1 CHAPTER 1
Sudden cardiac death

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.[1] 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.[2] 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%![6]

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 semipermeable 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

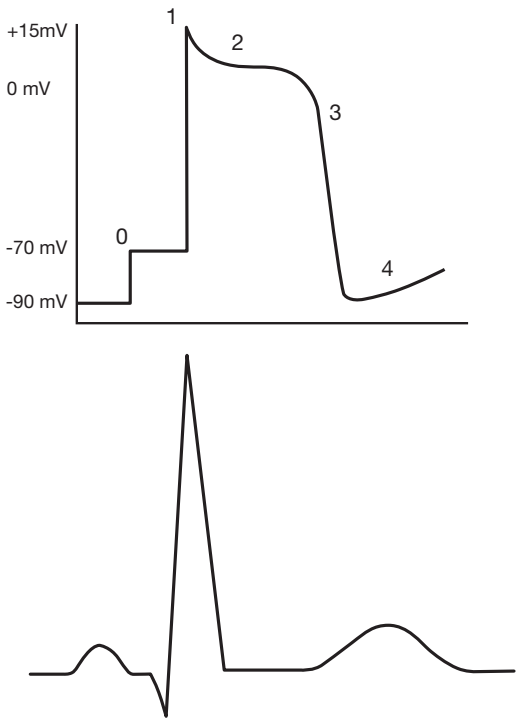


Fig. 1.1 Membrane potential. The membrane potential of a cardiac cycle involves five distinct phases (0 through 4) with phase 1 corresponding roughly to the peak of the R-wave.

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.

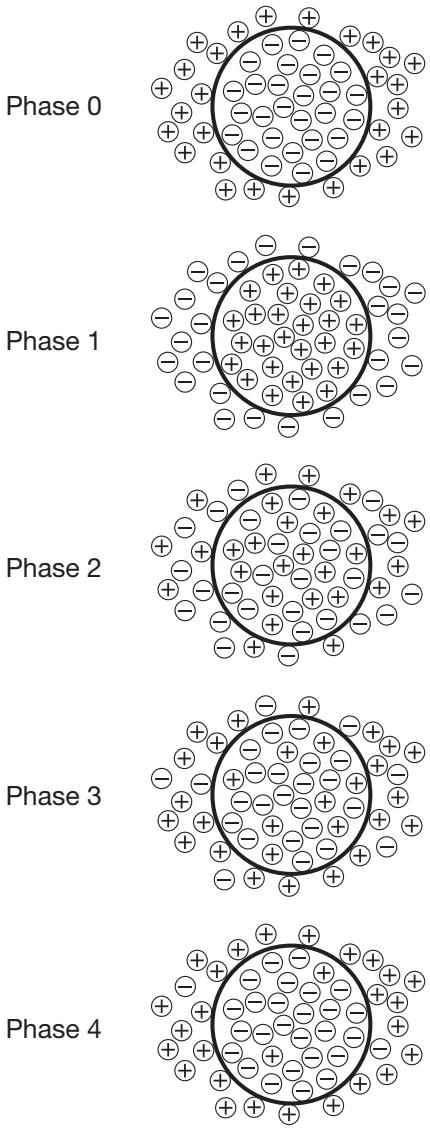


Fig. 1.2 Polarization of a cardiac cell. In phase 0 of the action potential, the cardiac cell contains a majority of negative ions within the cell with positive ions clustered around the immediate exterior. As ion channels open, positively charged ions rush into the cell (changing the cell's polarization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

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

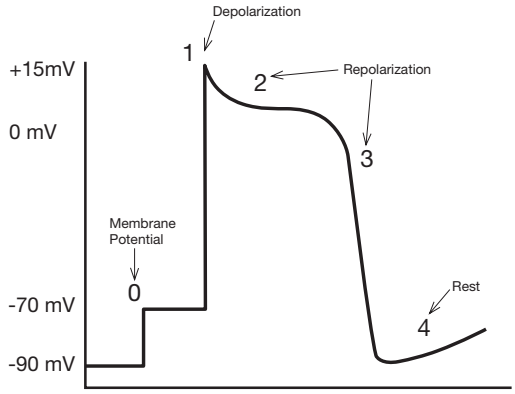


Fig. 1.3 Action potential. The action potential is an electrical way of describing the cellular changes that occur during depolarization and repolarization. At phase 0, the cardiac cell has a certain potential electrical energy. This ramps up quickly during phase 1 or depolarization when the cell rapidly 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.

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.

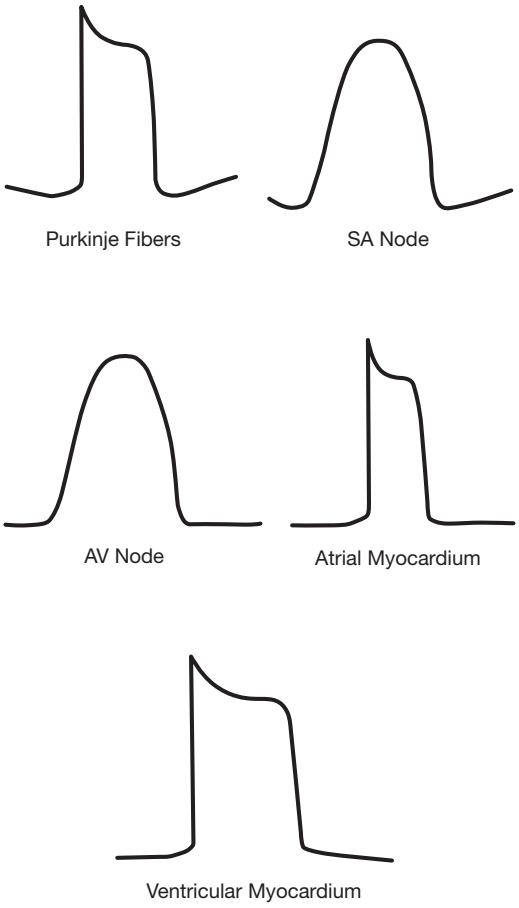


Fig. 1.4 Action potentials of various portions of the heart. The action potential varies with the electrical activity of specific types of cardiac cells. Note that an atrial myocardial action potential is much narrower (briefer in duration) than a ventricular myocardial action potential. The rounded curves of the AV and SA nodal action potentials suggest that the transition from depolarization (phase 1) to repolarization (phases 2 and 3) is not as marked and abrupt as the same change in the Purkinje fibers. These illustrations show the different electrical properties of various regions of the cardiac tissue, which cause them to conduct electricity differently.

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,

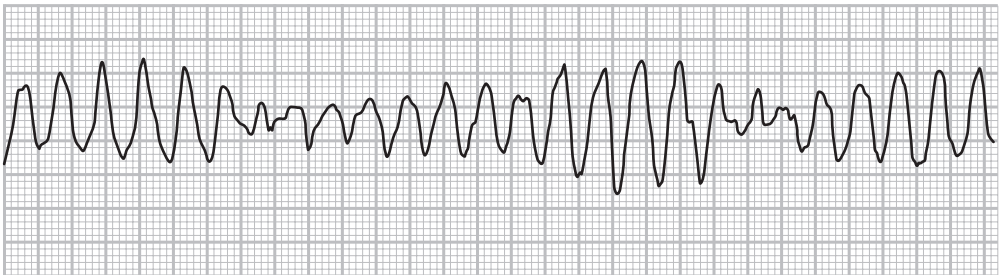


Fig. 1.5 Torsades-de-pointes. This is one of the best known types of ventricular tachyarrhythmia caused by triggered automaticity. Its name means 'twisting points', taken from the apparent twisting motion of the waveforms on an ECG. Torsades-de-pointes occurs when a trigger falls in the vulnerable phase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

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

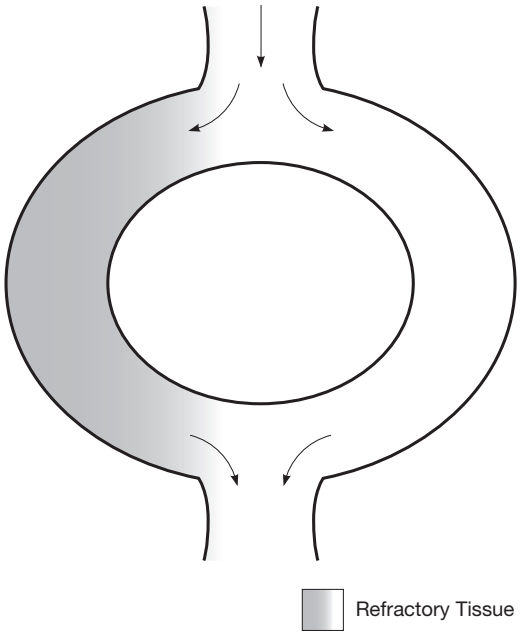


Fig. 1.6 Bypass tract. A bypass tract consists of an electrical conduction pathway which splits at one point and then reconnects. This bypass tract allows electrical energy to flow down either the right side or left side of the tract. If both sides of the tract conducted electricity at exactly the same 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

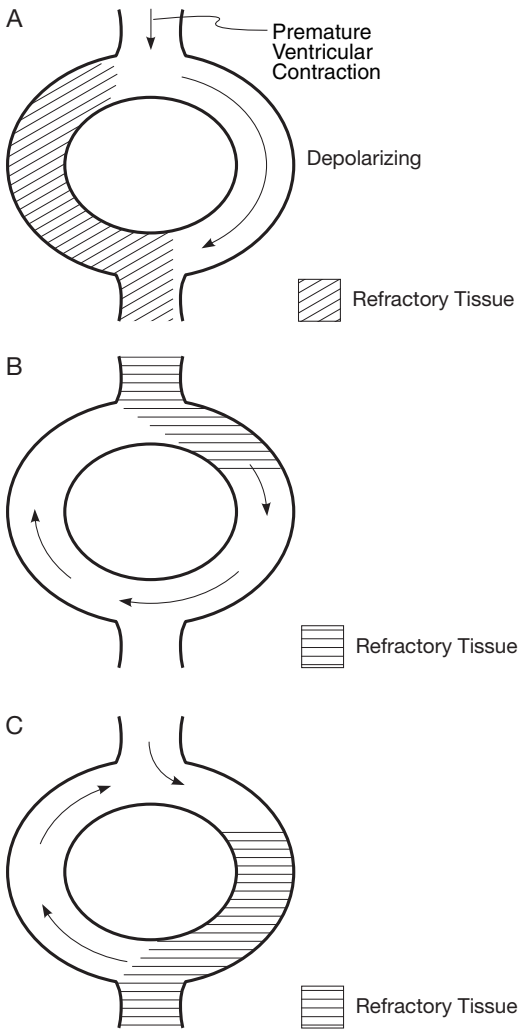


Fig. 1.7 Reentry tachycardia. Here is how a reentry tachycardia can get started. (A) A premature contraction or trigger enters the bypass tract. Since it is not properly timed (it arrives too early), it finds that tissue on one leg of the path is refractory. (B) The conduction on the fast path arrives 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.

AV node activates atria and ventricles, so the heart's upper and lower chambers both participate. One characteristic of AVNRT is that premature beats in one chamber do not affect the timing of beats in the other chamber.

Wolff-Parkinson-White (WPW) syndrome

In contrast to a micro-reentry circuit, sometimes a reentry tachycardia relies on a very large 'macro'-reentry circuit. An example of this is Wolff-Parkinson-White (WPW) syndrome where the bypass tract is large enough to connect the atrium directly to the ventricle. In this sort of tachycardia, the premature beats of one chamber will obviously advance the activation of the other chamber, since the bypass tract links atrium to ventricle.

Atrial fibrillation (AF)

The most common arrhythmia in the world is a type of intra-atrial SVT known as atrial fibrillation (AF). At one time, AF was described as chaotic atrial activity from multiple focal points, but modern theory holds that AF may actually be a more organized rhythm disorder than originally suspected. Many AF rhythm disorders originate in or near the pulmonary veins and not directly in the atria. AF is often inducible in the electrophysiology lab, but even there it can be hard to terminate. There are three broad classifications of AF, but even experts sometimes disagree as to where one category ends and the next begins. The three types of AF are paroxysmal (which terminates on its own), persistent (which requires medication, cardioversion, or both to terminate) and permanent (which cannot be stopped). While AF is not necessarily a lethal rhythm disorder in the same way that ventricular fibrillation is deadly, AF is associated with a greatly increased risk of stroke and other major health risks.

SVTs with rapid ventricular response

Patients with different types of SVT are typically treated with drugs to prevent the onset of the rhythm disorder or to slow down the ventricular response. Some SVTs can be treated with ablation, in which the reentry circuit is surgically destroyed. Ablation can be done by radiofrequency (RF), such as AV nodal ablation or by open-chest surgery (the Maze procedure). Newer, minimally invasive, catheter-based ablation procedures are being introduced. Ablation still poses considerable clinical challenges in terms of mapping (finding the bypass tract) and navigation (getting to it), but when properly done in the appropriate patient, ablation is curative.

SVTs can also be treated with external cardioversion and defibrillation. Implantable atrial cardioverter-defibrillators are plausible devices that were in development in the 1990s, but they failed to gain widespread acceptance.[8] For the most part, SVTs are not lethal and some are even asymptomatic. An implantable device that dispenses an uncomfortable therapy to treat these SVTs was perceived as painful and intrusive by most patients.

Monomorphic and polymorphic VT

Ventricular tachycardia (VT) describes any too-fast heart rhythm that originates in the ventricles. Generally defined as occurring at rates between 100 and 300 beats a minute, VT can be monomorphic (originating from one focus in the ventricle) or polymorphic (originating from multiple focal points in the ventricle).

Monomorphic and polymorphic VT are fairly easy to detect on a surface ECG. Monomorphic VT consists of rapid but fairly regularly shaped QRS complexes. In monomorphic VT, all of the complexes should look similar in terms of complex morphology. On the other hand, polymorphic VT is characterized by differently shaped QRS complexes (see Figs 1.8 and 1.9).

VT can also be described as sustained (over 30 seconds in duration) and nonsustained (less than 30 seconds in duration). Nonsustained VT (NSVT) is a short run of VT which spontaneously terminates and is usually asymptomatic. Sustained VT lasts for a longer period of time, but may also spontaneously terminate.

Ventricular fibrillation (VF)

By far, the most lethal arrhythmia in the world is ventricular fibrillation (VF), which usually occurs at rates between 200 and 300 beats a minute. Unlike VT, in which clearly discernible (if somewhat erratically shaped) QRS complexes can be seen, VF is a wildly disorganized rhythm with no clear, individual QRS complexes at all. When VF occurs, the heart is no longer really pumping; it's quivering. Cardiac output drops to zero and the patient quickly approaches hemodynamic collapse. VF causes

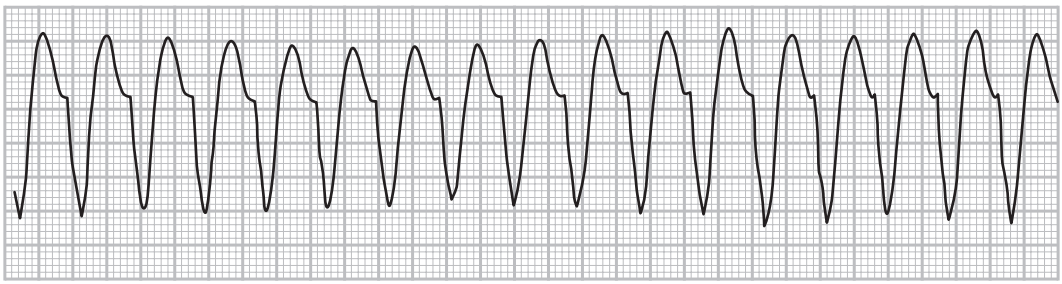


Fig. 1.8 Monomorphic VT. In a monomorphic VT, the tachyarrhythmia originates from a single place in the ventricles. This results in a series of QRS complexes which look similar to each other.

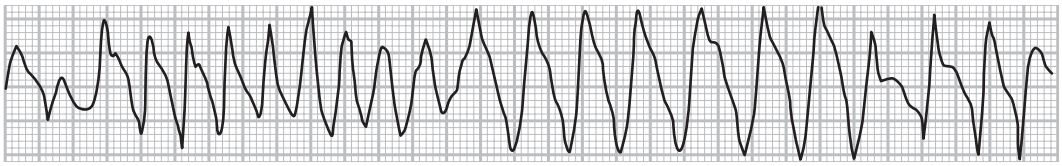


Fig. 1.9 Polymorphic VT. In a polymorphic VT, the tachyarrhythmia originates from more than one region in the ventricle. This causes the QRS complexes on the QRS to have different shapes, depending on their points of origin.

asystole (flat line) and leads to death, sometimes in as few as 4 minutes from time of onset. Fortunately, VF can be effectively reversed with timely defibrillation (see Fig. 1.10).

About 10% of all cases of VT have automaticity as the mechanism. An automatic VT is evidenced by an abnormal acceleration of phase 4 of the membrane potential. Ischemia and metabolic causes (electrolyte imbalance, acid/base imbalance) are the main causes of automatic VT, which can also be induced by drug toxicity. If the underlying cause of the automaticity can be addressed, automatic VT is completely reversible.

Long QT syndrome (LQTS)

Probably the best example of a VT involving triggered automaticity as a mechanism is long QT syndrome (LQTS). Although relatively rare, LQTS has received some attention in the cardiology community, particularly for an inherited form of the disease which strikes families and often causes premature deaths of children and adolescents. While there is a divergence of opinion on the appropriate role of ICDs (implantable cardioverter-defibrillators) in this population, there is evidence that ICD implantation even in young people with congenital LQTS is appropriate.[9] While high-risk patients with congenital LQTS may benefit from ICD therapy, its use is controversial and some investigators advocate concomitant beta-blocker therapy.[10]

LQTS may also be acquired. On the membrane potential curve, LQTS affects the repolarization phase of the slope of phases 1–3. This prolongs the refractory period and allows a window of opportunity for intruding abnormal rhythms.

Torsades-de-pointes

Torsades-de-pointes is an example of a triggered-automaticity polymorphic VT and is often treated with magnesium infusion (even if serum magne-

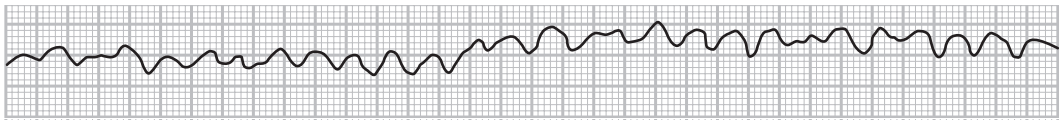


Fig. 1.10 Ventricular fibrillation. The most lethal of all arrhythmias, ventricular fibrillation lacks discernible QRS complexes. The tracing typically shows small-amplitude, bizarrely shaped signals that indicate a disorganized, quivering type of rhythm.

sium levels are normal), isoproterenol infusion or electrical rhythm management. Torsades-de-pointes is not a reentry tachycardia and is typically treated with pharmacological therapy. This type of arrhythmia is much more common in females than males, but the pathogenesis of torsades-de-pointes is not completely understood.[11]

Brugada syndrome

Brugada syndrome was first described just 10 years ago and has been the subject of considerable interest and study. A hereditary disorder, Brugada syndrome affects the early repolarization phase of the action potential (phases 1–2) and triggers a VT or even VF by automaticity. The disease exists all over the world, but is particularly prevalent in Asia. While more research needs to be done to better understand why a genetic mutation causes triggered automaticity in the ventricles, Brugada syndrome may respond to ICD therapy (see Fig. 1.11).[12]

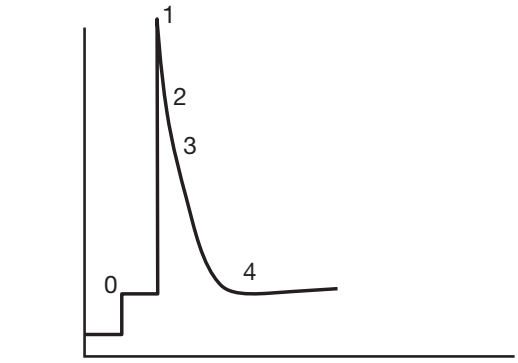
Reentry VT/VF

By far the most common mechanism for VT and VF is reentry.[13] Reentrant VT frequently starts around an area of scar tissue on the heart, such as might occur as a result of an MI or heart disease. This scar area forms what cardiologists sometimes call the 'substrate' or area of compromised tissue that can support a reentrant VT or VF.

For patients who have had an MI, it is easy to understand how substrates form. During an MI, the heart muscle is deprived of valuable oxygen-rich blood. This lack of oxygen results in the necrosis or death of certain areas of cardiac tissue. The damage caused by an MI depends on where this tissue necrosis occurs and how much tissue is involved. Heart attack survivors have portions of diseased or scar tissue in the heart muscle. It is around this scar tissue that the conduction defect can occur. The dead tissue no longer conducts, but the margin of viable tissue around the scar often acts as aberrant conduction pathways. This is such a prevalent method for reentry tachycardia that all heart attack survivors should be counseled about their potential susceptibility to rhythm disorders.

In theory, reentry VT and VF can be treated by ablation, that is, the surgical removal of part of the bypass tract. The practical realities of VT ablation are much different: the bypass tracts are difficult to locate and map and often involve a large area. Even if the bypass tract could be properly identified and surgically excised, the ablation procedure creates a new scar … and this can restart the cycle!

Medications to affect the action potential and slow the rate of conduction (thus reducing the heart rate and stopping or at least making the VT less severe) or drugs that change the refractory period (to inhibit reentry) have long been a mainstay in cardiology. However, most cardiologists know that cardiac drugs are toxic at incorrect dosages, require careful monitoring even at correct dosages, and sometimes have pro-arrhythmic effects. Implantable defibrillators, first introduced in the 1980s and pioneered in the next decade, offer real promise in treating VT and VF. The idea behind defibrillation



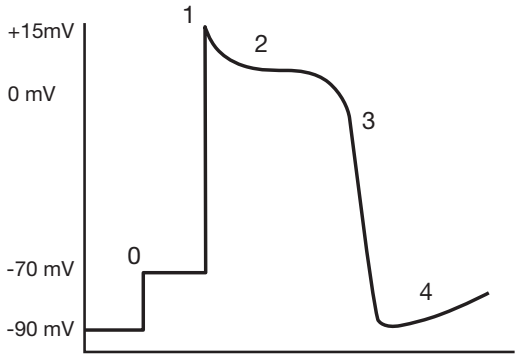


Fig. 1.11 Action potential for Brugada syndrome. The action potential for Brugada syndrome shows a very steep, almost immediate voltage decline after depolarization when contrasted to a normal action potential. Reentrant tachycardia occurs because of this shortened action potential.

is to treat the electrical cause of reentry VT/VF with a generic 'dose' of electricity. The implantable device was pioneered to make sure it was constantly on standby, ready to administer electrical energy whenever a potentially dangerous reentry ventricular arrhythmia occurred.

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The nuts and bolts of sudden cardiac death

- Sudden cardiac death (SCD), also known as sudden cardiac arrest (SCA), is one of the leading killers of Americans and remains a difficult disease to treat, because its first symptom is often death.
- There are many known risk factors for SCD including – but not limited to – coronary artery disease, smoking, inactivity, obesity, increasing age, hypertension, high cholesterol, and glucose intolerance.[14]
- Many malignant arrhythmias require a substrate or alternate conduction pathway in the heart and a triggering event to initiate a dangerous rhythm disorder.
- Reentry tachycardias occur when an electrical impulse gets 'trapped' in a bypass tract (substrate) and goes faster and faster, causing the heart to try to beat more and more rapidly.
- Automatic tachycardias are not caused by an electrical problem but occur when the heart tries to depolarize too quickly after repolarization, usually as a result of an acid/base imbalance, electrolytic imbalance, metabolic condition, or some form of drug or alcohol poisoning.
- The action potential describes what happens in the heart at the cellular level that causes it to beat. The action potential consists of five phases (0 through 4) in which 0 involves depolarization, phases 1, 2, and 3 describe repolarization and the short phase 4 describes a brief moment of rest. Many cardiac conditions and the mechanisms of action for many cardiac drugs involve changes to one or more phases of the heart's action potential.
- Membrane potential is the electrical potential of a cardiac cell at rest.
- Automaticity refers to the ability of many types of cardiac cells to spontaneously generate electricity. Although the sinoatrial (SA) node is the heart's 'natural pacemaker,' many cells, including some in the AV node and others in the ventricle possess automaticity as well.

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- Torsades-de-pointes is a form of automatic tachycardia and does not respond to electrical therapy.
- Most arrhythmias are named for where they originate (a supraventricular tachycardia is any too-fast rhythm that originates above the ventricles; it may affect atria and ventricles).
- For reentry to occur, there must be a bypass tract with a fast path and a slow path. This means that one portion of the tract will be refractory (that is, unable to depolarize) at the same moment that the other pathway is depolarizing.
- AV nodal reentrant tachycardia is a form of micro-reentry tachycardia because the reentry circuit is very small and entirely contained in the AV node. Wolff-Parkinson-White syndrome (WPW) is a form of macro-reentry tachycardia in that the reentry circuit directly links atrium to ventricle and covers a very large area of the heart.
- Two of the most common atrial arrhythmias are AV nodal reentrant tachycardia and atrial fibrillation.
- Atrial fibrillation (AF) is roughly classified into paroxysmal (sudden onset, terminates spontaneously), persistent (longer episodes that require intervention, typically drugs or cardioversion, to terminate) and permanent (refractory to treatment). AF is a progressive disorder.
- In general ventricular tachycardia (VT) is a too-fast heart rhythm that originates in the ventricles with a rate between 100 and 300 beats a minute. Ventricular fibrillation (VF), which is far more dangerous, is a too-fast, wildly disorganized heart rhythm that originates in the ventricles with a rate between 200 and 300 beats a minute or even higher. In terms purely of rate, it can be difficult to say if a ventricular tachyarrhythmia of 250 beats a minute is VT or VF without looking at an ECG. On an ECG, VF is a chaotic, disorganized rhythm and it is impossible to see a clearly differentiated QRS complex. VT, on the other hand, can be very fast but does show discernible QRS complexes.
- A monomorphic VT is one that originates from one source in the ventricle and all QRS complexes have a similar shape on the ECG. A polymorphic VT originates from more than one source in the ventricle and has different QRS morphologies present on the ECG.
- Long QT syndrome (LQTS) is a relatively rare disorder in which triggered automaticity causes ventricular fibrillation. It can be acquired or hereditary.
- Brugada syndrome is a hereditary disorder which provokes an automatic VT or even VF. It is more common in people from Asia and South America, and it may respond to defibrillation.