Ventricular arrhythmias during acute myocardial infarction: Prevention and treatment

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Literature review current through: **Jan 2024.**
This topic last updated: **Jun 14, 2023.**

INTRODUCTION

Sudden cardiac death (SCD) 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) 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 prevention and treatment of ventricular arrhythmias during and immediately after acute MI. The incidence, mechanisms, and clinical features of ventricular arrhythmias during acute MI, as well as treatment of ventricular arrhythmias late post-MI (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: Incidence, mechanisms, and clinical features".)

PREVENTION

Frequent ventricular premature beats (VPBs), VT, and ventricular fibrillation (VF) are all associated with increased long-term mortality following acute MI. An acute MI may be an ST-segment elevation MI (STEMI) or non-ST-segment elevation MI (NSTEMI). Most of the data available are in patients with a STEMI. While the data may also apply to patients with an NSTEMI, information in these patients is more limited. The following is a summary of the multi-modality approach to prevention of ventricular arrhythmias following MI (STEMI), which includes treatment of ischemia, electrolyte supplementation (if needed), and beta blockers. Treatment of symptomatic arrhythmias (should they arise) is discussed elsewhere. (See 'Treatment' below.)

Revascularization/treatment of myocardial ischemia — Patients with ventricular arrhythmias, especially polymorphic VT, in the setting of an acute MI should receive aggressive treatment for both the arrhythmia and myocardial ischemia. Therapy for ischemia usually includes drugs (eg, beta blockers, nitrates) and in most cases primary percutaneous coronary intervention or far less frequently, coronary artery bypass grafting for revascularization [1]. Fibrinolytic therapy is also effective but is used infrequently and generally only when PCI is not immediately available. The acute and long-term treatments of ischemic heart disease are discussed in detail separately. (See "Overview of the acute management of ST-elevation myocardial infarction" and "Overview of the acute management of non-ST-elevation acute coronary syndromes" and "Prevention of cardiovascular disease events in those with established disease (secondary prevention) or at very high risk".)

Electrolyte supplementation — In the post-MI setting, we maintain levels of serum potassium ≥4 mEq/L and serum magnesium ≥2 mg/dL. The dose and route of potassium and/or magnesium are discussed elsewhere. (See "Clinical manifestations and treatment of hypokalemia in adults" and "Hypomagnesemia: Evaluation and treatment".)

Hypokalemia is a risk factor for VF in the setting of an acute MI, and concomitant hypomagnesemia, which is detected in approximately 40 percent of cases of hypokalemia, prevents the correction of hypokalemia [2].

- In the GISSI-2 trial, the likelihood of VF among patients with a serum potassium <3.6 mEq/L was almost twice as high as among patients with a higher serum potassium (odds

ratio 1.97) [3]. (See "Ventricular arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features", section on 'Ventricular fibrillation'.)

- The MAGIC trial showed no benefit to empiric magnesium supplementation in acute MI patients [4].

Beta blockers — Oral beta blockers are administered universally to all patients without contraindications who experience an acute MI [5]. In addition to other beneficial effects, the immediate administration of a beta blocker during an acute MI reduces the risk of VF. In a systematic review, the overall mortality in 31 long-term trials that included almost 25,000 patients was 9.7 percent; beta blockers reduced the odds of death by 23 percent (95% CI 15-31 percent) [6]. These benefits are seen following both STEMI and NSTEMI (figure 1) [7]. The details of beta blocker use are discussed separately. (See "Acute myocardial infarction: Role of beta blocker therapy".)

Beta blockers are of use as the etiology of ventricular arrhythmia in the early or acute stages of an MI is in part related to enhanced automaticity, resulting from elevated catecholamines and beta receptor stimulation. (See "Ventricular arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features".)

Antiarrhythmic drugs — We do not administer prophylactic antiarrhythmic agents in the post-infarction period.

- The prophylactic administration of class IC antiarrhythmic agents (eg, encainide, flecainide) in the post-MI period is associated with increased mortality and is **not** recommended [8-10].

- Due to the suggestion of possible harm and unsure benefit, the routine prophylactic administration of lidocaine in the acute MI period is **not** recommended [11].

- Unlike the other antiarrhythmic drugs, prophylactic amiodarone is not associated with an increase in mortality. However, its unselected use in all patients does not appear to improve outcomes. As such, we do not administer prophylactic amiodarone in the post-MI period.

The use of these agents is reserved for patients with documented ventricular tachyarrhythmias. (See 'Treatment' below.)

Heart failure therapy — Although not considered antiarrhythmic drugs, angiotensin converting enzyme (ACE) inhibitors, aldosterone antagonists, angiotensin II receptor blockers (ARBs), and combination ARB and neprilysin inhibitor all reduce the incidence of SCD in patients

with heart failure (HF). Reduced SCD rates have been reported specifically in post-MI populations with ACE inhibitors and aldosterone antagonists, and in a broader population of HF patients, approximately 50 percent of whom had a prior infarction, with an ARB. These topics are discussed separately. (See "Angiotensin converting enzyme inhibitors and receptor blockers in acute myocardial infarction: Clinical trials", section on 'Effect on sudden death'.)

Wearable defibrillator for primary prevention — Among patients with left ventricular ejection fraction (LVEF) ≤35 percent who are less than 40 days post-MI, we discuss the potential benefits and risks of wearable cardioverter-defibrillator (WCD) use (picture 1) and consider providing it to motivated patients with NYHA functional class II or III, or LVEF <30 percent and in NYHA class I, as these patients would be candidates for implantable cardioverter-defibrillator (ICD) implantation after 40 days. However, one study has not shown improvement in mortality in such patients as a result of WCD use [12]. In another analysis of this trial, it was reported that the lack of benefit might be in part related to poor compliance among patients prescribed the WCD [13]. The role of the WCD is discussed in detail separately. (See "Wearable cardioverter-defibrillator".)

TREATMENT

Ventricular premature beats — In the post-MI patient with ventricular premature beats (VPBs) that cause significant or disabling symptoms (eg, palpitations, lightheadedness), beta blockers are administered, although most patients will already be taking them. In the rare circumstance that more aggressive antiarrhythmic therapy is considered for control of refractory symptoms, we prefer amiodarone, as it is likely to be effective and unlikely to cause significant harm, although there is an appreciable incidence of side effects with long-term amiodarone therapy. Mexiletine, which is a class IB agent that resembles lidocaine, also appears safe in the post-MI patient, and although there are no randomized trials in this population, it may be effective for arrhythmia suppression [14,15]. (See "Amiodarone: Adverse effects, potential toxicities, and approach to monitoring".)

There is no role for chronic antiarrhythmic drug therapy to suppress asymptomatic VPBs. (See 'Prevention' above.)

VPBs, particularly if frequent (more than 10 per hour) or complex (ie, couplets or non-sustained ventricular tachycardia) appear to be associated with a worse prognosis in patients with a prior MI. Based upon this association, trials of both class I and class III antiarrhythmic medications were conducted to determine if suppression of ventricular ectopy would reduce SCD. Patients

with frequent asymptomatic VPBs post-MI were randomly assigned to receive suppressive antiarrhythmic therapy or placebo in an effort to suppress the ectopy [8,9].

- The CAST study, which randomly assigned patients to treatment with encainide, flecainide, moricizine, or placebo, was prematurely terminated when it was noted that, despite suppression of VPBs, total mortality among the patients receiving encainide and flecainide was significantly **increased** compared with those on placebo (7.7 versus 3.0 percent); this was due primarily to an excess in arrhythmic deaths (figure 2).

- The CAST II study, which limited treatment to moricizine or placebo, was also terminated early due to an increased risk of death or cardiac arrest in the first 14 days of therapy among patients treated with moricizine (2.6 versus 0.5 percent with placebo) [9].

- The CAMIAT study, which randomly assigned patients with frequent (≥10 per hour) or repetitive VPBs to amiodarone or placebo (approximately 60 percent were also treated with a beta blocker), showed that although arrhythmia suppression was more common with amiodarone (84 versus 35 percent with placebo), there was no significant difference in yearly all-cause or cardiac mortality (4.0 versus 5.2 percent) [16].

Nonsustained VT — For patients with symptomatic (eg, palpitations, lightheadedness) nonsustained ventricular tachycardia (NSVT) after an MI, beta blockers are administered, although most patients should already be taking them. If antiarrhythmic drug therapy is considered due to persistent symptoms, we prefer amiodarone, as it is likely to be effective and unlikely to cause significant harm, although there is an appreciable incidence of side effects with long-term amiodarone therapy. An alternative agent is mexiletine as it is safe and has been found to be effective for arrhythmia suppression in other groups of patients [14,15]. (See "Amiodarone: Adverse effects, potential toxicities, and approach to monitoring".)

In the absence of data specific to patients with NSVT, we do not prescribe chronic antiarrhythmic drug therapy to suppress asymptomatic NSVT. The presence of NSVT in post-MI patients with an LVEF ≤40 percent is an indication for further risk stratification, if the patient does not already meet criteria for ICD placement (LVEF ≤30 percent without heart failure symptoms, or LVEF ≤35 percent with NYHA class II or III heart failure). Electrophysiologic testing prior to hospital discharge may be appropriate in patients with late NSVT (ie, more than 24 to 48 hours into acute MI). (See "Incidence of and risk stratification for sudden cardiac death after myocardial infarction".)

The development of NSVT one week or later post-MI carries at least a twofold increase in the risk of SCD [17]. The risk of NSVT is even further increased in post-MI patients with significantly

diminished LV function (LVEF less than 40 percent). In this setting, the risk of SCD is increased more than fivefold [17,18].

Large randomized trials of antiarrhythmic drugs limited to patients with NSVT have not been performed. However, many of the patients included in the CAST and CAMIAT (39 percent) trials had NSVT. These trials showed an increased mortality in patients treated with class IC antiarrhythmic medications [8] and no significant reduction in overall mortality with amiodarone [16].

Accelerated idioventricular rhythm — An accelerated idioventricular rhythm (AIVR), which has also been called "slow VT," arises below the atrioventricular (AV) node and has, by definition, a rate between 50 and 100 beats/minute (waveform 1). Most episodes are transient, benign, and require no treatment. Furthermore, pharmacologic therapy is contraindicated if there is complete heart block and an escape ventricular rhythm (which is not actually AIVR), since suppression of the pacemaker focus can result in profound bradycardia and possibly asystole.

AIVR is most often seen in the peri-infarction period. AIVR often occurs after reperfusion therapy (PCI or fibrinolytic therapy) and is felt to be a reperfusion arrhythmia. It is likely the result of enhanced automaticity of ectopic foci in the ventricular myocardium. AIVR occurring after the peri-infarction period is uncommon. When it occurs, reversible causes should be sought such as digitalis toxicity, hypokalemia, or hypomagnesemia. There are no convincing data linking AIVR to sustained VT, ventricular fibrillation (VF), or a worse prognosis. Thus, no therapy is warranted for asymptomatic arrhythmias, while symptomatic arrhythmias can be treated with a beta blocker (if the patient is not already receiving this therapy), antiarrhythmic drugs, or perhaps ablation.

Polymorphic VT — Polymorphic VT associated with a normal QT interval is an uncommon arrhythmia following an acute MI (waveform 2 and waveform 3). When it occurs, it is often associated with signs or symptoms of recurrent or ongoing myocardial ischemia [19]. Even if there are no signs or symptoms of myocardial ischemia, underlying myocardial ischemia is the most likely etiology. If polymorphic VT lasts for more than 8 to 10 seconds, it often degenerates into VF. This type of polymorphic VT generally fails to respond to class I antiarrhythmic drugs, magnesium, or overdrive pacing but may respond to intravenous amiodarone or lidocaine [19]. In patients treated with primary percutaneous coronary intervention who then manifest polymorphic ventricular tachycardia , revisualization of the coronary arteries is frequently warranted. An intraaortic balloon pump or other mechanical unloading therapy may help stabilize these patients. (See "Ventricular arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features", section on 'Polymorphic VT'.)

Revascularization has traditionally been considered to be adequate therapy for polymorphic VT due to ischemia in the absence of acute MI. However, more recent data suggest that ICD implantation in addition to revascularization may be optimal [20].

There is a second form of polymorphic VT that develops during the healing phase (at 3 to 11 days) and occurs in association with QT prolongation [21]. This arrhythmia resembles an acquired long QT syndrome and is treated in a similar fashion. One report of eight such patients found that defibrillation (if the arrhythmia is sustained), magnesium, lidocaine, beta blockers, and rapid overdrive pacing were effective therapies [21]. Mexiletine, which is similar to lidocaine but is an oral medication, may be of benefit. In general, the QT interval shortened within 10 days, and long-term outcomes were uneventful.

More commonly, polymorphic VT results from either acquired or congenital long QT interval and in this situation it is called torsades de pointes. Acquired QT prolongation may result from class IA or class III antiarrhythmic drugs, which can prolong the QT interval. An exception is amiodarone, which rarely produces torsades de pointes when used alone. Amiodarone should be discontinued if the burden of polymorphic VT increases, which is a possible sign of proarrhythmia. Many other drugs, including antibiotics, psychotropic agents, antihistamines, and GI medications may prolong the QT interval. Magnesium supplementation may be of benefit, even in the absence of hypomagnesemia. Bradycardia frequently facilitates the initiation of torsades de pointes VT in a susceptible patient with drug-induced QT prolongation as the QT interval lengthens further with slower heart rates. Increasing the heart rate, as with temporary pacing, may help prevent recurrent episodes. Although increasing the heart rate with intravenous isoproterenol or dobutamine may also be effective, this should be done with caution in the patient with an acute myocardial infarction. (See "Acquired long QT syndrome: Definitions, pathophysiology, and causes" and "Temporary cardiac pacing", section on 'Indications'.)

Sustained monomorphic VT and VF — Unstable, poorly-tolerated arrhythmias are a life-threatening emergency that are treated according to established advanced cardiac life support (ACLS) protocols [22].

- VF is almost universally lethal if not treated. VF does not self-terminate nor does it revert with antiarrhythmic drugs. Defibrillation (nonsynchronized delivery of a shock) is the definitive therapy for VF. If available, a biphasic waveform defibrillator is preferable since the success rate for defibrillation is higher than with monophasic waveforms. The 2015 American Heart Association (AHA) guidelines for adult ACLS recommended that, for biphasic defibrillators, the initial shock should be at 120 to 200 joules, with subsequent shocks at the highest available biphasic energy level (200 joules for most devices) [22]. For

monophasic defibrillators, nonescalating shocks beginning at 360 joules should be used. (See "Advanced cardiac life support (ACLS) in adults" and "Basic principles and technique of external electrical cardioversion and defibrillation".)

- Hemodynamically unstable or pulseless sustained monomorphic VT (SMVT) without identifying a distinct QRS should be treated with **unsynchronized** electrical shocks (ie, defibrillation and not cardioversion, which is the delivery of an electrical shock synchronized to the QRS complex). If available, a biphasic waveform defibrillator is preferable since the success rate for defibrillation is higher than with monophasic waveforms. For biphasic defibrillators, the initial shock should be at 120 to 200 joules, with subsequent shocks at the highest available biphasic energy level (200 joules for most devices). For monophasic defibrillators, nonescalating shocks beginning at 360 joules should be used. (See "Cardioversion for specific arrhythmias", section on 'Ventricular tachycardia'.)

- SMVT (in which a distinct QRS complex can be identified) associated with angina, pulmonary edema, or hypotension (systolic blood pressure <90 mmHg) should be treated immediately with **synchronized** electrical cardioversion using an initial energy of 50 to 100 joules. Subsequent shocks at increasing energy can be given as necessary. Brief anesthesia is desirable if hemodynamically tolerable.

- SMVT that is hemodynamically tolerated and asymptomatic can be treated initially with intravenous amiodarone (or lidocaine or procainamide). Synchronized electrical cardioversion with brief anesthesia should be performed if VT persists after the administration of the initial 150 mg of amiodarone (or 100 to 300 mg of lidocaine). (See "Sustained monomorphic ventricular tachycardia in patients with structural heart disease: Treatment and prognosis", section on 'Stable patients' and "Cardioversion for specific arrhythmias", section on 'Ventricular tachycardia'.)

- Recurrent nonsustained and sustained VT, particularly polymorphic, should trigger consideration of investigation for ischemia, possibly (but not always) involving the infarct-related artery. (See 'Revascularization/treatment of myocardial ischemia' above.)

- Patients who manifest sustained VT or polymorphic VT more than 24 to 48 hours after acute MI should generally undergo ICD implantation prior to hospital discharge. (See "Secondary prevention of sudden cardiac death in heart failure and cardiomyopathy".)

The acute and long-term management of SMVT and VF in patients with a prior MI are discussed in detail separately. (See "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".)

Electrical storm — Electrical storm is defined as multiple recurrent episodes of VF. The optimal therapy of electrical storm in patients with an acute MI is uncertain but is likely no different than in patients with electrical storm in any setting and includes defibrillation, antiarrhythmic medications, beta blockers, treatment of myocardial ischemia, and long-term therapies to prevent recurrent arrhythmias (eg, catheter ablation, cardiac sympathetic denervation, etc). (See "Electrical storm and incessant ventricular tachycardia".)

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: Non-ST-elevation acute coronary syndromes (non-ST-elevation myocardial infarction)" and "Society guideline links: ST-elevation myocardial infarction (STEMI)" and "Society guideline links: Ventricular arrhythmias".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5^{th} to 6^{th} grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10^{th} to 12^{th} grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Ventricular tachycardia (The Basics)" and "Patient education: Ventricular fibrillation (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Revascularization** – Patients with ventricular arrhythmias, especially polymorphic VT, in the setting of an acute MI should receive aggressive treatment for both the arrhythmia and myocardial ischemia. Therapy for ischemia usually includes drugs (eg, beta blockers, nitrates) and either primary percutaneous coronary intervention or coronary artery bypass grafting for revascularization. (See 'Revascularization/treatment of myocardial ischemia' above.)

- **Preventive therapies** – Other therapies that reduce ventricular arrhythmias during and immediately after acute MI include maintaining levels of serum potassium ≥4.0 mEq/L and serum magnesium ≥2.0 mg/dL, administering beta blockers, and appropriate therapies for heart failure, when present. (See 'Prevention' above.)

- **Antiarrhythmic drugs** – The prophylactic administration of antiarrhythmic agents to asymptomatic patients during and immediately after acute MI has at best no benefit and potentially can cause harm. The use of these agents is reserved for patients with documented ventricular tachyarrhythmias. (See 'Antiarrhythmic drugs' above and 'Treatment' above.)

- **Ventricular premature beats** – In the post-MI patient with ventricular premature beats or nonsustained VT that cause significant or disabling symptoms (eg, palpitations, lightheadedness), beta blockers are administered, although most patients will already be taking them. In the rare circumstance that more aggressive antiarrhythmic therapy is considered for control of refractory symptoms, we prefer amiodarone, as it is likely to be effective and unlikely to cause significant harm. (See 'Ventricular premature beats' above.)

- **Accelerated idioventricular rhythm** – Most episodes of accelerated idioventricular rhythm (AIVR) are transient, benign, and require no treatment. Furthermore, pharmacologic therapy is contraindicated if there is complete heart block and an escape ventricular rhythm (which is not actually AIVR), since suppression of the pacemaker focus can result in profound bradycardia and possibly asystole. (See 'Accelerated idioventricular rhythm' above.)

- **Polymorphic VT** – This is associated with a normal QT interval, is an uncommon arrhythmia, and is often associated with signs or symptoms of recurrent or ongoing myocardial ischemia, in which case revisualization of the coronary arteries is usually warranted. Polymorphic VT that results from acquired long QT interval is called torsades de pointes and is usually related to medications. (See 'Polymorphic VT' above.)

- **Poorly-tolerated arrhythmias** – Sustained monomorphic VT or VF is a life-threatening emergency that is treated according to established advanced cardiac life support (ACLS)

protocols. (See "Advanced cardiac life support (ACLS) in adults" and 'Sustained monomorphic VT and VF' above.)

ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Leonard Ganz, MD, FHRS, FACC, who contributed to an earlier version of this topic review.

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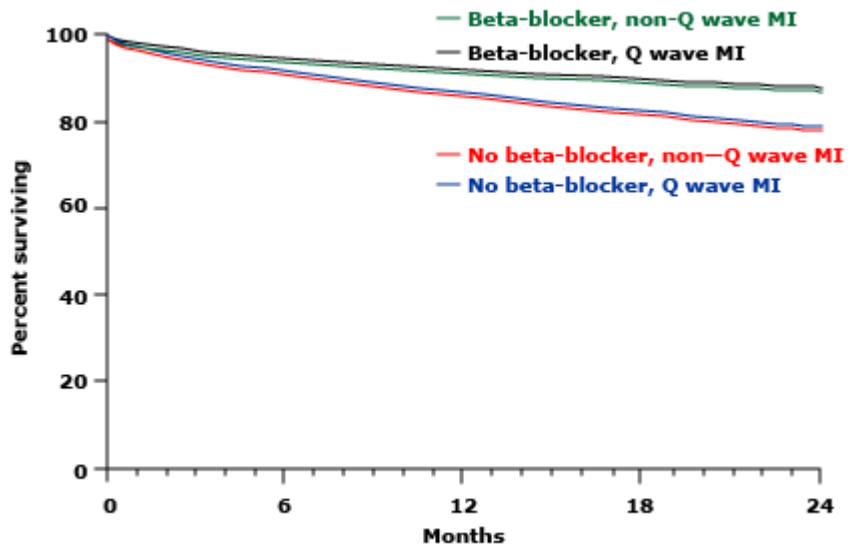
Topic 118996 Version 17.0

GRAPHICS

Beta blockers are equally effective after a Q wave or non-Q wave MI

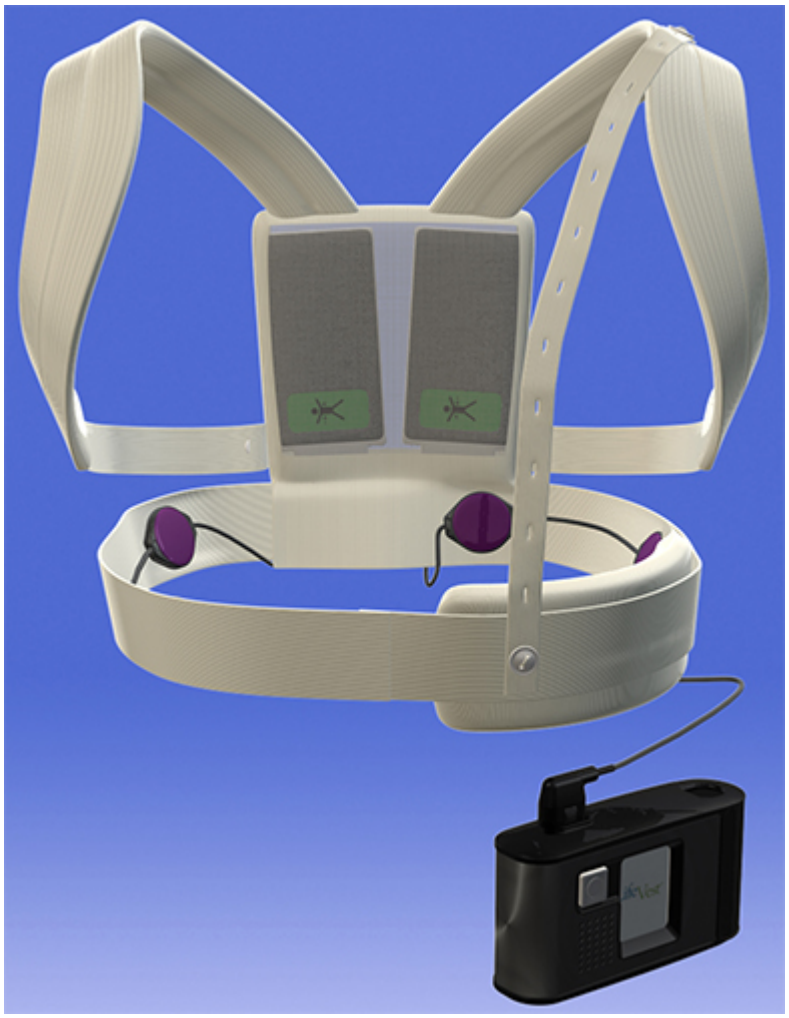


Analysis of data from 201,752 patients with a myocardial infarction (MI) demonstrates that the reduction in mortality at two years with beta blockers is similar in those with a Q wave (14.2 versus 23.6 percent for those not receiving beta blockers) or non-Q wave MI (14.4 versus 23.9 percent).

Data from Gottlieb SS, McCarter RJ, Vogel RA. N Engl J Med 1998; 339:489.

Graphic 79592 Version 2.0

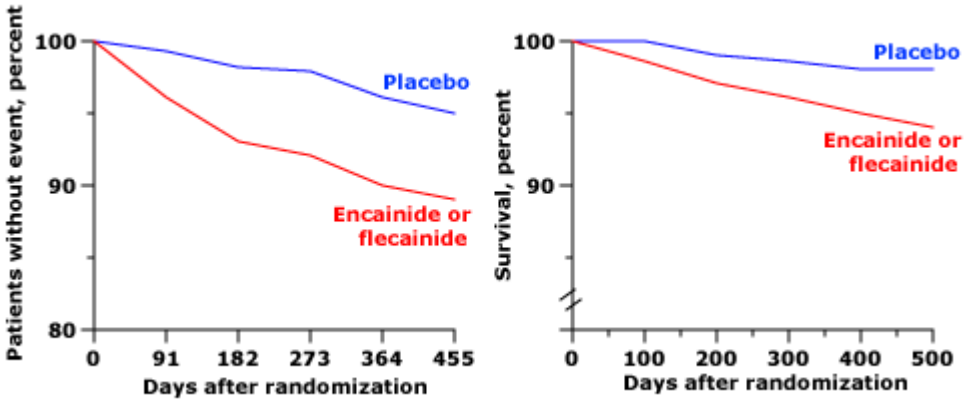
Wearable cardioverter-defibrillator



The wearable cardioverter-defibrillator consists of a vest incorporating two defibrillation electrodes and four sensing electrocardiographic electrodes connected to a waist unit containing the monitoring and defibrillation electronics.

Graphic 60103 Version 3.0

Encainide and flecainide increase cardiac mortality

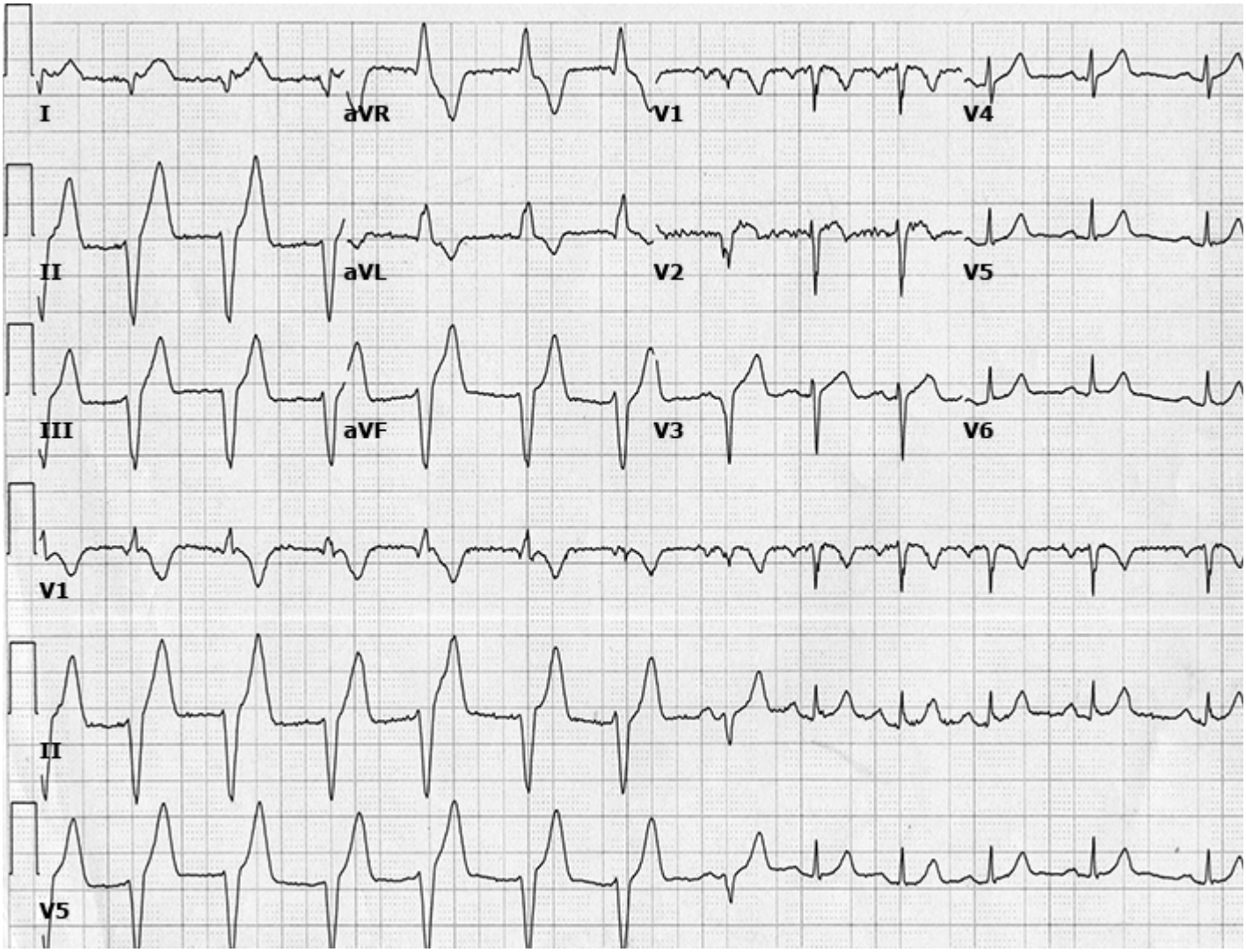


Results of the Cardiac Arrhythmia Suppression Trial (CAST) in patients with ventricular premature beats after myocardial infarction. Patients receiving encainide or flecainide had, when compared with those receiving placebo, a significantly lower rate of avoiding a cardiac event (death or resuscitated cardiac arrest) (left panel, p = 0.001) and a lower overall survival (right panel, p = 0.0006). The cause of death was arrhythmia or cardiac arrest.

Data from Echt DS, Liebson PR, Mitchell B, et al. N Engl J Med 1991; 324:781.

Graphic 59975 Version 5.0

ECG 12-lead accelerated idioventricular rhythm

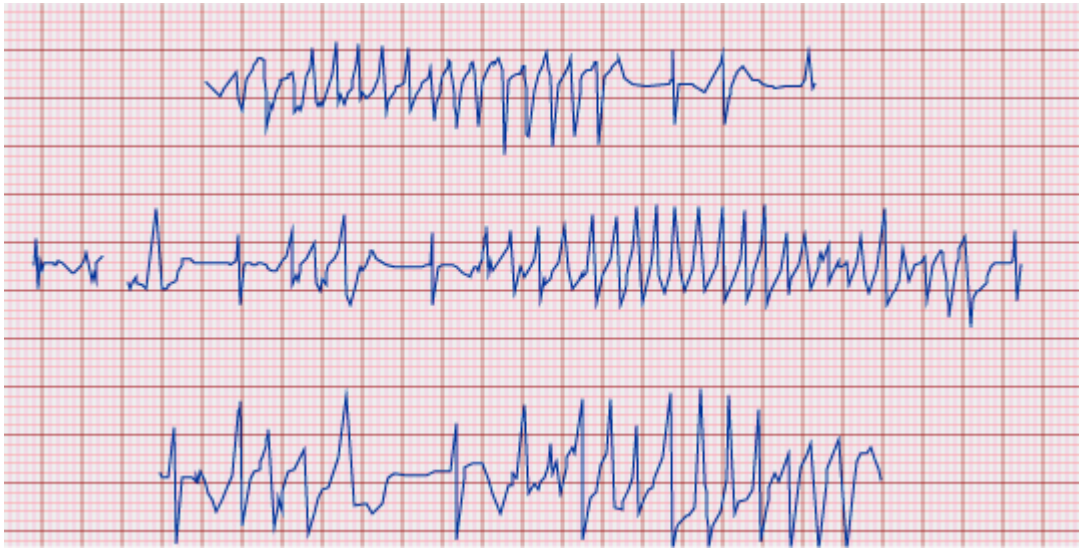


12-lead ECG showing idioventricular rhythm with AV dissociation and wide QRS complexes occurring at a rate faster than the sinus rate but slower than 100 bpm (hence not meeting the criteria for ventricular tachycardia).

ECG: electrocardiogram.

Graphic 118943 Version 2.0

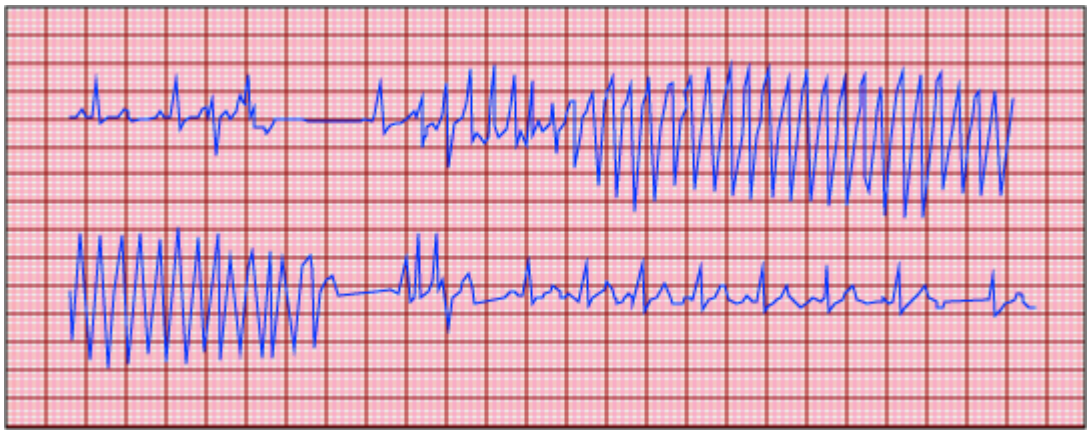
ECG_1 showing polymorphic ventricular tachycardia in ischemia



Continuous rhythm strip revealing several episodes of nonsustained ventricular tachycardia (VT) occurring during an acute ischemic event. The QRS complexes are variable in morphology and RR intervals; thus, the VT is polymorphic. The QT interval is normal. This form of VT should be distinguished from torsade de pointes in which polymorphic VT is associated with QT interval prolongation.

Graphic 54538 Version 3.0

ECG_2 showing polymorphic ventricular tachycardia in ischemia



Continuous rhythm strip showing an episode of very rapid polymorphic ventricular tachycardia which is often referred to as ventricular flutter. The QT interval is normal and the QRS complex morphology is highly variable. The patient had an underlying sinus tachycardia, suggesting increased sympathetic activity secondary to an ischemic event.

Graphic 67322 Version 3.0