implantable cardioverter-defibrillators (ICDs) substantially reduce sudden cardiac death. Paradoxically, even though ICDs are implanted to prevent sudden cardiac death by delivering therapies, appropriate and inappropriate ICD shocks have been associated with an increased risk of mortality.1 However, patients treated with ICD shocks show even poorer survival than patients treated with antitachycardia pacing (ATP) only.2 Important studies like the PainFREE Rx II (Pacing Fast Ventricular Tachycardia Reduces Shock Therapies),3 ADVANCE III (Avoid Delivering Therapies for Nonsustained Arrhythmias in ICD Patients III),4 and the MADIT-RIT (Multicenter Automatic Defibrillator Implantation Trial–Reduce Inappropriate Therapy)5 trials have markedly improved our standards of care. With this knowledge, sophisticated programming of devices has by now become an important component in long-term care for ICD patients, ensuring optimized detection rates, detection duration, supraventricular tachycardia discriminators, and programming of ATP.6

Terminating ventricular tachycardia (VT) by rapid ventricular burst stimulation was introduced about 50 years ago.7 First experiences were reported with an implanted pacemaker for terminating supraventricular tachycardia by patient activation via a magnet.8 After their introduction in ICD devices,9 ATP therapies have been studied with the goal of optimizing them.10 ATP has now largely been proven to be effective and reasonable3,11 and has been given a Class IA recommendation in current consensus documents.6 Nevertheless, inappropriate ATP therapies are still associated with an increased mortality.5,12

Even though ICD manufacturers provide a large variety of programmable parameters, a great lack of knowledge remains for optimal ATP programming with respect to duration, number of pulses, coupling intervals, and number of attempts. All of these parameters have been developed empirically and were consecutively tested in the clinical setting. Thus, optimal duration, optimal number, or optimal coupling intervals of ATP attempts are still uncertain.6 Furthermore, every choice of programming ATP, both tailored or empirical, is always done antecedent to any potential arrhythmia without knowing its characteristics. Thus, the identical ATP therapy may be programmed for a wide range of possible tachycardia cycle lengths. This strategic dilemma is comparable to AV or VV optimization in cardiac resynchronization therapy when trying to optimize a dynamic process. In this case, continuous optimization through an automatic hemodynamic sensor has been recently introduced.13,14 Such individualized automatized optimization seems reasonable to address the variabilities expected in the occurrence of ventricular tachyarrhythmias.

Even though a physician-tailored approach of programming ATP was found to be inferior to empirical programming in the EMPIRIC trial (Comparison of Empiric to Physician-Tailored Programming of Implantable Cardioverter-Defibrillators),11 there are numerous factors known to affect the success of ATP attempts,15 as

See Article by Yee et al

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- sudden cardiac death
- ventricular tachycardia
shown in Table. Automatic ATP optimization would address the need for adaptation to these variabilities in clinical ventricular tachyarrhythmias. This represents a reasonable target for further improvement in ICD programming and therapy success.

In this issue, Yee et al. present the evaluation of a new automated ATP algorithm in implantable defibrillators, which provides individualized ATP therapy not only to the patient but also to each different possible VT the patient may develop. The actual study was a multicenter, prospective, single-cohort study on safety and feasibility in 144 ambulatory patients. The authors are to be congratulated for addressing the topic of ATP optimization again, which is often handled in a cursory fashion. Electrophysiologists will appreciate the new automated ATP algorithm, as it incorporates basic electrophysiological principles into the treatment of ventricular arrhythmias. The outstanding feature of the algorithm is the automatic real-time adaption of the ATP sequence by sequence. The algorithm comes back to traditional electrophysiological knowledge on wavefront propagation. Authors claim 4 key elements of the new algorithm: (1) VT resetting, (2) estimating the refractory period at the pacing site, (3) analysis of the return cycle length, and (4) response to VT acceleration or deceleration. The electrophysiological precision of this algorithm might be especially efficient for slow VT.

However, as with traditional ATP attempts, a new ATP algorithm bears the risk of reduced shock success or VT acceleration that may result in syncope. Unfortunately, even though patients were provided with a syncpe diary, no one returned any data. VT acceleration may contribute to an increase in syncpe or shock rates or both and might thereby have an impact on morbidity. Even though this study was prospective, as the authors acknowledge in the limitations section, this study substantially lacks a control group and, therefore, provides only preliminary data. Because of the uniqueness of the presented algorithm, comparison to historical data seems inapt. Therefore, considering the promising results of the present study, this new algorithm should now be used in a prospective trial involving a control group with traditional ATP programming to prove the new algorithm to be superior to empirical and traditional ATP programming. This study should be designed to identify possible advantages and disadvantages of this algorithm. Furthermore, identification of subgroups of tachycardia characteristics and substrates susceptible to this automated ATP such as (1) slow or fast VT, (2) ischemic or nonischemic VT, and (3) reentry or triggered activity might be possible.

**Table. Factors Affecting Success of ATP Attempts in Terminating Ventricular Tachycardias**

<table>
<thead>
<tr>
<th>Tachycardia mechanism</th>
<th>Reentry, enhanced automaticity, triggered automaticity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia properties</td>
<td>Cycle length, stability, wavelength</td>
</tr>
<tr>
<td>Substrate</td>
<td>Scar, channels, number of entry/exit sites, size of the circuit, location, progressive disease</td>
</tr>
<tr>
<td>Local electrophysiology</td>
<td>Conduction velocity, effective and relative refractory periods</td>
</tr>
<tr>
<td>Stimulation site</td>
<td>Pacing threshold, distance from pacing site to circuit/focus, tissue inhomogeneity, stimulus to QRS duration</td>
</tr>
<tr>
<td>Other factors</td>
<td>Electrolyte dysregulation, catecholamines, vegetative tone, antiarrhythmic drugs, ischemia</td>
</tr>
</tbody>
</table>

ATP indicates antitachycardia pacing.

**DISCLOSURES**

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**FOOTNOTES**

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