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Ventricular arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features

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INTRODUCTION

Sudden cardiac death in the setting of an acute myocardial infarction (MI) is most frequently the result of a ventricular tachyarrhythmia. The appearance of a sustained ventricular tachyarrhythmia following an MI, such as ventricular tachycardia (VT; monomorphic or polymorphic) or ventricular fibrillation (VF), in the early period post-MI may be the harbinger of ongoing myocardial ischemia, the development of proarrhythmic myocardial scar tissue, elevated sympathetic tone or increase in circulating catecholamines, or an electrolyte disturbance such as hypokalemia. In-hospital mortality approaches 20 percent or more in patients who develop VT or VF following an MI. As such, rapid identification and treatment of these arrhythmias can be life-saving. Although all patients with a prior MI have an elevated risk of malignant arrhythmias, the magnitude of risk varies from patient to patient, with reduced left ventricular ejection fraction being the most prominent risk stratifier.

This topic will focus on the incidence, mechanisms, and clinical features of ventricular arrhythmias during and after acute MI. Treatment for established ventricular arrhythmias during acute MI and in the post-MI patient, using defibrillation with or without antiarrhythmic medications, is discussed separately. (See "Advanced cardiac life support (ACLS) in adults" and "Sustained monomorphic ventricular tachycardia in patients with structural heart disease: Treatment and prognosis" and "Secondary prevention of sudden cardiac death in heart failure

and cardiomyopathy" and "Ventricular arrhythmias during acute myocardial infarction: Prevention and treatment".)

INCIDENCE

While many studies have evaluated the incidence of ventricular arrhythmias in the peri-infarct period, comparison of these studies is difficult due to differences in populations (percutaneous intervention therapy versus fibrinolytic therapy versus no therapy), type of infarct (ST segment elevation MI [STEMI] versus non-ST segment elevation MI [NSTEMI] versus both), and arrhythmia reported (ventricular tachycardia [VT] versus ventricular fibrillation [VF] versus both). The clinical significance of different types of ventricular ectopy following MI also varies markedly.

Ventricular arrhythmias, ranging from isolated premature ventricular complexes/contractions (PVCs; also referred to a premature ventricular beats, premature ventricular depolarizations, ventricular premature complexes, or ventricular premature beats) to VF, are common in the immediate post-infarction period (ie, within the first 48 hours). Observations in the pre-fibrinolytic era found the following range of incidence [1-3]:

- PVCs – 10 to 93 percent
- VT – 3 to 39 percent
- VF – 4 to 20 percent

Life-threatening ventricular arrhythmias, VT and VF, are infrequent but serious complications of an acute STEMI. The largest experience on the incidence of VT and VF during an acute STEMI comes from the GUSTO-1 trial of 40,895 patients who were treated with thrombolytic therapy [4]. The overall incidence of sustained VT or VF was 10.3 percent: 3.5 percent developed VT, 4.1 percent VF, and 2.7 percent both VT and VF. Approximately 80 to 85 percent of these arrhythmias occurred in the first 48 hours.

Rates of VT and VF have declined with time. In a study of 11,825 patients with acute MI between 1986 and 2011, there was a decrease in the incidence of both VT (from 14.3 to 10.5 percent) and VF (from 8.2 to 1.7 percent) over the 25 years reviewed [3].

Sustained ventricular arrhythmias are less common in patients with an acute NSTEMI or unstable angina compared to patients with STEMI, as illustrated in a pooled analysis of four major trials of over 25,000 such patients [5]. The overall incidence of VT or VF was 2.1 percent, lower than the 10.3 percent incidence in GUSTO-1 in STEMI [6]. VT occurred in 0.8 percent, VF in 1 percent, and VT and VF in 0.3 percent. The median time to arrhythmia was 78 hours.

Early versus late arrhythmias — The definition of "early" versus "late" ventricular arrhythmias can vary among cardiologists and electrophysiologists and is also changing with time. Most now consider "late" arrhythmias to be those that occur beyond 24 to 48 hours post-MI onset. Late VT is a predictor of a worse prognosis [4]. Among post-MI patients who survived for 30 days, subsequent one-year mortality was significantly higher among those who had late VT (24.7 percent) compared with those without sustained ventricular arrhythmia (2.7 percent) [4].

Pre-revascularization (fibrinolytic/PCI) era versus PCI era — In the era of early percutaneous coronary intervention (PCI), VT (especially non-sustained VT) remains fairly common. In the MERLIN-TIMI 36 study of 6355 patients with non-ST elevation acute coronary syndromes who underwent seven days of continuous electrocardiographic (ECG) monitoring following their presentation to the hospital to assess for VT and ischemia, 25.3 percent were found to have VT (20 percent VT without ischemia, 5.3 percent VT with ischemia) [7]. Compared with patients with neither VT nor ischemia on continuous ECG monitoring, patients with VT without concurrent ischemia had a significantly increased risk of both cardiovascular death and sudden cardiac death (SCD; adjusted hazard ratio [HR] 2.2 and 2.3, respectively). Patients with VT and ischemia had an even higher risk of both cardiovascular death and SCD (adjusted HR 5.4 and 6.5, respectively).

ST-elevation MI — Data regarding the incidence of ventricular arrhythmias at the time of acute STEMI come from studies of patients treated with either fibrinolysis or primary PCI. Although the incidence of ventricular arrhythmias is probably lower with contemporary therapies [4,5,8-11], these data are also probably underestimating the true incidence of arrhythmias because patients with prehospital SCD may not have been included in studies of fibrinolysis or primary PCI. The risk factors for and the prognosis of early VT or VF in patients with STEMI are discussed separately. (See 'Monomorphic ventricular tachycardia' below.)

Fibrinolytic therapy — Among patients with acute MI in the fibrinolytic era, the incidence of VF has ranged from 3.7 to 6.7 percent in large studies [4,5,10-13]. The largest experience in patients (40,895) with acute STEMI treated with fibrinolytic therapy comes from the GUSTO-1 trial [4]. The overall incidence of sustained VT or VF was 10.2 percent (3.5 percent developed VT, 4.1 percent VF, and 2.7 percent both VT and VF). Approximately 80 to 85 percent of these arrhythmias occurred in the first 48 hours. Two limitations of fibrinolytic trials are the exclusion of patients who died of SCD in the prehospital arena, which affects the total denominator of patients, as well as the fact that fibrinolysis and reperfusion may lead to reperfusion arrhythmias, falsely increasing the incidence of MI-associated arrhythmias.

Primary PCI — Reported rates of primary VF among patients with STEMI treated with primary PCI range from 6 to 9 percent in different studies.

- Among 5745 STEMI patients with planned PCI enrolled in the APEX AMI trial, VT or VF occurred in 329 (5.7 percent), with most events (282, or 86 percent) occurring within the first 48 hours [14].

- In a study of 13,253 patients with STEMI transported by ambulance in one city between 2006 and 2014, 749 patients (5.6 percent) had witnessed pre-hospital cardiac arrest [15].

- In a study from the Spanish Codi AMI network which included 10.965 patients with STEMI treated with primary PCI between 2010 and 2014, 949 patients (8.7 percent) experienced primary VF [16].

Non-ST elevation MI — The best data on the incidence of sustained ventricular arrhythmias in patients with acute NSTEMI or unstable angina come from a pooled analysis of four major trials of over 25,000 patients with a non-ST elevation acute coronary syndrome (NSTEMI or unstable angina) [5]. The overall incidence of sustained VT or VF was 2.1 percent, which is lower than the 10.2 percent STEMI incidence in GUSTO-1 [4]. VT occurred in 0.8 percent, VF in 1 percent, and VT and VF in 0.3 percent. The median time to arrhythmia was 78 hours.

MECHANISMS OF ARRHYTHMOGENESIS

Ventricular arrhythmias in the setting of acute MI result from an interplay among three basic components:

- The damaged myocardium, which produces a substrate capable of developing reentrant circuits or associated with enhanced automaticity
- Arrhythmia triggers, including variations in cycle length and heart rate
- Modulating factors, such as electrolyte imbalance (eg, hypokalemia), dysfunction of the autonomic nervous system (eg, increased sympathetic activity), continued ischemia, and impaired left ventricular (LV) function

The proper milieu for the development and maintenance of ventricular arrhythmias is ultimately based upon the rapid and profound effects that acute myocardial ischemia has on the electrophysiologic characteristics of the myocyte. Changes in the resting membrane potential and in the inward and outward ionic fluxes during the action potential lead to alterations in conduction, refractoriness, and automaticity of cardiac muscle cells, all of which contribute to the occurrence of ventricular arrhythmias [17]. (See "Cardiac excitability, mechanisms of arrhythmia, and action of antiarrhythmic drugs".)

Sustained ventricular tachycardia (VT) and ventricular fibrillation (VF) in the setting of MI result from the complex interaction of multiple factors, including:

- Myocardial ischemia (with resulting local electrolyte abnormalities).
- Necrosis.
- Reperfusion.
- Healing.
- Scar formation.
- Autonomic changes (especially activation of the sympathetic nervous system and elevated levels of catecholamines).
- Damage to the His-Purkinje fibers and pathways within the ventricular myocardium. Changes are heterogenous and result in heterogenous changes in conduction velocity and repolarization of these pathways.

These events produce the mechanisms that initiate arrhythmias and the substrate for arrhythmia perpetuation. Arrhythmia pathogenesis varies at different stages in this process. For ventricular arrhythmias occurring more than 48 to 72 hours after an acute MI, scar formation is of primary importance.

Acute phase (first 30 minutes) arrhythmias — A distinction must be made between the acute phase of myocardial ischemia/MI (during the first 30 minutes) and the subacute phase (6 to 48 hours post-MI) when considering the mechanisms responsible for peri-infarction ventricular arrhythmias. Acute and delayed arrhythmias are distinguished by clinical arrhythmia type, cellular mechanisms, and immediate prognostic implications.

Arrhythmias occurring within the first 30 minutes of experimental coronary artery occlusion demonstrate the following bimodal distribution [18]:

- Arrhythmias occurring after the first 2 to 10 minutes are known as "immediate" or phase 1a ventricular arrhythmias and have a peak incidence after five minutes of ischemia. Reentry is the likely dominant mechanism for these arrhythmias.
- "Delayed" or phase 1b arrhythmias occur after approximately 10 to 60 minutes of coronary artery occlusion. Abnormal automaticity as well as reentry are likely the dominant mechanisms for these arrhythmias.

Delayed phase (6 to 48 hours) arrhythmias — Delayed-phase arrhythmias generally occur between six hours and as long as one to two days after the onset of the MI. These are also the result of abnormal or enhanced automaticity. The most frequently observed ventricular arrhythmias are [19,20]:

- Premature ventricular complex/contraction (PVC; also referred to a premature ventricular beats or premature ventricular depolarizations)
- Nonsustained VT (NSVT; three or more sequential PVCs lasting less than 30 seconds)
- Accelerated idioventricular rhythm, which is generally felt to be due to reperfusion of the occluded artery but is neither sensitive nor specific for definitive reperfusion

Chronic phase arrhythmias — During the chronic, healing phase of an acute MI (more than 48 hours), the typical patient with sustained monomorphic VT (SMVT) has had a large, often complicated infarct with an LV ejection fraction ≤30 percent [21,22]. The VT in this situation is usually reentrant and scar mediated, resulting from fibrosis due to the infarction. (See "Sustained monomorphic ventricular tachycardia in patients with structural heart disease: Treatment and prognosis".)

New ischemia can be superimposed upon this substrate with the appearance of a variety of ventricular arrhythmias, most commonly polymorphic VT or VF but also including NSVT and SMVT [23]. Polymorphic VT (associated with a normal QT interval of the sinus beat) and VF are the only arrhythmias associated with active ischemia, regardless of the cause. Thus, revascularization and anti-ischemic therapy may be part of the antiarrhythmic regimen. Autonomic imbalance, electrolyte abnormalities, and the proarrhythmic effects of antiarrhythmic drugs can also contribute to the appearance of SMVT or other ventricular arrhythmias.

LV remodeling after an MI produces structural changes in the myocardium that may also be factors in the pathogenesis of ventricular arrhythmia.

Reperfusion arrhythmias — Ventricular arrhythmias in some patients have been thought to be related to reperfusion, which may be spontaneous or occurring within a period of minutes after reperfusion has been achieved via fibrinolysis or mechanical means [24-26]. The evidence is best for an accelerated idioventricular rhythm (AIVR) as a reperfusion arrhythmia [25-29], usually the result of enhanced automaticity. However, AIVR is neither a sensitive nor very specific marker for successful reperfusion, as it may occur in other situations. (See "Diagnosis and management of failed fibrinolysis or threatened reocclusion in acute ST-elevation myocardial infarction", section on 'Primary failure'.)

Serious ventricular arrhythmia induced by reperfusion does not appear to be a major clinical problem [29,30]. As an example, large trials of intravenous thrombolytic therapy did not demonstrate any increase in life-threatening arrhythmias that could be attributed to reperfusion, although frequent PVCs and AIVRs did occur [8,9]. Similar findings have been noted after primary percutaneous coronary intervention [29].

CLINICAL FEATURES

Premature ventricular complex/contraction — Premature ventricular complexes/contractions (PVC; also referred to a premature ventricular beats, premature ventricular depolarizations, ventricular premature complexes, or ventricular premature beats), which are typically asymptomatic, are common after acute MI with a reported incidence as high as 93 percent [1]. The early occurrence of PVCs does not predict short- or long-term mortality, but frequent and/or multiform PVCs that persist more than 48 to 72 hours after an MI may be associated with an increased long-term arrhythmic risk, especially in patients with reduced left ventricular ejection fraction (LVEF) [31,32]. In the GISSI-2 trial, which evaluated 8676 patients with ST elevation MI (STEMI) treated with thrombolytic therapy who underwent 24-hour Holter monitoring before discharge, more than 10 PVCs per hour was a significant predictor for both total (relative risk [RR] 1.62, 95% CI 1.16-2.26) and sudden mortality (RR 2.24, 95% CI 1.22-4.08). [31]. (See "Incidence of and risk stratification for sudden cardiac death after myocardial infarction".)

While PVCs are not generally predictive of a risk of more serious arrhythmia, an exception is the presence of an R-on-T PCV, ie, those that occur right after the apex of the T wave. In the presence of ischemia, the R-on-T PVC is associated with an increased risk of provoking polymorphic VT or VF. (See "Premature ventricular complexes: Clinical presentation and diagnostic evaluation", section on 'R-on-T phenomenon'.)

PVCs produce few or no symptoms in the vast majority of patients, although some individuals may be incapacitated by palpitations or dizziness. PVCs rarely cause true hemodynamic compromise, except when they occur frequently in a patient with severely depressed LV function or when they are associated with an underlying bradycardia. The most common symptoms resulting from PVCs are palpitations secondary to the hypercontractility of a post-PVC beat or a feeling that the heart has stopped secondary to a post-PVC pause. Less often, frequent PVCs can result in a pounding sensation in the neck, lightheadedness, or near syncope.

Accelerated idioventricular rhythm — An accelerated idioventricular rhythm (AIVR), which has also been called "slow ventricular tachycardia," arises below the atrioventricular (AV) node (within the ventricular myocardium) and has, by definition, a rate between 50 and 100 beats/minute (waveform 1) [33]. It may be the result of pacemaker failure, and therefore be an escape rhythm, or it may represent an abnormal ectopic focus in the ventricle that is accelerated by sympathetic stimulation and circulating catecholamines.

The clinical presentation of AIVR can vary, but given the relatively slow rate and transient nature patients are frequently asymptomatic. Some patients may notice palpitations or lightheadedness, and there may be some associated hypotension if the rate results in a drop in cardiac output.

AIVR occurs in up to 50 percent of patients with acute MI, although it is not always documented on ECG as it may be transient. Some studies have suggested an association with reperfusion following fibrinolytic therapy [25]. However, AIVR is neither a sensitive nor very specific marker for successful reperfusion.

Monomorphic ventricular tachycardia — Nonsustained ventricular tachycardia (NSVT) is defined as three or more consecutive PVCs lasting less than 30 seconds, originating below the AV node (within the ventricular myocardium), with a heart rate greater than 100 beats/minute (waveform 2). VT is considered sustained if it lasts more than 30 seconds or is associated with hemodynamic collapse requiring prompt therapy. Monomorphic VT is identified by the presence of a single, consistent QRS morphology, which suggests that each beat arises from the same location (or the same reentrant circuit) and activates the ventricle in the same sequence. This uniformity should be present in all 12 ECG leads. As a result, a 12-lead ECG should be obtained in stable patients in order to fully characterize the VT morphology.

The clinical presentation may include palpitations, dizziness or lightheadedness, shortness of breath, worsening ischemic symptoms due to the elevated heart rate, and hemodynamic compromise or collapse. Patients with faster tachycardias and worse LV systolic function are less likely to tolerate the arrhythmia. Another concern is that sustained monomorphic VT (SMVT) may produce ischemia, which may be an important concern for the degeneration of SMVT into ventricular fibrillation (VF).

The impact of NSVT on mortality was evaluated in 6560 patients with non-ST-elevation acute coronary syndrome [34]. Only 1.2 percent of patients without VT experienced sudden cardiac death (SCD). Compared with patients with no VT, patients with four to seven beats of NSVT (2.9 percent SCD; adjusted hazard ratio [HR] 2.3, 95% CI 1.5-3.5) and patients with eight or more beats of NSVT (4.3 percent SCD; adjusted HR 2.8, 95% CI 1.5-4.9) had a higher risk of SCD.

The probable mechanism and the prognostic significance of NSVT depend upon the time at which it occurs in relation to infarction. In the early stage of an evolving infarction (ie, the first 6 to as many as 48 hours), monomorphic VT may result from transient arrhythmogenic phenomena in ischemic and infarcting tissue, such as abnormal automaticity, triggered activity, and reentrant circuits created by heterogeneous conduction and repolarization. In the first 24 to 48 hours after an infarction, nonsustained VT (NSVT) is usually due to abnormal automaticity or triggered activity in the region of ischemia or infarction. In comparison, NSVT that occurs later is more often due to reentry.

SMVT in any other setting is considered a marker of permanent arrhythmic substrate (ie, fibrosis and reentry) and an increased long-term risk of arrhythmia recurrence and SCD. Because of the physiologic and mechanistic link between SMVT and permanent substrate, it is not clear that SMVT at any point, even early after an MI, should be attributed to transient phenomena. Furthermore, SMVT in the setting of an acute MI may reflect permanent substrate from a prior infarction. As a result, the prognostic significance of SMVT in the early period after an MI is unclear. (See "Sustained monomorphic ventricular tachycardia in patients with structural heart disease: Treatment and prognosis".)

In fact, studies suggest that early SMVT is associated with higher in-hospital mortality due to cardiac arrest and possibly to exacerbation of ischemia and extension of the infarct [4,11,35-39], and may be a higher risk substrate than polymorphic VT [40].

Whether early SMVT is associated with an increased long-term mortality risk among contemporary patients undergoing revascularization who survive to hospital discharge is less well studied [4,11,14,35,37]. The SWEDEHEART registry included 2200 STEMI patients who underwent revascularization within 48 hours of presentation [40]. Among these patients, 150 had hemodynamically unstable VT, 35 had monomorphic VT, and 115 had nonmonomorphic VT (ie, polymorphic VT or VF). Mortality at eight years was higher in those with monomorphic compared with the nonmonomorphic VT (63 versus 37 percent). Among patients who did not have early hemodynamically significant VT, mortality was 27 percent.

Polymorphic VT — Compared with monomorphic VT and VF, polymorphic VT (with changing QRS morphology and axis and irregularly irregular rate in the absence of QT prolongation or congenital short QT interval) is much less common in the setting of an acute MI, occurring in 0.3 percent of patients in one report [41]. When polymorphic VT (waveform 3) does occur early after an acute MI, usually within the first 12 hours after the onset of symptoms, it is typically associated with symptoms or signs of recurrent myocardial ischemia [41]. Even if signs and symptoms are absent, myocardial ischemia, which is the most common cause, should be suspected. Polymorphic VT frequently accompanies episodes of coronary vasospasm

(Prinzmetal's angina). In some patients, the arrhythmia may be the only manifestation of ischemia (without angina or other more typical symptoms) [41]. The arrhythmia is not consistently related to electrolyte abnormalities, sinus bradycardia, preceding sinus pauses, or an abnormally long QT interval [41]. Polymorphic VT with prolonged QT interval or a congenital short QT interval of a sinus complex is termed "torsades de pointes," which has a different etiology and treatment. (See "Acquired long QT syndrome: Clinical manifestations, diagnosis, and management".)

When polymorphic VT develops, the type and intensity of symptoms will vary depending upon the rate and duration of VT along with the presence or absence of significant comorbid conditions. Patients with polymorphic VT and symptoms typically present with one or more of the following: sudden cardiac arrest, syncope/presyncope, "seizure-like" activity, or palpitations. Importantly, polymorphic VT that persists for more than 10 to 15 seconds often degenerates into VF.

Ventricular fibrillation — VF is the most frequent mechanism of SCD. It is a rapid, disorganized ventricular arrhythmia, resulting in no uniform ventricular activation or contraction, no cardiac output, and no recordable blood pressure. As such, patients invariably present with sudden collapse (syncope and/or sudden cardiac arrest). The ECG in VF shows rapid (300 to 400 beats/minute), irregular, shapeless QRST undulations of variable amplitude, morphology, and interval (waveform 4). Over time, these waveforms, which are initially coarse, decrease in amplitude and become finer undulations. Ultimately, asystole occurs.

The majority of episodes of VF occur within the first 48 to 72 hours after the onset of symptoms [4,5,10]. It is presumably a manifestation of ischemia and is associated with lack of perfusion via the infarct-related artery [37,42]. Factors that are associated with an increased risk of VF include [5,10,14,43-46]:

- STEMI
- Early repolarization (see "Early repolarization")
- Hypokalemia
- Hypotension
- Larger infarcts (based upon myocardial enzyme levels or the extent of wall motion abnormality on echocardiogram or LV angiogram)
- Male sex
- History of smoking
- Preinfarction angina (see "Myocardial ischemic conditioning: Clinical implications")
- Pre-PCI Thrombolysis in MI (TIMI) flow grade 0
- Inferior infarction

- Total baseline ST segment deviation
- Killip Class (table 1) greater than I

Prognosis after early VF — The occurrence of VF among patients with an acute STEMI, if occurring within the first 48 hours, is associated with an increase in early mortality (eg, in-hospital mortality) but little or no increase in mortality at one to two years among patients who survive to hospital discharge [4,10,37-39,47]. These data are quite old; similar data do not exist in contemporary patients treated with early revascularization.

Data are more limited in patients with a non-STEMI (NSTEMI). In a pooled analysis of patients with NSTEMI or unstable angina, VF was a significant predictor of increased mortality at both 30 days and six months (adjusted HR 23 and 15, respectively) [5]. The increase in risk was largely due to more deaths in the first 30 days.

Late arrhythmias — Late VT and VF are related to healing of the MI. Usually this reflects the development of scar tissue, which serves as an arrhythmogenic substrate that can promote the development of VT/VF. Fibrosis present in the scar leads to areas of conduction block with interdigitation of viable myocardium; the ensuing slowing of conduction at the border of the infarct can lead to stable reentry circuits and subsequent arrhythmias [48]. In general, late VT/VF is thought to portend risk of recurrent malignant arrhythmia and is usually treated specifically. (See "Secondary prevention of sudden cardiac death in heart failure and cardiomyopathy".)

In the CARISMA study, which prospectively observed 297 patients with a history of MI and LVEF of 40 percent or less (on excellent contemporary medical therapy) for late arrhythmias using an insertable cardiac monitor (also sometimes referred to as an implantable cardiac monitor or an implantable loop recorder), 13 percent of patients had at least one episode of NSVT (defined in this study as 16 or more beats, but less than 30 seconds in duration), 3 percent had an episode of sustained VT, and 3 percent had an episode of VF [49]. Over an average follow-up of two years, there was a nonsignificant trend toward increased mortality in patients with any ventricular arrhythmia.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Ventricular arrhythmias" and "Society guideline links: ST-elevation myocardial infarction (STEMI)" and "Society guideline links: Non-ST-elevation acute coronary syndromes (non-ST-elevation myocardial infarction)".)

SUMMARY AND RECOMMENDATIONS

- **Incidence** – Ventricular arrhythmias, ranging from isolated premature ventricular complexes/contractions (PVCs; also referred to a premature ventricular beats, premature ventricular depolarizations, ventricular premature complexes, or ventricular premature beats) to ventricular fibrillation (VF), are common in the immediate post-MI period. In the era of early percutaneous coronary intervention (PCI) and aggressive medical therapy, approximately 25 percent of patients with a non-ST elevation acute coronary syndrome experience ventricular tachycardia (VT) within the initial seven days, an event that portends a significantly greater risk of dying compared with patients without VT. (See 'Incidence' above.)

- **Mechanisms of arrhythmogenesis** – VT and VF in the setting of MI result from the complex interaction of multiple factors, including myocardial ischemia, necrosis, reperfusion, healing, and scar formation. Ventricular arrhythmias in the setting of acute MI result from an interplay among three basic components: injured myocardium, which is capable of developing reentrant circuits; arrhythmia triggers (eg, spontaneous PVCs, variations in cycle length); and modulating factors (eg, electrolyte imbalance, ongoing ischemia, autonomic nervous system). (See 'Mechanisms of arrhythmogenesis' above.)

- **Premature ventricular complex/contraction** – PVCs are common after acute MI with a reported incidence as high as 93 percent. PVCs produce few or no symptoms in the vast majority of patients, although some individuals may be incapacitated by palpitations or dizziness. The early occurrence of PVCs does not predict short- or long-term mortality. (See 'Premature ventricular complex/contraction' above.)

- **Accelerated idioventricular rhythm** – This is generally felt to be due to reperfusion of the occluded artery, which occurs in up to 50 percent of patients with acute MI. The clinical presentation of AIVR can vary, but given the relatively slow rate and transient nature patients are frequently asymptomatic, although some patients may notice palpitations or lightheadedness, and there may be some associated hypotension if the rate results in a drop in cardiac output. (See 'Accelerated idioventricular rhythm' above.)

- **Monomorphic VT** – VT is considered sustained if it lasts more than 30 seconds or is associated with hemodynamic collapse requiring prompt therapy. The clinical presentation may include palpitations, worsening ischemic symptoms due to the elevated heart rate, and hemodynamic compromise or collapse. Patients with faster tachycardias and worse LV

systolic function are less likely to tolerate the arrhythmia. (See 'Monomorphic ventricular tachycardia' above.)

- **Polymorphic VT** – Polymorphic VT (in the absence of QT prolongation of a sinus complex) is much less common in the setting of an acute MI, but when polymorphic VT occurs early after an acute MI, usually within the first 12 hours after the onset of symptoms, it is typically associated with symptoms or signs of recurrent myocardial ischemia. Patients with polymorphic VT and symptoms typically present with one or more of the following: sudden cardiac arrest, syncope/presyncope, "seizure-like" activity, or palpitations. Importantly, polymorphic VT that persists for more than 10 to 15 seconds often degenerates into VF. (See 'Polymorphic VT' above.)

- **VF** – VF is the most frequent mechanism of SCD. It is a rapid, disorganized ventricular arrhythmia, resulting in no uniform ventricular contraction, no cardiac output, and no recordable blood pressure. As such, patients invariably present with sudden collapse (syncope and/or sudden cardiac arrest). (See 'Ventricular fibrillation' above.)

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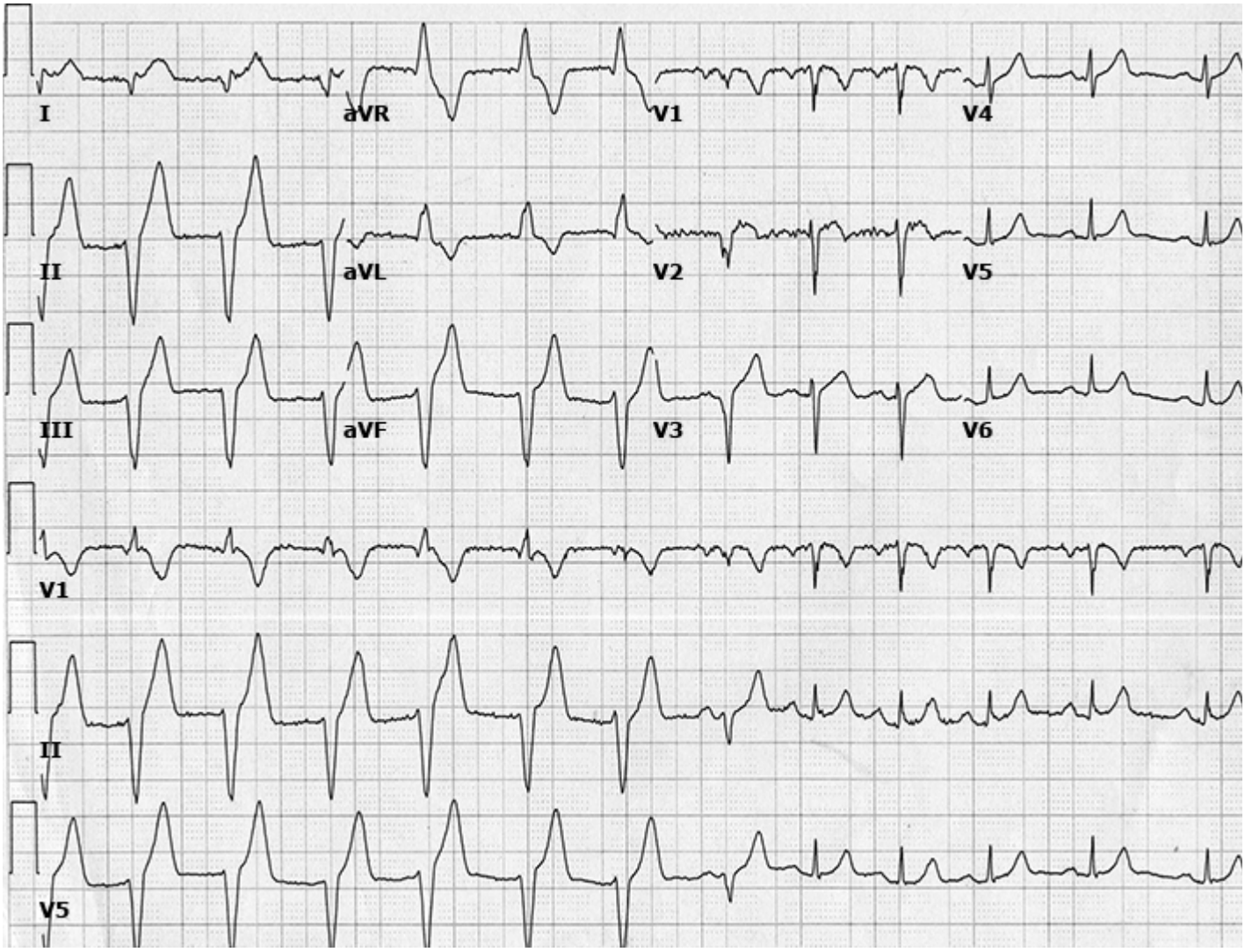
Topic 1060 Version 41.0

GRAPHICS

ECG 12-lead accelerated idioventricular rhythm

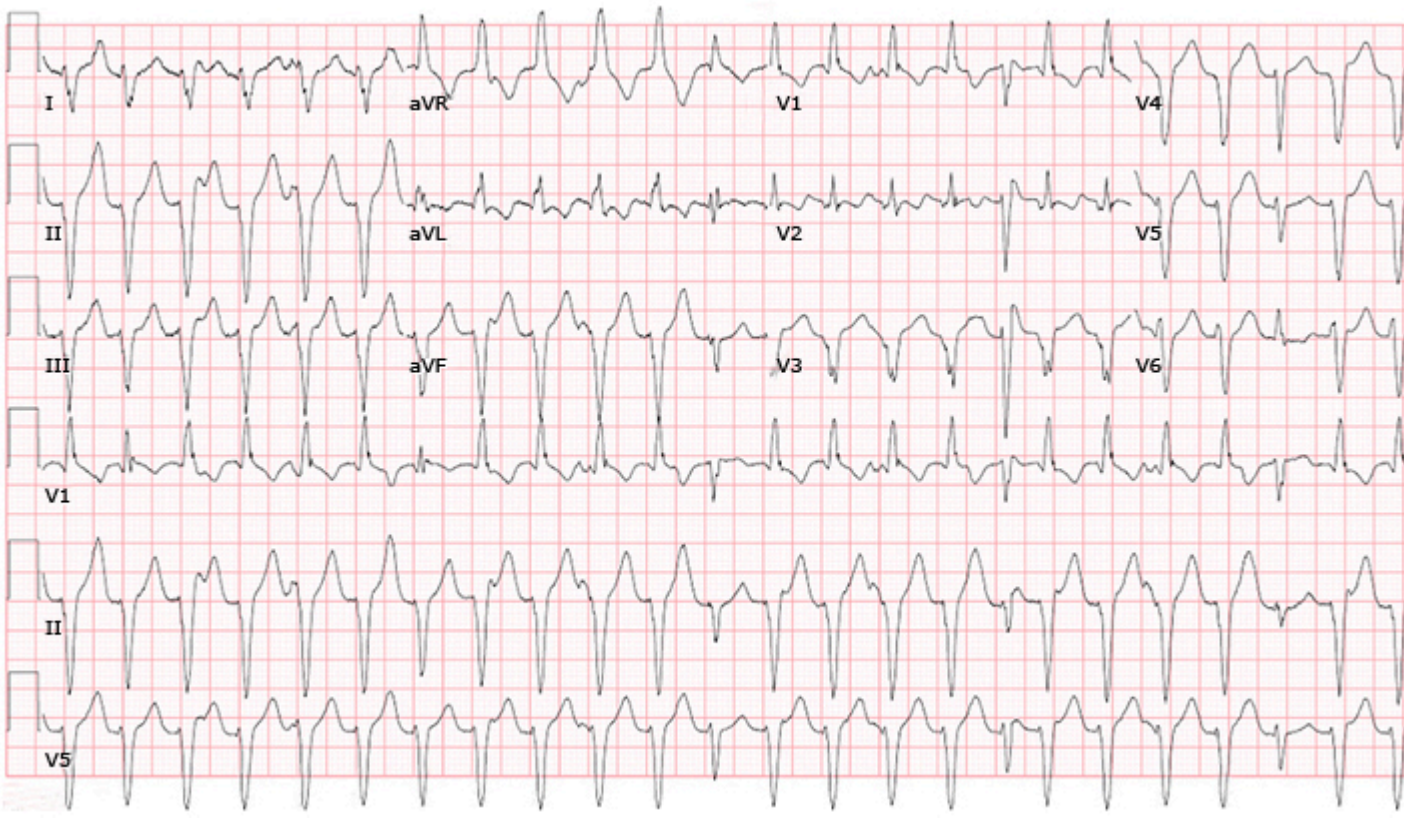


12-lead ECG showing idioventricular rhythm with AV dissociation and wide QRS complexes occurring at a rate faster than the sinus rate but slower than 100 bpm (hence not meeting the criteria for ventricular tachycardia).

ECG: electrocardiogram.

Graphic 118943 Version 2.0

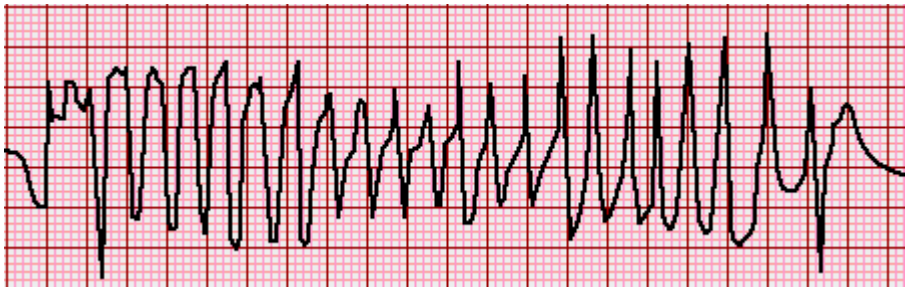
12-lead ECG sustained monomorphic VT



The electrocardiogram (ECG) hallmark for the diagnosis of sustained monomorphic ventricular tachycardia (SMVT) is a wide complex tachycardia with the obvious presence of atrioventricular (AV) dissociation. AV dissociation is suggested by the presence of fusion complexes (which reflect a supraventricular impulse coming from above the AV node fusing with an impulse generated in the ventricle) or captured complexes (which reflect an impulse coming from above the AV node that depolarizes the ventricles when they are no longer refractory but before the next ventricle-generated complex). Beats 12, 17, and 22 on this ECG likely represent capture beats.

Graphic 111254 Version 1.0

Single lead electrocardiogram (ECG) showing polymorphic ventricular tachycardia (VT)



This is an atypical, rapid, and bizarre form of ventricular tachycardia that is characterized by a continuously changing axis of polymorphic QRS morphologies.

Graphic 53891 Version 5.0

ECG 12-lead ventricular fibrillation

12-lead ECG showing course ventricular fibrillation.

ECG: electrocardiogram.

Graphic 118944 Version 1.0

Killip classification of acute myocardial infarction

Class I	No evidence of heart failure
Class II	Findings consistent with mild to moderate heart failure (eg, S3 gallop, lung rales less than one-half way up the posterior lung fields, or jugular venous distension)
Class III	Overt pulmonary edema
Class IV	Cardiogenic shock

Graphic 65592 Version 7.0