CREATIVE CONCEPTS

Electrophysiology at a crossroads

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I recently was asked to give a talk entitled "What is Left for the Diagnostic Electrophysiologist in the Current Ablation/ICD Era?" The hidden message in this question is the assumption by the current electrophysiology community that pretty much most things are treatable by either ablation or an implantable cardioverter-defibrillator (ICD). As someone who for 35 years has been actively "pushing catheters" in hopes of understanding the pathophysiologic basis for arrhythmias and their therapies, it occurred to me that this query is a manifestation of the fact that present-day electrophysiologists are in the midst of a credibility crisis. As such, my initial response to the question would be "to understand electrophysiology."

Training programs today train "ablationists" and "defibrillationists" (a.k.a. implanters), not electrophysiologists. There is no systematic training in the use of the ECG for localizing arrhythmia origin. There no longer is education about the mechanisms of arrhythmias and how they might influence the therapeutic options that are chosen. There has become a total dependence on technology, but no understanding of the limitations of that technology. Most importantly, there has been abrogation of their role as physicians in order to apply wasteful, potentially harmful, cost-ineffective therapy. Our programs no longer teach our electrophysiology fellows how to be "physicians" who can listen to their patient's complaints and formulate a plan of action that is based on scientific evidence *(both pro and con)* and sound judgment. For example, there is widespread, thoughtless, and inappropriate application of the results of clinical trials in the name of "evidence-based medicine." This has led to implantation of ICDs in patients unlikely to benefit to a greater degree than the harm that might be done simply because implantation is easy to do and for fear of lawsuits. The latter is a poor excuse but is a commonly quoted reason for implantation. According to both St. Jude and Guidant, approximately 40% of ICDs now are implanted in patients older than 70 years, even though data on efficacy (i.e., mortality benefit) in such patients are limited. More outrageous is that of these devices (two thirds of which are implanted for primary prevention), one third are single-chamber devices, one third are dual-chamber devices, and one third are biventricular devices. More than 10% of such devices are implanted in patients older than 80 years. Little time is spent discussing the complications of the devices, which include death, heart failure, inappropriate shocks, infection, and proarrhythmia (Table 1). More often than not, electrophysiologists appear to badger patients into getting the devices with statements such as "if you don't have this device you are going to die" or "you shouldn't leave the hospital without a device." I have personally heard these statements too often in the past several years.

The results of clinical trials suggesting a statistical benefit of survival in patients have led to the assumption that these results are applicable to all patients. This is particularly being applied to elderly patients; however, comorbidities in the elderly population may prevent these patients from receiving any benefit from the device. In the Multicenter Automatic Defibrillator Implantation Trial (MADIT)-II, no benefit was noted until 18 months after the device was implanted.[1] The average time from infarct to implant was more than 3.5 years. Meaningful survival benefit was not really seen until at least 3 years after implantation. In elderly patients with congestive heart failure who are potential ICD candidates, comorbidities, including age (*added risk* for each 5 years over age 70), coronary artery disease, dementia, peripheral vascular disease, systolic hypertension, diabetes, and azotemia, have a marked effect on survival. In the presence of four of these risk factors, 18-month mortality is 90%.[2] As such, a significant number of elderly patients with ICDs and based on MADIT-II criteria will have died before accruing any benefit from the device. Of the patients younger than 70 years (the average age of patients in most ICD trials is 60–65 years), the risk of dying or even receiving an appropriate shock is extremely low. The risk is lower when patients are enrolled in private practice than if they are enrolled in hospital.

Dependence on technology has resulted in inappropriate and/or excessive radiofrequency lesions because of the lack of understanding of both the electrophysiology and the limitations of the technology being used. There is a widespread failure to recognize the limitations of depending on technology. Computerized mapping can give misleading activation data ("earliest" site) depending on the fiducial point (reference electrogram) and the boundaries of the acquisition window. This is compounded by the failure of

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Table 1 Complications increasing costs and impairing quality of life

1. Inappropriate number of shocks: Occur in 5%–25% of patients.
2. Proarrhythmia leading to inappropriate shocks for death: The incidence is unknown, but most studies have demonstrated a greater number of appropriate shocks in the device group than in sudden cardiac death or syncope in the control group. This is a twofold to threefold magnitude and is consistent in all trials.
3. Pocket, lead, and vascular complications: Approximately 2% (infected systems) require removal, 0.5%–1% for first implants and up to 3% for replacements.
4. Device malfunction: Is underestimated and probably increasing.
5. Hospitalizations: Worse in congestive heart failure patients by ICD shocks and/or pacing.
6. Death.

the electrophysiologist to map the opposite chamber, which might be the source of an arrhythmia. An example of this scenario is an apparent high right atrial tachycardia with earliest activation that is slightly presystolic in the right atrium, but the atrial tachycardia really arises in the left atrium. Appropriate interpretation of the ECG might have led the electrophysiologist toward a left atrial site and accurate identification of the tachycardia. Computerized mapping also cannot correctly identify the timing of an electrogram (EGM) that is intermittent if acquisition occurs when the EGM is present. Figure 1 shows an example of an intermittent mid-diastolic isolated potential that could have been interpreted as early.

Likewise, use of computerized voltage mapping in sinus rhythm to assess the substrate of ventricular tachycardia (VT) can give misleading results. For example, an interesting EGM with a small isolated late potential often is associated with near-normal far-field signals. The computer would assign a "normal" voltage to that site (implying an unimportant site), which may represent the critical component of a reentrant pathway. Thus, interaction between a knowledgeable electrophysiologist and the data being acquired is needed.

Loss of critical thinking

In the early decades of clinical electrophysiology, all new findings and therapeutic options were subject to considerable discussion and debate. In the current era, there seems to be a lack of willingness to critically determine mechanisms or assess hypotheses. A prime example is the rush to ablate atrial fibrillation (AF) because it is in vogue. There are major limitations to this aggressive attitude. All AFs are not the same and may not be responsive to ablative procedures. The concept of a "curative AF ablation" is an oxymoron until we can turn ourselves into teenagers with new ablation strategies, as aging is a major risk factor for the development of AF, something that ablation does not deal with. There also is a tendency to believe all reported results and to adopt them without skepticism and critical analysis. This is related in part to publication bias toward positive results, preventing knowledge of the true incidence of complications and negative results. This also has led to electrophysiologists doing what is easy in preference to what is proven. An AF ablation drawing perfect circumferential lesions without an understanding of what is accomplished electrophysiologically or the rush to ablate ganglia[3] or fractionated EGMs[4] in the absence of proof of concept in man is remarkable in view of the potential complications of the procedure. Currently, I believe that training for AF ablation in many laboratories involves no mechanistic or electrophysiologic approach but mainly involves designing ways to complete and connect dots around and between the pulmonary veins. The routine use of additional "lines" probably is proarrhythmic (with a 10%–40 % incidence of macroreentrant left atrial flutter) and results in more collateral damage (strokes, phrenic nerve paralysis, coronary occlusion, perforation, and even death). The latest rage is targeting fractionated EGMs with no proof of hypothesis and with the danger of posterior wall lesions. This technique is based on the hypothesis that these sites represent continuous reentry, critical turning points for reentry, or rotors.[4] In fact, there is no proof that such EGMs during AF represent any of the above. They may represent overlapping wavefronts of activation in a three-dimensional structure and/or nonuniform isotropic conduction. These fractionated EGMs come and go, and they are rate related. As such, they cannot be required for maintenance of AF. The additional stated endpoint of a decreased amplitude of a fractionated EGM during AF or normalization of that EGM on return to sinus rhythm is meaningless. If these endpoints were endpoint, then we would be ablating irrelevant right ventricular sites for ventricular fibrillation. The vast majority of fractionated EGMs are around the pulmonary veins in areas frequently ablated during pulmonary vein isolation. Unnecessary ablation of these fractionated EGMs in the posterior wall is

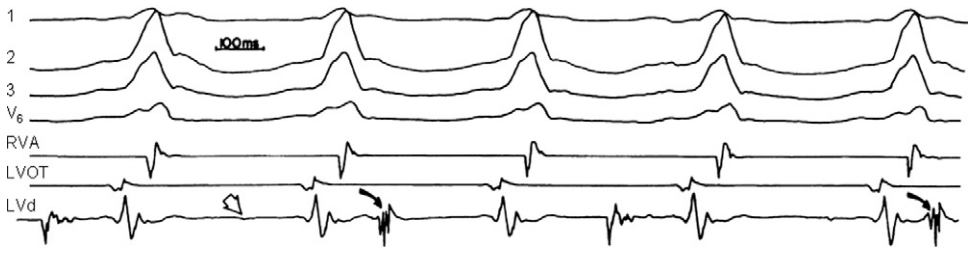

Figure 1 Intermittent middiastolic potential that could be misinterpreted by automated computer mapping (see text for details).