Successful defibrillation by modifying from fixed-tilt to tuned-duration defibrillation waveform: When less is more?

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Introduction
Implantable cardioverter-defibrillator (ICD) therapy has become more efficient owing to changes in waveform morphology and output. Current ICD technology is capable of delivering 35–40 J, raising the question of the value of routine defibrillation threshold (DFT) testing. However, when DFT testing is done in specific populations, elevated DFTs have been reported in 2.2%–12% of subjects.1 Furthermore, Siddiqi and colleagues2 reported that patients with high DFTs have a higher mortality than those with acceptable DFT. Identifying patients with potentially high DFTs or system failure is difficult with the tools of ICD interrogation and programming. Reducing this defibrillation failure risk, with a low DFT, is an important goal.

Defibrillation efficacy is altered by noninvasively modifying the shock waveform.3 Herein we report a case of noninvasive improvement of defibrillation efficacy by changing from a fixed-tilt to a tuned-duration defibrillation waveform, preventing excessive extension of shock pulse duration with a fixed-tilt defibrillation waveform.

Case report
A 58-year-old male patient with dilated cardiomyopathy and ventricular fibrillation (VF) had an ICD (Secura DR; Medtronic Inc, Minneapolis, MN), with Durata single-coil defibrillation lead (Model 7122; Abbott Inc, Sylmar, CA) and CapSureFix atrial lead (Model 5076; Medtronic Inc) implanted in 2009. Defibrillation testing was successful at 9 J.

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Subsequently, the patient was hospitalized for heart failure repeatedly, with low left ventricular ejection fraction (26%); the ICD was upgraded to cardiac resynchronization therapy defibrillator (CRT-D) therapy in 2010 and the patient required a surgical mitral valve replacement in 2014. The CRT-D generator was replaced, owing to battery depletion, by a Unify Assura (Abbott Inc) in 2016. At that procedure, DFT was not performed and fixed-tilt waveform (phase 1 65% / phase 2 65%) was programmed. In December 2019 the amiodarone dose was reduced from 100 mg to 50 mg daily. The patient had no sustained ventricular arrhythmia episodes after the upgrade.

In June 2020, his family noticed that while working on the farm, he lost consciousness, the CRT-D delivered shock therapy, and he immediately recovered consciousness. After waiting 20 minutes, he was transported by ambulance to the hospital. During transport, his electrocardiographic monitor revealed VF and shock therapies of CRT-D did

KEY TEACHING POINTS
- There is a possibility that high defibrillation threshold (DFT) is caused by extended shock pulse duration owing to high shock impedance by fixed-tilt waveform. In particular, extreme extension of pulse duration in second phase results in the elevation of DFT.
- There are cases in which defibrillation efficacy can be modified noninvasively by changing the programming from fixed tilt to tuned pulse duration.
- DeFT Response (Abbott Inc, Sylmar, CA), which is the option to program a patient-adjusted shock pulse duration, has clinical value, enabling an estimate of defibrillation safety with fewer inductions of ventricular fibrillation and defibrillation tests.
not terminate the VF; eventually, external electrical defibrillation rescued the patient, with return of spontaneous circulation. Device interrogation detected 2 VF events induced by short-coupled premature ventricular contraction when the function of automatic threshold capture was activated. Fusion beat preceded by PVC resulted in first and second loss of capture (LOC). Third LOC occurred by automatic threshold capture test. Finally, VF was induced by short-coupled PVC. A-EGM = Atrial Electrogram; AS = atrial sensing; BP = biventricular pacing; F = VF zone sensing; V-EGM = Ventricular Electrogram; VS = ventricular sensing.

Troubleshooting of the event
There were no metabolic abnormalities at admission. The automatic capture threshold function was deactivated to reduce the risk of inducing VF. Amiodarone was discontinued and sotalol was initiated to prevent ventricular arrhythmias and to potentially reduce the DFT. We then attempted to optimize the defibrillation waveform. We observed that with the fixed-tilt (phase 1 65% / phase 2 65%) waveform the pulse duration was automatically extended, with phase
1 at 10 ms and phase 2 at 10 ms owing to high shock impedance (84 Ω). Therefore, we changed from a fixed-tilt to a tuned-duration calculated defibrillation waveform (phase 1 at 4.5 ms and phase 2 at 2.0 ms) based on DeFT Response (Abbott). As a result, defibrillation was successful at 28.4 J.

**Discussion**
Defibrillation efficacy can be modified noninvasively through device programming by adjusting the shock waveform or shock polarity, or altering the shocking vector. If noninvasive approaches fail, a more invasive procedure, such as replacing with a higher-energy device or adding shocking coils, can be attempted. The present case presented multiple risk factors contributing to high DFT: progression of heart failure disease status, low left ventricular ejection fraction, and oral treatment of antiarrhythmic medications (amiodarone).\(^4\) Another contributing factor that reduced defibrillation efficacy was an automatically extended shock pulse duration (phase 1 10 ms / phase 2 10 ms) owing to high shock impedance of 84 Ω by fixed-tilt (phase 1 65% / phase 2 65%) waveform.
Phase duration and DFT
Appropriate biphasic shocks can result in reductions in DFT. However, extreme extension of pulse durations in the shock pulse wave, especially in second-phase duration, can result in the elevation of the DFT. Extension of the second-phase duration induces hyperpolarization, and defibrillation efficacy is inversely related to the residual membrane voltage at the end of phase 2, as indicated by previous reports. Actually, in a randomized prospective clinical study, mean DFT was significantly lower with a shorter phase 2 duration, 9.5 ± 4.5 J with 2.0 ms vs 11.3 ± 5.2 J with 5.0 ms; $P < .0001$). Since the goal of phase 2 is to reverse the membrane-charging effect of phase 1, there is no advantage to additional waveform phases. In the present case, phase 2 duration was automatically extended to 10 ms because of the high shocking impedance noted upon admission; therefore the shock pulse waveform needed to be modified.

Fixed-tilt defibrillation waveform vs tuned-duration defibrillation waveform
Regarding defibrillation, ICDs use either fixed-tilt or fixed-duration waveform, depending on the manufacturer. These approaches result in different waveform duration and different changes in waveform duration for varying pathway resistance, as shown in Figure 3. The more common approach is to use predetermined phase 1 and phase 2 tilts. In a tilt-based approach, both pulse wave durations change proportionately to the pathway resistance. Although the optimal duration of phase 2 decreases with increasing resistance, tilt-based waveform reverses the response. With the duration-based approach, the optimal waveform duration is provided with respect to impedance. Previous investigations reported that for the patients whose tilt-based DFT was ≥15 J, the DFT was reduced from 18.7 ± 4.1 J to 13.4 ± 3.5 J, for a mean DFT reduction of 28% ($P = .009$) using fixed-duration phase 1 and phase 2 defibrillation.

In the present case, high shock impedance (84 Ω) resulted in a dysfunctional extension of the pulse duration (phase 1 10 ms / phase 2 10 ms) and failure of defibrillation at 40 J. When we modified the programming from fixed-tilt to tuned pulse-duration (phase 1 4.5 ms / phase 2 2.0 ms) defibrillation waveform, defibrillation was successful at 28.4 J.

DeFT Response
Abbott Inc ICDs provide the option to program a patient-adjusted shock pulse duration to achieve a low DFT, called the DeFT Response. DeFT Response provides a suggested value of appropriate adjustment of the first and second phase of the waveform as an aid in achieving adequate safety margins. It is important when attempting to achieve an adequate defibrillation safety margin to match the time constant of the system to that of the patient’s own cardiac tissue time constant ($\tau$). Direct measurement of $\tau$ is not possible outside of scientific laboratories. Therefore, by measuring the duration of an evoked potential using a custom electrogram from the tip of the right ventricular (RV) lead to RV shocking coil during RV pacing, it determines an estimated cardiac time constant in a practical manner. Thus we can calculate the optimal pulse duration based on this measurement using the DeFT Response. Previously Conelius and colleagues reported the successful example of converting VF to sinus rhythm by programming the device to the optimal pulse width based on cardiac membrane time constant.

DFT management using ventricular stimulation to measure the time constant ($\tau$) has clinical value, enabling an estimate of defibrillation efficiency (DeFT Response) with fewer inductions of VF and defibrillation tests. However, there have been no reports to provide the efficacy of DeFT Response in a case with demonstrated defibrillation failure.
To the best of our knowledge, this is the first report of successful defibrillation using DeFT Response against the excessive extension of shock pulse duration of a fixed-tilt defibrillation waveform in a case of defibrillation failure.

Conclusion
Defibrillation efficacy is determined in part by the defibrillation waveform. Since fixed-tilt waveforms are dependent upon defibrillation impedance, there are cases, such as presented, where the fixed-tilt waveform will be unsuccessful and where adjusting the waveform produces a more acceptable result. Optimal waveform modification using a tuned-duration defibrillation waveform should be considered in cases with high defibrillation threshold.

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