The Nuts and Bolts of ICD Therapy

Tom Kenny
Vice President Clinical Education & Training
St Jude Medical, Austin, Texas
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Preface

When I first started my clinical career, implantable defibrillators were ‘dream machines.’ We could understand in theory how they might work, but the technological obstacles seemed insurmountable. Now, halfway through my career, these devices are not only possible, they are almost commonplace. The technical restrictions that once seemed overwhelming are gone. Today’s ICDs (implantable cardioverter defibrillators) are smaller than some of the pacemakers I once worked with! Despite the fact that a modern ICD is only about one-tenth the size of the original devices pioneered in the 1980s, these new devices last longer and do more.

In the early years of ICD therapy, it was practically a miracle that anyone ever qualified for an ICD. Indications required patients to have survived sudden cardiac death or SCD – not once, but twice! On top of that, patients had to be refractory to drug treatment and yet strong enough to survive an open-chest implantation procedure. Nevertheless, increasing numbers of patients received ICDs.

Today, we know that ICDs are not just a treatment of last resort for people with multiple documented episodes of ventricular fibrillation. As ICDs proved their mortality benefits to patients with known potentially lethal arrhythmias, investigators looked at other arrhythmia-rich populations. As a run of large randomized clinical trials has proven, ICDs have been shown to reduce mortality in primary prevention patients, that is, patients with no documented arrhythmias. Expanding ICD indications have saved lives by extending the proven mortality benefits of devices to more and more people. But this has simultaneously caused an interesting problem for the healthcare community: how can we care for this new influx of patients?

That’s why this book was written. More and more clinicians are going to be confronted with managing ICD patients or at least understanding the role of the ICD in their care. Yet medical schools rarely devote much time to the subject of device-based therapy. Most so-called ‘device experts’ got their status through on-the-job training and the help of colleagues who taught them bits and pieces along the way. There are many fine books on device-based therapy for the heart, but many are written for the experts, not the newcomer.

With expanding ICD indications and hundreds of thousands of potential new ICD patients in the coming years, there are bound to be a lot of ‘newcomers’ to the care of the ICD patient!

Whether you’re a rookie in terms of ICD therapy or whether you’re just an occasional player, this book is a good place to start. Whether you read it cover-to-cover or use it for reference (or both), it was written primarily with you in mind. In my own experience, I learned about defibrillation from on-the-job mentoring from knowledgeable colleagues. Mentors are invaluable, and I’m glad to say it’s a bit of a tradition in clinical practice. You may also find support and training opportunities through device manufacturers. I wrote this book to be one part of the solution for helping the busy clinician manage ICD patients.

Even if you’re a veteran of ICD therapy, it is my hope that you’ll find this book contains tips, pointers, facts, and information to which you’ll want to refer. I have worked in various capacities in the field of device-based therapy since before there even were ICDs … and I am still learning about defibrillation. This book is not an in-depth volume for device experts; it’s the nuts and bolts of ICD therapy for people who actually are involved in the clinical care of these patients.
No book is ever the work of one person. I have to thank my editorial team of Jo Ann LeQuang and Alan Yurkevicius for helping me put this manuscript together and my publisher, Blackwell, for ongoing support and encouragement. But most of all I want to thank my family for their continuing understanding for a busy husband and father who just had to take on one more project. For my wife Diane and our children, Christine, Brian, David, Matthew and Kevin, I want to express my love and affection for such generosity.

Tom Kenny
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Austin, Texas
Sudden cardiac death (SCD) – also known as sudden cardiac arrest (SCA) – has been defined as the unexpected natural death from a cardiac cause within a short time period from the onset of symptoms in a person without any prior condition that would appear fatal. SCD has been described as an ‘electrical accident of the heart,’ in that SCD is a complex condition which requires the patient to have certain pre-existing conditions and then certain triggering events in order to occur. SCD is responsible for about 400,000 deaths a year in the US. Despite our growing knowledge about the mechanisms and markers of this killer disease, SCD remains difficult to treat because the first symptom of SCD is often death.

Many risk factors have been identified for SCD. About 80% of those who suffer SCD have coronary artery disease (CAD), and the incidence of SCD parallels the incidence of CAD (men have CAD and SCD more often than women do, for example). One distinction is that while both CAD and cardiac-related death increase with age, sudden cardiac death decreases with age versus nonsudden cardiac death (NSCD). Older individuals are more likely to experience NSCD than SCD. The peaks of incidence of SCD occur in infants (birth to 6 months) and again between ages 45 and 75 years.1

Several risk factors have been identified for SCD. Some of them are the usual risk factors for any form of heart disease: smoking, inactivity, obesity, advancing age, hypertension, elevated serum cholesterol, and glucose intolerance. Anatomical abnormalities have been associated with SCD. For instance, acute changes in coronary plaque morphology (thrombus or plaque disruption) occur in the majority of cases of SCD cases; about half of all SCD victims have myocardial scars or active coronary lesions.3 For people with advanced heart failure, a nonsustained ventricular arrhythmia was found in one study to be an independent predictor of SCD.4 One report bolstered the popular belief that emotional distress can bring on SCD, in that it was found that the incidence of SCD spiked in Los Angeles right after the Northridge earthquake in 1994.5 Other risk factors include the presence of complex ventricular arrhythmias, a previous myocardial infarction (MI) (particularly post-MI patients with ventricular arrhythmias) and compromised left ventricular systolic function. A low left ventricular ejection fraction is a risk factor that affects people with and without CAD. SCD survivors with a left ventricular ejection fraction < 30% have a 30% risk of dying of SCD in the next 3 years, even if they are not inducible in an electrophysiology study. If these patients are inducible to a ventricular arrhythmia despite drugs or empirical amiodarone, the risk can climb to as high as 50%!

SCD typically involves a malignant arrhythmia. In order for SCD to occur, a triggering event must occur which then has to be sustained by the substrate long enough to provoke the lethal rhythm disorder. The vast majority of SCD cases occur in people with anatomical abnormalities of the myocardium, the coronary arteries, or the cardiac nerves. Typical substrates are anatomical (scars from previous MIs, for example) but electrophysiologists also recognize functional substrates (such as those created by hypokalemia or certain drugs). By far the most common structural abnormalities are caused by CAD and its aftermath, the heart attack or MI. Cardiomyopathy is estimated to be the substrate for about 10% of SCD cases in adults.7 Many people possess the substrates or conditions that make an SCD possible, yet they will never experience the disease. This is because SCD requires a triggering event which not only must occur, it must be sustained on the substrate long enough to develop into a deadly arrhythmia.
Reentry is by far the most common electrophysiologic mechanism involved in SCD. Reentry occurs when a natural electrical impulse from the heart gets ‘trapped’ in a circular electrical pathway in such a way that the impulse keeps re-entering the circuit, faster and faster, provoking a disordered and rapidly accelerating cardiac arrhythmia.

If the human heart were electrically homogenous, reentry and SCD could not occur. The healthy heart has electrical heterogeneity, which means that at any given moment, some cardiac cells are conducting while others are resting. At any point in time, different areas of the healthy heart are at different stages in the electrical cycle. To understand this better, it is useful to review the basics of cellular depolarization, repolarization, and membrane potential.

**Action potential**

All cells in the human body are covered with a semi-permeable membrane that selectively allows some materials to penetrate into the cell while filtering out others. For cardiac cells, the membrane allows charged particles (ions) to flow in and out of the cell at specific times. By regulating the inflow and outflow of ions (electrical charge), cardiac cells are capable of generating and conducting electricity.

Even at rest, a cell in the heart has a certain number of ions within it that give it what scientists would call an ‘electrical potential.’ Electrophysiologists refer to this measurable electrical charge as ‘membrane potential,’ in that it is the electrical potential contained within the cardiac cell’s membrane. The action potential describes five phases (numbered 0 through 4) that show how a cardiac cell goes from resting membrane potential (about –90 millivolts or thousandths of a volt) through depolarization, repolarization, and back to resting membrane potential (see Fig. 1.1).

In its resting state (phase 0), a cardiac cell contains a large quantity of negative ions (anions). Positive ions (cations) outside the cell are blocked from entering by the cell’s membrane but they line up around the cardiac cell, attracted to the negatively charged particles within. It almost appears as if the inside of the heart cell was a negative pole and the immediate exterior of the cardiac cell was the positive pole. From this situation where opposites attract, the term ‘polarization’ is given. The charges polarize negative against positive. The membrane potential phases go from this polarized state (resting membrane potential) to depolarization, then repolarization, and back to the polarized state (resting membrane potential) (see Fig. 1.2).

When an electrical impulse reaches a cardiac cell, the cardiac cell membrane becomes permeable to positively charged sodium ions. Attracted by the negative pole within the cell, sodium ions rush into the cell until the interior of the cell is less negative and the exterior immediately around the cell’s membrane is less positive. This shift decreases the cell’s resting membrane potential to the point where fast sodium channels open in the cell membrane. Fast sodium channels are just like they sound; they allow a very rapid influx of positively charged sodium ions into the cell. As a result, the interior of the cell becomes positive and the exterior around the cell becomes negative. This phase – where polarization is reversed – is called depolarization.
The main physiological effect of cardiac depolarization is that the heart cells contract. That’s why electrophysiologists frequently refer to the squeezing or pumping action of the heart muscle as ‘depolarization,’ since that best describes what is going on in the heart’s cells. At the cellular level, cardiac cells are becoming positively charged on the interior, negatively charged on the exterior – and this results in a heart beat.

The very process of contraction begins the next phase of the membrane potential, in that the positively charged sodium ions start to leave the inside of the cardiac cell when the cell contracts. Electrophysiologists call this process of getting back to the original resting membrane potential ‘repolarization,’ and it is characterized physiologically by the heart returning to a relaxed or resting state. At the cellular level, the positive ions flow out while negative ions flow back in using sodium as well as calcium and potassium channels. It is impossible for the cardiac cell to depolarize again until it has completed all three phases involved in repolarization; during phases 1–3, the cardiac cell is refractory.

The final phase of the action potential (phase 4) might best be viewed as a brief moment of rest. At the cellular level, there is very little activity going on, with only a few ions crossing the cell membrane either way (see Fig. 1.3).

The morphology of the action potential varies depending on the type of cardiac cell involved. Phase 0 shows how quickly the cell depolarizes, while phases

![Fig. 1.2 Polarization of a cardiac cell. In phase 0 of the action potential, the cardiac cell contains a majority of negative ions within the cell with positive ions clustered around the immediate exterior. As ion channels open, positively charged ions rush into the cell (changing the cell’s polarization or ‘depolarizing’ it). Positive ions flow back out using sodium as well as potassium and calcium channels, ‘repolarizing’ the cell to its original status.](image)

![Fig. 1.3 Action potential. The action potential is an electrical way of describing the cellular changes that occur during depolarization and repolarization. At phase 0, the cardiac cell has a certain potential electrical energy. This ramps up quickly during phase 1 or depolarization when the cell rapidly changes polarity. Repolarization occurs more gradually in phase 2 and 3. There is a brief vulnerable period in phase 4 as the heart rests before resuming its membrane potential (phase 0) and starting the cycle over again.](image)
1–3 show how long the refractory period is. The action potentials from some main locations in the heart show that cardiac cells are specialized in terms of how fast they depolarize and how long they remain refractory (not able to depolarize) (see Fig. 1.4).

**Automaticity**

Automaticity is the heart’s ability to spontaneously generate electricity. The specialized cells in the heart’s sinoatrial (SA) node possess this remarkable property. The SA node is sometimes called the heart’s ‘natural pacemaker’ for its ability to keep the healthy heart beating properly. Other myocardial cells possess automaticity and may spontaneously deliver an electrical output. In fact, many regions of the heart, including the atrioventricular (AV) node and even ventricular tissue, possess enough automaticity to ‘fire’ an electrical output. However, the heart’s conduction system requires the electrical output to travel a specific path through the heart. At any given moment, the electrical pathway can only accommodate one output, and the heart works on a first-come, first-served principle. (In the cardiac conduction system, the fastest impulse wins.) The first output that gets on track is the one that travels. Other cells might generate an electrical output based on the principle of automaticity, but the pathway they will travel is refractory (not subject to depolarization because it is in phase 1, 2, or 3 of the action potential) and thus, the electricity will have no effect on the cells.

Automaticity and triggered automaticity are two of the three main causes of tachycardia, although altogether they account for only about 10% of all tachycardias. Automaticity involves abnormal acceleration of phase 4 of the action potential, causing the heart to launch into another depolarization too quickly. Its cause is increased activity across the heart’s membrane in phase 4, usually involving a mechanism known as the sodium–potassium pump. As such, automaticity and triggered automaticity tachycardias have metabolic or cellular causes, and since they are not caused electrically, they do not respond to defibrillation. In fact, tachycardias caused by automaticity cannot be reproduced in the electrophysiology lab. The main causes of automatic tachycardias are thought to be ischemia (diseased heart tissue caused by CAD), electrolyte imbalances, acid/base imbalances, drug toxicity, and myopathy (muscle disorder).

It is often possible to observe the locations and types of cardiac disturbances by viewing variations in the action potential. Triggered automaticity looks a lot like reentry tachycardia on the action potential. It occurs when something triggers an automaticity-type tachycardia. A typical trigger might be a bradycardic pause or a catecholamine imbalance. This trigger accelerates phase 4 of the action potential,
causing the heart to launch the next depolarization too quickly, resulting in an accelerating heart rate. A common example of triggered automaticity is the torsades-de-pointes type of tachycardia. Torsades-de-pointes (twisting points) takes its name from the French and describes the twisting or turning action the ECG seems to show (see Fig. 1.5).

**Reentry**

By far the most common mechanism for tachycardias anywhere in the heart is reentry, responsible for about 90% of all tachycardias. Although common, reentry is a complex mechanism which requires several specific conditions to be met before it can occur.

Reentry tachycardia first requires a bypass tract. The conduction pathway through the heart (from SA node over the atria to the AV node then out across the ventricles) is ideally a series of relatively straightforward unidirectional pathways from origin to termination. An impulse entering the pathway travels down through the cells, creating a cascade effect of depolarization and repolarization. Electrical impulses that enter the pathway after the initial impulse may still travel, but they encounter only refractory cells and cause no depolarization. A bypass tract occurs when the conduction pathway forms a branch that splits but then reconnects. As a result impulses traveling down the pathway may go down one side or the other, but will eventually regroup at the end (see Fig. 1.6).

For reentry to occur, this bypass tract must have a fast path and a slow path, that is, the two arms of the bypass tract must be electrically heterogeneous, that

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Fig. 1.5 Torsades-de-pointes. This is one of the best known types of ventricular tachyarrhythmia caused by triggered automaticity. Its name means ‘twisting points’, taken from the apparent twisting motion of the waveforms on an ECG. Torsades-de-pointes occurs when a trigger falls in the vulnerable phase 4 of the action potential, causing the heart to start its next depolarization too quickly (and thus accelerating the heart rate).

Fig. 1.6 Bypass tract. A bypass tract consists of an electrical conduction pathway which splits at one point and then reconnects. This bypass tract allows electrical energy to flow down either the right side or left side of the tract. If both sides of the tract conducted electricity at exactly the same speed, this would not be a problem. However, if one side conducts electricity faster than the other side, it means that the cardiac tissue on one side of the bypass tract is going to be refractory at the same time as the other side is capable of conducting electricity. This means that electrical energy can get ‘trapped’ in the loop and circulate around and around the tract rather than flowing downward and out.
is, they must conduct electricity at different speeds. This, in turn, means that the two pathways will have different refractory periods. Impulses will travel more quickly through one path than the other, and one pathway can be refractory (not subject to depolarization) at the same time as the other pathway is depolarizing.

Finally, a reentry tachycardia requires some sort of triggering event, most commonly a premature contraction. This trigger enters the bypass tract and sets off a chain of events, which results in an endless loop of accelerating depolarizations, causing a very rapid heart rate (see Fig. 1.7).

Types of tachycardia

Supraventricular tachycardias (SVTs)
Supraventricular tachycardias (SVTs) originate above the ventricles and allow impulses to travel downward via the His-Purkinje network. SVTs can be caused by either automaticity or reentry mechanisms, and they are rarely life-threatening. When caused by automaticity, SVTs tend to be chaotic and multifocal, meaning they originate from many points in the upper areas of the heart. Usually caused by some sort of metabolic disorder (including digitalis toxicity, pulmonary disease, or acute alcohol poisoning), automatic SVT does not respond to pacing or cardioversion but can sometimes be reversed by treating the underlying cause.

Reentrant SVT is the more common form of SVT and may be congenital or acquired, and is known to occur even in patients without heart disease or acute illness. Intra-atrial reentry tachycardias (atrial flutter, atrial fibrillation) are caused when the reentry circuit occurs within the atria. SA or AV nodal reentrant tachycardias are sometimes also described as micro-reentry tachycardias because the whole bypass tract resides entirely in the SA or AV node – a very small (‘micro’) area.

AV nodal reentrant tachycardia (AVNRT)
AV nodal reentrant tachycardia (AVNRT) accounts for about 60% of all atrial, narrow-complex tachycardia seen in clinical practice, excluding atrial fibrillation. This micro-reentry tachycardia does not actually involve the atria or ventricles directly, since the whole bypass tract is contained in the AV node. On the other hand, the micro-reentry circuit in the

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**Fig. 1.7** Reentry tachycardia. Here is how a reentry tachycardia can get started. (A) A premature contraction or trigger enters the bypass tract. Since it is not properly timed (it arrives too early), it finds that tissue on one leg of the path is refractory. (B) The conduction on the fast path arrives at a juncture where it can either go down or back up. If it were not for the premature contraction, it would only be able to go down because the slow path of the bypass tract would all be refractory. However, the premature contraction has caused only part of the slow path to be refractory (shaded). This means that the electrical impulses in the fast path may travel back up and around the tract. (C) Because it is a loop, the bypass tract allows one impulse to keep traveling around and around the circuit. It accelerates as it does and causes rapid contractions of cells. New impulses can enter, but since the principle of cardiac conduction is ‘fastest impulse wins,’ the new impulses cannot take control.