Ensuring Automatic Detection of Ventricular Fibrillation

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The sensitivity and specificity of ventricular fibrillation (VF) detection by automatic implantable cardioverter-defibrillators (ICDs) have received relatively little attention by clinical investigators compared with other aspects of defibrillator use. The paucity of reports on ventricular fibrillation detection is not related to its lack of importance but rather reflects how difficult it is to confirm or refute the reliability of a device's detection system.

Ventricular fibrillation, by its very nature, is a complex and variable process, and automatic detection poses a substantial engineering challenge. ICD devices must discern very low amplitude signals that fluctuate both rapidly and gradually; avoid double sensing QRS complexes; reject ambient electromagnetic interference; reject myopotentials; reject cardiac repolarization signals; count the intracardiac signals yet not count too readily lest every run of nonsustained ventricular tachycardia be treated and not too slowly lest syncope and ischemia supervene; know which tachycardia it must treat; be prepared to change therapy should an arrhythmia change; redetect an arrhythmia, possibly a different one after therapy; contend with signal alterations following a shock; and confirm that therapy was successful. Thus, with so many tasks for a device to perform, it is no surprise that problems can arise with some aspect of the process.

In a recent issue of Circulation, Jung and colleagues identified a problem with several ICDs when connected to a new endocardial lead system that failed to redetect VF after an unsuccessful defibrillation pulse. In five patients undergoing transvenous cardioverter-defibrillator implantation, an initially unsuccessful high-energy defibrillation pulse was demonstrated to diminish VF electrogram amplitude sufficiently to cause failure to redetect VF. This problem is disconcerting because redetection of VF is rarely tested with the complete ICD device lead system during ICD implants. The authors suspect that this phenomenon was a direct consequence of the transvenous lead system design that uses a right ventricular coil electrode for defibrillation as well as for pacing and sensing. Such a lead design is customarily called an integrated bipolar sensing system.

In the study by Jung et al, substantial decreases in electrogram amplitude were noted after a shock in both sinus rhythm and VF. For a successful defibrillation pulse, sinus rhythm amplitude fell from 10.5±3.8 mV before VF to 1.9±1.2 mV. This was well below the sinus rhythm electrogram amplitude implant criterion of 5 mV. For an unsuccessful defibrillation shock, the continued VF electrogram amplitude diminished from 6.3±1.9 mV before shock to 2.2±1.3 mV after shock. The consequences of this large diminution in electrogram amplitude was delay or failure to redetect VF by the cardioverter-defibrillators used in this study. The authors found the postshock alterations in intracardiac electrogram amplitude sufficiently worrisome to avoid further implantations of devices that use integrated bipolar lead systems. The authors argue for use of true bipolar sensing from separate electrodes that are relatively far removed from the defibrillation electrodes. The long and good experience with true bipolar epicardial sensing supports this view.

The decreases in intracardiac electrogram amplitudes following a high-energy defibrillation shock, as observed in the Jung et al report, is probably a temporary tissue effect caused by the high current density in the tissue surrounding the high-voltage electrodes during the shock. These tissue effects and their influence on subsequent sensing depend on both the ICD circuitry and lead system design. Other investigators have observed these effects for integrated bipolar leads. Kühlkamp et al and Isbruch et al reported similar effects with the same lead system, and Yee et al with a now-extinct older integrated lead system, have reported diminution in electrogram amplitude after a shock. The duration of the effect is typically several minutes. Isbruch et al also observed the magnitude of the decrease in electrogram amplitude after shock to be proportional to shock amplitude. Postshock pacing thresholds have been observed to rise as well. These observations with integrated bipolar electrode systems stand in contrast to those observed with true bipolar sensing configurations where such events have not been observed. Usually, true bipolar sensing ensures prompt and reliable redetection of VF because the sensing electrodes are removed from the shock and therefore less likely to experience the full effect of shock-induced tissue dysfunction. True bipolar sensing also minimizes the possibility of electrode polarization effects at the electrode-tissue interface.

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Dose-dependent cellular injury following exposure to an electric field has been known for some time.\textsuperscript{8–12} High-energy pulses have been shown to decrease resting membrane potentials by creating holes in the cell membrane bimolecular lipid layer, like holes in an insulative dielectric.\textsuperscript{11,12} This electroporation process creates holes large enough to allow cytosolic sodium influx and/or potassium efflux and, at very high field strengths, cytosolic calcium overload.\textsuperscript{11} These ionic shifts in turn reduce resting membrane potentials, prolong depolarization, and decrease peak activation. Electroporation occurs in proportion to the amplitude of the shock, the number of shocks, and the time interval between shocks.\textsuperscript{9–11} The time period for recovery of the cell membrane, a self-scaling process of the bimolecular lipid layer, can take seconds to minutes accounting for the time dependent nature of the electrogram changes observed in the studies by Jung et al,\textsuperscript{1} Kühlkamp et al,\textsuperscript{2} Isbruch et al,\textsuperscript{3,4} and Yee et al.\textsuperscript{5,6}

Ensuring reliable detection of a variable phenomenon such as VF, especially after a high-energy shock, requires the sensing circuitry to have either an automatic gain control or an automatically adjusting sensitivity threshold.\textsuperscript{13,14} Devices without such features have had trouble detecting the rapid changes in electrogram amplitude typically observed during VF even when the compounding problem of electroporation is not present.\textsuperscript{15} Automatic gain control or an automatically adjusting sensitivity threshold does not, however, guarantee VF detection when other features of the system are deficient. In Jung et al’s report, the devices employed had automatic gain control, yet the problem of undersensing low-amplitude VF occurred.

Some leakage of the shocking current to the sensing electrodes can also cause re-detection problems and may explain the findings in Jung et al’s report. If any part of the defibrillation current flows through the small surface area of the sensing electrodes, whether part of an integrated system or a true bipolar system, the high current density in the adjacent tissue may reduce the electrogram amplitude.

Jung et al have shown that altering the sensing electrodes of an ICD lead system can cause total system failure even though most aspects of the system have stood the test of time. A detection system is an amalgam of electrodes, electronics, and software that interfaces with a complex dynamic biological system. Each ICD device has different generator circuitry and detection algorithm software that may interact differently with each lead system. This complexity requires that each combination of an ICD device and lead system be tested in humans in the usual rigorous investigative manner before broad clinical use.

References


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