Clinical Spectrum of Ventricular Tachycardia

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A great deal of new information has emerged concerning the mechanisms, electrocardiographic (ECG) recognition, electrophysiological behavior, and management of a variety of ventricular tachyarrhythmias. It has now become possible to identify several distinct clinical entities of ventricular tachycardia (VT) that have different electrophysiological bases, prognoses, and treatment strategies. Therefore, it seems timely to review the current state of knowledge regarding the more commonly encountered forms of VT. This communication is not intended to present the extensive body of knowledge available regarding various types of VT but is meant to be a comprehensive description covering salient features of the more commonly encountered varieties.

The term VT in this communication refers to symptomatic ventricular arrhythmias that have organized ventricular activity. Complex ectopy, asymptomatic VT, or ventricular fibrillation, therefore, are excluded from this category. To facilitate clinical recognition of the various types of VT presented here, a separation into monomorphic and polymorphic appearance (Figure 1) is being made at the beginning. The term monomorphic implies a uniform beat-to-beat QRS morphology, whereas in polymorphic VT there is a constant change in QRS configuration albeit the beat-to-beat change may be subtle in some situations. It should be further pointed out that at times the polymorphic nature of VT may not be apparent from a single ECG lead and that multiple leads may be required to reveal the true pattern.

Monomorphic Ventricular Tachycardia

Several distinct types of VT are included in this category. There seems to be a relation between the type of VT and the underlying anatomic substrate. In the discussion below, it is not implied that a given type of VT cannot occur in other settings. However, the frequency with which certain forms of VT occur in specific clinical settings makes it important to keep the substrate in mind to facilitate recognition.

Sustained Ventricular Tachycardia in Association With Chronic Coronary Artery Disease

This variety of monomorphic VT constitutes the most commonly encountered form in the adult population. The typical substrate is within the vicinity of a healed myocardial infarction with or without associated ventricular aneurysm. Evidence of prior myocardial infarction can often be documented from surface ECG, various imaging techniques, and often directly at the time of surgery. Although the VT origin is within or near the damaged myocardium, the precise location is difficult to determine. The cure of such a VT with surgical techniques targeted at the prior myocardial infarction or aneurysm attests to the close anatomic proximity of VT origin to the damaged myocardium.

From clinical studies as well as animal models of VT in association with myocardial infarction, it can be reasonably assumed that the underlying mechanism is reentry. This is based on several observations including 1) induction and termination of VT with programmed ventricular stimulation, 2) ability to entrain VT with properly timed atrial or ventricular stimulation techniques, 3) responses to antiarrhythmic drugs (such as an unchanged overall QRS morphology after aggravation to incessant forms by class I agents), and 4) direct myocardial mapping techniques. Various patterns of reentrant excitations, including simple loop and figure-eight configurations have been delineated. Such reentrant excitation is undoubtedly facilitated by areas of slow conduction due to damaged myocardium, by alteration of passive properties, such as anisotropy, and by islands and streaks of fibrosis providing anatomic obstacles.

The role of acute myocardial ischemia as an essential part of the substrate has not been convincingly demonstrated. Although this type of reentrant monomorphic VT has been primarily studied in patients with chronic coronary artery disease, other cardiac pathologies resulting in critical amount and orientation of myocardial fibrosis may also be associated with similar arrhythmias.

Clinical presentations vary and depend on a variety of factors, including global left ventricular function, rate of VT, and the posture at the time of VT onset. In patients with significant coronary artery disease or left ventricular dysfunction, rapid degeneration to
Ventricular fibrillation may occur. Cardiac arrest, syncope, and presyncope are common manifestations. A 12-lead ECG is seldom available with such a clinical presentation. However, when the VT is well tolerated, the patients present in an ambulatory setting. This type of VT is the most common cause of wide QRS tachycardia in patients first seen in the outpatient environments, where it is frequently mistaken as supraventricular tachycardia with aberrant conduction. Because of good hemodynamic tolerance in this subgroup, a 12-lead ECG is frequently recorded. The VT can be induced and replicated in the electrophysiology laboratory in a vast majority of cases. The induced or spontaneous VT of this type produces a surface ECG appearance of a wide QRS tachycardia that can be readily distinguished from supraventricular tachycardia with aberrant conduction by established ECG criteria. The QRS morphology may either show a right bundle branch block (RBBB) or a left bundle branch block (LBBB) pattern. Both morphologies may be seen in the same patients when the interventricular septum is involved. Due to its myocardial origin, the QRS appearance is different from a typical LBBB or RBBB pattern, which forms one of the bases for distinction from aberrant conduction (Figure 3). From the myocardium, the excitation wave front penetrates the His-Purkinje system retrogradely. Therefore, on a His bundle electrogram, the His bundle deflection either follows the onset of QRS (i.e., the value is negative for the interval from initial His bundle deflection to the onset of ventricular activity [H-V interval], as seen in Figure 3) or, more commonly, is obscured within the corresponding local ventricular electrogram.

Literature is replete with information dealing with the various approaches used in the management of VT in association with chronic coronary artery disease;
hence, it is not detailed here. In fact, the bulk of the published data dealing with monomorphic VT has focused on this form of VT. The prognosis and consequent selection of therapeutic options is dependent on many factors: 1) hemodynamic tolerance of VT, 2) overall left ventricular function, and 3) extent of coronary artery disease. In patients presenting with cardiac arrest, syncope, or presyncope, recurrences can be life threatening, and more definitive therapeutic approaches, such as curative VT surgery and implantation of a cardioverter defibrillator, may be the preferred options. Drug-resistant VT cases would also be considered for the nonpharmacological therapy mentioned above. Hemodynamically well-tolerated VT can be managed in a variety of ways, including pharmacological therapy. It is important to point out, however, that a beneficial response to a pharmacological agent should not be automatically assumed even in this population. Ideally, efficacy of a given therapeutic intervention should be assessed with some objective end point, such as programmed electrical stimulation. This is particularly important when the initial presentation is hemodynamic collapse.

The foregoing description is that of a sustained VT, which is defined as one lasting for more than 30 seconds or requiring immediate intervention. Although the sustained variety of monomorphic VT has been more extensively studied, such tachycardias may also manifest themselves in nonsustained forms. However, with a nonsustained episode, the underlying mechanisms, the response to therapeutic interventions, and even the prognosis may be different and often more difficult to characterize.

Generally, VT seen in patients with coronary artery disease seems to arise from damaged myocardial substrate. However, when significant cardiac dilatation coexists, another type of VT, which involves the two bundle branches and the His bundle in the reentry circuit, may also occur. This variety of VT is seen more frequently in patients with idiopathic dilated cardiomyopathy (DCM). The underlying mechanism is referred to as bundle branch reentry (BBR) and is covered in greater detail below.

Sustained Ventricular Tachycardia in Association With Dilated Cardiomyopathy

As pointed out above, patients with DCM seem to have at least two anatomic substrates that can cause sustained reentrant VT. In most patients with DCM in association with coronary artery disease and in approximately 60% of patients with idiopathic DCM, the sustained VT is indistinguishable from that seen
in patients with prior myocardial infarction. It is also quite likely that in such instances the underlying mechanism is related to intramyocardial reentry circuits due to diseased myocardium or fibrosis. However, in our experience, more than one third of the patients with idiopathic DCM have sustained BBR as the cause of monomorphic VT. Even though BBR is also seen in DCM in association with coronary artery disease, VT in the latter population is far more frequently caused by the non-BBR mechanism described earlier.

The prerequisite for BBR seems to be prolonged conduction in the His-Purkinje system, such that, invariably, there is intraventricular conduction delay either in the form of a bundle branch block pattern or, more frequently, a nonspecific QRS widening that is detectable on baseline ECG (Figure 4). Intracardiac studies demonstrate a prolonged H-V interval. Since transeptal conduction is an essential part of the circuit, it is possible that some abnormality of intramural conduction may also be playing a critical role. The exact role of cardiac dilatation in facilitation of BBR is not known at the present time.

Since ventricular activation occurs by way of bundle branches, the resultant QRS morphology has a typical LBBB or RBBB appearance (Figures 4 and 5). Even the His bundle electrogram may be suggestive of supraventricular tachycardia (Figure 5). The His bundle potential usually precedes the ventricular activation with the H-V interval equal to or exceeding the H-V interval of baseline sinus or other supraventricular rhythms. As a rule, atrioventricular dissociation or ventriculoatrial block are present, which suggests that the origin of arrhythmia is within or below the level of the atrioventricular node. Although DCM, particularly of the idiopathic variety, is the most common substrate, BBR can occur in DCM from any etiology. Conduction delays in the His-Purkinje system, such as those associated with valvular heart disease, may also provide a suitable substrate in some cases.

These tachycardias are rapid and usually produce serious hemodynamic sequelae, including presyncope, syncope, and cardiac arrest, and therefore, 12-lead ECG is seldom available. In the laboratory the VT can be initiated with programmed ventricular stimulation. In the series reported from our laboratory, the induction was facilitated by a sudden cycle length change before the extrastimulus. When the right ventricle is the site of stimulation, induced morphology shows an LBBB pattern since induction of retrograde RBBB is easier to set up with the right ventricle as the pacing site, as in the circuit depicted in Figure 6. This in part is the reason why most published examples of sustained BBR show an LBBB pattern. Initiation of BBR with an RBBB configuration may require pacing from the left ventricle (Figures 5 and 6).

Once the diagnosis is established that the BBR is the only form of VT, a specific therapeutic option is available for this type of VT. Catheter ablation of the right bundle branch provides an effective treatment and can be readily achieved without production of atrioventricular block. Pharmacological agents with negative inotropic affects may be less desirable. The coexistence of sustained BBR in patients with DCM may carry a poor prognosis. Further deterioration of left ventricular function is likely, and serious consideration to cardiac transplantation should be given in appropriate candidates. However, right bundle branch ablation should successfully ameliorate the immediate arrhythmic problem.
FIGURE 5. Recordings showing induction of sustained bundle branch reentry. VT, ventricular tachycardia; CL, cycle length; I, 2, and VI, surface electrocardiographic leads; HB(P) and HB(D), recordings from proximal and distal His bundles, respectively; RV, right ventricle; V and V1-V5, onset of ventricular activity; H and H2-H4, deflection of His bundle; Sb, first heart sound; S2, second heart sound; LB, left bundle potential. Panel A depicts induction of sustained bundle branch reentry with a left bundle branch block pattern with RV stimulation. Note H before each QRS with the H-V interval equal to the H-V interval of baseline rhythms (panel B). VT with a right bundle branch block pattern could only be induced with pacing from left ventricle (panel C). LB precedes the QRS during both VT and baseline rhythm (panel D). Reproduced with permission from Tchou et al.41 (p252)

Ventricular Tachycardia in Association With No Overt Structural Heart Disease

This category includes a variety of VT seen in individuals with no obvious organic heart disease.43–57 It is important to realize that in many of the published cases the presence of myocardial pathology has not been excluded with myocardial biopsy. Even if the latter were normal, a localized abnormality may exist. Nonetheless, there are some common features of VT in this population, which include 1) no obvious structural heart disease, 2) usually a single monomorphic configuration of QRS complex, 3) less well-defined underlying electrophysiological mechanisms, and 4) generally a good prognosis in terms of arrhythmic mortality. A more detailed description of these features is provided below.

The discussion that follows is organized according to QRS morphology rather than a specific underlying anatomic-electrophysiological substrate, since the latter is rather unclear at the present time. This approach should aid clinical recognition of these forms.

Sustained ventricular tachycardia with QRS configuration of right bundle branch block and left axis deviation. This type of VT has been primarily seen in a young population (Figure 7). Literally all of the cases reported have been younger than 50 years of age. Although there are several case reports of VT showing such a QRS morphology in an apparently healthy population, larger series were reported by German et al43 and Ohe et al.45 In the latter report, two of 16 patients exhibited RBBB and right axis
deviation, but the electrophysiological behavior of VT was otherwise similar to that of the remaining patients.

The clinical presentation of VT has been relatively benign. The vast majority of patients experienced only palpitations; a minority presented with near syncope or syncope. The fact that VT can be easily induced by programmed stimulation suggests, but does not prove, that reentry is the underlying mechanism. The involvement of the ventricular myocardium or peripheral Purkinje system is unclear, but the latter has been often implicated in VT.

A unique feature of this VT has been its uniform responsiveness to intravenous and oral verapamil administration. This has led to the suggestion that calcium-dependent slow conduction or triggered activity may be involved. In the report by Ohe et al., the VT was also readily terminated by ajmaline. The responses to drugs have not provided any definitive clues to the underlying electrophysiological mechanism.

Biopsy data are not available in numbers sufficient to draw any meaningful conclusions. However, cardiac arrest or sudden death has seldom been reported in this population, possibly, in some cases, because of alteration of the natural history by pharmacological or nonpharmacological intervention. From published reports it seems that aggressive antiarrhythmic therapy is often not required and that oral verapamil continues to control VT in cases requiring treatment.

**Sustained ventricular tachycardia with QRS configuration of left bundle branch block.** An LBBB morphology of VT probably represents a more heterogeneous group as compared with the RBBB pattern discussed above. Typically the QRS axis is right (Figure 8) or normal.

The catheter mapping techniques suggest an origin near the right ventricular outflow tract (i.e., at or above the infundibulum on interventricular septum rather than the right ventricular free wall). The clinical presentation is often palpitation, but presyncope or syncope is not infrequent. The VT can be provoked in the electrophysiology laboratory in the majority of cases by programmed electrical stimulation, suggesting reentry as a mechanism. However, a distinct group of patients exists with a similar QRS appearance, in which the VT cannot be provoked with programmed stimulation alone. In these
patients, exercise, isoproterenol infusion, or both are more effective in initiation of VT.\textsuperscript{48,49} The effectiveness of isoproterenol infusion has led to the suggestion that catecholamine-related automaticity may be operative, particularly when programmed electrical stimulation fails to initiate the VT in the same patients. Furthermore, many patients with VT showing an LBBB and a right axis pattern respond to both \(\beta\)-blockers and verapamil; these findings are compatible with the belief that catecholamine-triggered afterdepolarization is the mechanism of VT.\textsuperscript{49,52,53} This scenario is further complicated by a recent report in a small group (four patients) in whom VT was initiated and terminated by programmed electrical stimulation and in whom VT was also terminated by intravenous administration of adenosine.\textsuperscript{53} However, VT could also be terminated or prevented by valsalva/carotid sinus massage and propranolol or verapamil or both. All of the above are known to decrease slow inward calcium current either directly by modulating calcium channels or indirectly by production of cyclic AMP. Therefore, cyclic AMP-mediated triggered activity (delayed afterdepolarizations) was suggested as the possible underlying mechanism. Suffice it to say that, in patients with an apparently normal heart, VT presenting with LBBB may have a variety of potential underlying mechanisms. It is to be realized that none of the criteria used to establish a given mechanism in the clinical laboratory is sufficiently specific to distinguish triggered activity from reentry.\textsuperscript{21,54} Although VT presenting with LBBB in an overtly normal population seems to be an identifiable clinical entity, the exact anatomic-electrophysiological basis remains elusive and requires further work.

Myocardial biopsy data have shown no detectable abnormality in the majority of such patients.\textsuperscript{55} In the remaining patients, increased amount of fibrosis is the main histopathologic feature.\textsuperscript{55} The response to therapy has been unpredictable, and a wide variety of drugs may have to be explored before an effective regimen is found. Patients with exercise-provoked VT respond well to \(\beta\)-adrenergic blockade. Many of these cases also respond to verapamil, but treatment with class I antiarrhythmic agents or amiodarone is often necessary. In difficult cases or those prone to hemodynamically compromising VT, nonpharmacological therapeutic options may have to be entertained. Even though the overall prognosis is quite favorable, sudden death has occasionally been reported.\textsuperscript{56,58}

Most patients who have VT in association with a structurally normal heart are young and have not been followed for long. The natural history of VT in these settings and the outcome in terms of longevity is not certain at the present time. These patients should nonetheless be followed very closely since it is conceivable that underlying myocardial pathology may become more apparent at a later date. In the event that ventricular function deteriorates over a period of time, the risk of future sudden arrhythmic death could rise substantially.

Before ending this section on monomorphic VT with an LBBB configuration, two other entities should be mentioned briefly. The first is the so-called arrhythmogenic right ventricular dysplasia.\textsuperscript{59} Although the heart is not structurally normal in arrhythmogenic right ventricular dysplasia, VT is typically the first manifestation in what appears to be an otherwise healthy population. The cardiac structural abnormalities, as a rule, are defined later during workup. Significant abnormalities, such as dilatation or fibrosis, can exist in the right ventricle without obvious alteration in left ventricular function and cardiac output. VT typically shows an LBBB morphology, but left axis deviation is common.\textsuperscript{59} It would seem prudent to entertain this possibility in patients presenting with VT who exhibit an LBBB similar to that in apparently healthy subjects. The diagnosis of arrhythmogenic right ventricular dysplasia can be suspected from T wave inversion in right precordial leads and cardiac enlargement on chest x-ray film in some cases and can be strengthened with right ventricular imaging techniques.\textsuperscript{59} VT is inducible in the laboratory with electrical stimulation, and reentry circuits can be mapped. Class I antiarrhythmic agents or amiodarone are generally effective, and in drug refractory cases, right ventriculotomy and catheter ablative techniques have been successful.\textsuperscript{59–61} The clinical course is variable, but overall prognosis is favorable. A relatively small number of cases have been followed; hence, a true long-term prognosis in these patients must await further information.

The second entity is referred to as repetitive monomorphic VT, which is characterized by short runs showing an LBBB pattern and right axis compatible with an origin that is in or near the right ventricular outflow.\textsuperscript{62} It is unclear whether this entity has a different morphological-mechanistic basis than the sustained counterpart. From the available literature it seems that these patients with no overt heart disease cannot be distinguished from those with sustained VT from right ventricular outflow in terms of VT origin and response to exercise or isoproterenol. Drug responses to agents, such as \(\beta\)-blockers, verapamil, and class I agents, are also similar, as is the prognosis. The main difference, of course, is the presentation of repetitive well-tolerated nonsustained VT versus more symptomatic sustained VT. However, some patients with repetitive monomorphic VT do have a history of presyncope and syncope, raising the possibility of sustained episodes at other times. When these patients are asymptomatic or exhibit minimally symptomatic nonsustained VT, no treatment may be necessary. Nonetheless, a close follow-up is desirable, in the event that cardiac dilatation, worsening symptoms, or both are present.

**Polymorphic Ventricular Tachycardia**

Clinical recognition of the polymorphic nature of VT is important since it often represents a distinct
substrate requiring different management strategy. Even though the underlying electrophysiological mechanisms and substrates for polymorphic VT may be quite diverse, there are at least three clinical scenarios that deserve more detailed discussion.

**Torsade de Pointes**

Torsade de pointes\(^{63-80}\) literally implies a twisting of points (QRS around the baseline) and fairly accurately describes the morphological appearance of this tachycardia (Figure 1B).\(^{64}\) It is rapid and polymorphic with a beat-to-beat change in QRS morphology (Figure 1B). This type of tachycardia is seen primarily in association with prolongation of the QT interval (Figures 1A and 9). The QT (QT\(_c\)) prolongation could be subtle and dynamic at times only appreciable before the onset of arrhythmias or after a pause (Figure 1). Both congenital and acquired forms of QT prolongation have been described.\(^{67,76}\)

In the acquired form (the more frequent form seen in the adult population), the QT prolongation seems to be mostly in association with the use of agents known to prolong myocardial repolarization, such as class Ia antiarrhythmic drugs (quinidine, procainamide, disopyramide), amiodarone and in the presence of hypokalemia, or hypomagnesemia.\(^{67}\) Hypokalemia is often produced by the use of diuretics routinely employed in the management of patients with cardiovascular disease.

The precise mechanism for torsade de pointes is not known. However, recent data strongly suggest that triggered activity due to early afterdepolarizations may be playing an important role in the acquired form.\(^{70-75}\) In a model of quinidine-induced prolongation of action potential duration, triggered activity was facilitated by slower rates and lower levels of extracellular potassium and magnesium.\(^{75}\) All three factors seem to make an independent contribution.\(^{75}\) These findings are in line with clinical observations in which, in the acquired form, QT interval prolongation is primarily seen in the clinical settings of hypokalemia, often in combination with antiarrhythmic drugs. The other predisposing factor for initiation of torsades de pointes is the presence of bradycardia. It is quite possible that torsade de pointes induced by bradycardia would aggravate the symptoms of bradycardia and even be the main cause of bradycardia-associated fatigue in some cases.

The management of torsade de pointes in the adult population is relatively straightforward once the proper diagnosis is made. The first step is to withdraw the offending agent, such as an antiarrhythmic drug or any other drug known to prolong the QT interval. Immediate administration of intravenous magnesium and replacement of potassium may be effective. An additional approach often taken is to accelerate the heart rates since potassium usually facilitates or aggravates torsade de pointes. This can be accomplished with either overdrive pacing (Figure 9) or administration of atropine or catecholamines. The beneficial effect of \(\beta\)-adrenergic stimulation may not only relate to acceleration of heart rate but could
include direct cellular actions. It is important to point out that torsade de pointes could lead to ventricular fibrillation and, hence, could be potentially fatal. The exact magnitude of such fatalities is uncertain, but considering the frequent use of class I antiarrhythmic agents and potassium-depleting diuretics, it could be considerably higher than currently realized.

Torsade de pointes represents the predominant form of VT in association with congenital prolonged QT syndrome with or without deafness. In this situation, sympathetic stimulation with physical exertion or excitement seems to be the main trigger initiating episodes of torsade de pointes. Therefore, increased sympathetic activity, which often controls the acquired form of torsade de pointes, tends to provoke the congenital forms. This makes it difficult to extrapolate much of the recently acquired knowledge regarding the mechanisms of acquired prolonged QT syndrome related torsade de pointes to the congenital counterpart. Traditionally, though, torsade de pointes cannot be induced with conventional pacing protocols in the laboratory, which argues against reentry as the mechanism in this population.

Torsade de pointes, in association with congenital prolonged QT syndrome, best responds to β-blockers. Even though β-blockers may not shorten the QT interval, they are the mainstay in the treatment and have been known to reduce mortality from an expected 78% (in symptomatic patients without treatment) to 6%. Agents known to shorten QT interval, such as digitalis, lidocaine, calcium, and potassium, have not proven useful in the management of these cases. The role of calcium channel entry blockers is not certain at the present time. It goes without saying that the pharmacological agents known to prolong QT interval are considered contraindicated in congenital prolonged QT interval syndrome. In resistant cases, cavothoracic sympathectomy has also been used with success. Since syncope and sudden cardiac death frequently occur in this population, implantation of an automatic implantable cardioverter defibrillator should be seriously considered in appropriate cases.

**Polymorphic Ventricular Tachycardia in the Absence of QT Prolongation**

To avoid confusion, the term torsade de pointes in this communication is only used when polymorphic VT occurs in the setting of prolonged QT interval, whether congenital or acquired. All other nonuniform VT QRS morphologies are covered as polymorphic regardless of how closely they simulate torsade de pointes in terms of QRS appearance. This separation is critical because of significantly different therapeutic approaches.

Most, but not all, patients with polymorphic VT in the absence of QT prolongation have coronary artery disease. At least two subgroups of patients may exist. In patients with stable coronary artery disease with prior myocardial damage but no evidence of acute ischemia, polymorphic VT may occur as an isolated phenomenon (Figure 10). Frequently, these patients also have bouts of monomorphic VT, the type covered earlier under chronic coronary artery disease. Polymorphic VT in association with stable chronic coronary artery disease is not an extensively studied phenomenon by virtue of either its short duration or its rapid and unstable nature frequently leading to sustained monomorphic VT or ventricular fibrillation. This may have led to a perception that polymorphic VT is not a common arrhythmia, which may not necessarily be correct. The exact frequency of spontaneous polymorphic VT in the presence of chronic coronary artery disease is unknown, but it could be common. This is particularly true in victims of sudden cardiac death, in whom the initiating event could be either a monomorphic or polymorphic VT. In fact, polymorphic VT is frequently induced in the laboratory in survivors of out-of-hospital ventricular fibrillation. Polymorphic VT can also be induced in the laboratory in patients without spontaneous polymorphic or any VT. Such a response is often dismissed as nonspecific. It is quite conceivable that in some cases with life-threatening ventricular arrhythmias manifesting themselves as presyncope, syncope, or cardiac arrest, polymorphic VT is the only or main arrhythmic...

![Figure 10](http://circ.ahajournals.org/)

**FIGURE 10.** Recordings showing polymorphic ventricular tachycardia with normal QT interval. Top panel shows spontaneous episode of polymorphic ventricular tachycardia in association with normal QT interval. Sustained episode (middle panel) leads to ventricular fibrillation (bottom panel). This patient had coronary artery disease but continued to have such episodes after myocardial revascularization with patent grafts as well as β-blockade class I agents readily controlled this arrhythmia.
event. However, it should be pointed out that, even if spontaneous polymorphic VT may be a rather frequent and prognostically important arrhythmia, the use of its induction in the laboratory as a reliable guide for serial pharmacological testing remains unproven.

The mechanisms of spontaneous polymorphic VT in these settings may be several, but the inducible forms are compatible with reentry. Since symptomatic polymorphic VT could be life-threatening arrhythmia, aggressive therapy is warranted. Treatment options are similar to the sustained monomorphic variety in association with chronic coronary artery disease, except that polymorphic VT is not an ideal arrhythmia for map-guided surgical therapy. Implantation of a cardioverter defibrillator may be a more desirable approach in these cases.

Polymorphic VT is a rather important manifestation of acute myocardial ischemia (Figure 11). When polymorphic VT is accompanied or preceded by angina or diagnostic ECG changes, ischemic etiology is often suspected. However, clinical manifestation for acute ischemia in the form of other symptoms, such as angina, may or may not coexist. Similarly ECG evidence of acute ischemia may not be apparent. Nonetheless, these patients do have evidence of a critical degree of coronary artery stenosis. Typically, these arrhythmias cannot be replicated in the laboratory with programmed electrical stimulation. However, due to obvious underlying active ischemia substrates, rarely have such patients been studied in the electrophysiology laboratory, and thus, the underlying mechanisms remain uncertain. As would be expected, this type of polymorphic VT responds well to anti-ischemic therapy, including β-blockers and myocardial revascularization.

Polymorphic VT can occur in settings other than coronary artery disease, such as primary myocardial pathology, particularly in association with hypertrophic states. However, rather limited information currently exists for appropriate characterization.

A proper understanding of the relative clinical importance of various forms of VT is difficult without some perspective of their relative frequencies. These rather crucial data are not available in the published literature. In fact, in some ways the published data may not accurately reflect the real preponderance of various forms of VT. Some reasons for this are explained below.

Only the survivors of a given VT episode are available for further scrutiny, and they constitute a minority compared with nonsurvivors. This is not a trivial issue if one realizes that a substantial number of cardiovascular mortality is sudden and that most victims of sudden cardiac death have VT as the initial arrhythmia, whenever ECG documentation is available. Even among survivors resuscitated by rescue squad, the nature of arrhythmia at the beginning of the episode is seldom available. Therefore, in the overwhelming majority of these patients, the type of initial VT is not documented, and the relative frequency of monomorphic versus polymorphic VT as the initial cause remains unknown. From the limited data that are available, monomorphic and polymorphic VT seem equally common. Therefore, induced forms of VT are heavily relied on for extrapolation regarding the nature of clinical VT in a high risk population; conclusions drawn from such information concerning the relative frequency of monomorphic versus polymorphic VT is speculative at best. The exception to this is the small number of patients in whom the induced VT represents replication of prior ECG-documented VT. In essence, therefore,
the bulk of the published information deals with VT in survivors or with electrically induced forms of VT. Much of the information regarding VT comes from arrhythmia centers, and proper interpretation of such data requires understanding of population selection. For example, referral centers tend to receive patients with recurrent arrhythmia problems, which are more difficult to manage. Many of these patients have hemodynamically stable sustained VT, which in all likelihood carries a less ominous prognosis compared with the more malignant forms (see below). Therefore, the population with stable VT is probably overrepresented in the literature. The more malignant forms of VT (monomorphic or polymorphic), in which VT degenerates into ventricular fibrillation, are seen less frequently. This is particularly so in communities in which out-of-hospital resuscitation is nonexistent. In such circumstances, only the natural survivors who may have a better prognosis regarding mortality from arrhythmic events are encountered. This can produce a significant difference concerning the incidence of a given variety of VT as reported by various centers. On the other extreme, forms of VT that can be easily mistaken for supraventricular tachycardia with aberrant conduction, by virtue of benign symptoms or easy control with drugs traditionally used in supraventricular tachycardia, may be seen less often at arrhythmia centers. Therefore, VT in association with a structurally normal heart, particularly VT controlled with β-blockers and verapamil, may be underreported in the literature.

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