only 93% of true VT episodes overall were appropriately detected. The authors offer the reassurance that only 24% of these true VT episodes that were misclassified as VT were sustained (lasting >30 seconds), implying that the remaining 76% were clinically irrelevant because they terminated spontaneously. We are not told how the sustained, misclassified true VT episodes terminated and whether they were associated with symptoms (ie, syncope). Furthermore, although it is well known that many episodes of VT terminate spontaneously during capacitor charging,13 there is an important distinction between this situation and serendipitous termination of VT misclassified as SVT, which is operationally a device failure.

The difficulty of properly classifying SVTs with a 1:1 AV relationship also is highlighted by this study. These rhythms are often sudden onset (simulating VT) and regular (also mimicking VT and rendering ventricular interval stability criteria useless). The situation is even more complex when there is a varying AV timing relationship resulting from discontinuous AV node behavior at high atrial rates, which mimics AV dissociation, a hallmark of VT. Although the use of ventricular EGM morphology4 would seem particularly helpful in this situation, specificity with the technique used in the Detect SVT study has been reported to be only ~70%.3 Rhythm discrimination using template matching based on ventricular EGM morphology analysis is predicated on 2 fallible assumptions: that the template sinus rhythm EGM (which must be periodically updated) is constant and that the VT EGM is always significantly different from the template. Other potential reasons for algorithm failure such as template misalignment, maturation, and postshock distortion have been reviewed.4 Whether another approach to ventricular EGM morphology4,14 would perform superiorly is unknown and difficult to establish because the number of patients studied influences comparisons between different techniques.15

Finally, SVT detections were registered in the VF zone in nearly 20% of patients. Traditionally, all ventricular rhythms detected at rates >180 to 190 bpm are classified as VF with the consequences that shocks are nominally applied and
Detection enhancements are not applied. This historical practice should be abolished because ~70% to 80% of all true ventricular rhythms in the VF zone are actually fast VT that can be painlessly and safely terminated with ATP. It seems reasonable that application of detection enhancements in the VF zone would further reduce inappropriate detections.

The Detect SVT study provides some modestly reassuring evidence that fits with intuition and experience; namely, when properly applied, more information about the distinguishing characteristics of atrial versus ventricular arrhythmias should increase detection specificity without excessively jeopardizing patient safety. On the other hand, inappropriate therapy rates >25% despite increasingly sophisticated detection enhancements cannot be viewed as satisfactory. Although the authors emphasize the distinction between inappropriate detections and therapies, every inappropriate detection risks exposing the patient to a full sequence of therapies. The likelihood of an inappropriate therapy for misclassified SVT significantly exceeds the likelihood of an appropriate therapy for VT in primary prevention patients. One of the principal limitations of ICD therapy is the discomfort associated with shocks. A direct correlation between poor quality-of-life scores and painful shocks has been described in trials of primary and secondary prevention. The results of the Detect SVT study should further inform physicians about the value and limitations of detection enhancements in optimal ICD therapy. More importantly, the impact of inappropriate therapies on quality of life and costs should become a central part of the national dialogue about the choice of specific hardware for ICD therapy.

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