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Tachyarrhythmias in Myocardial Infarction

By ROMAN W. DESANCTIS, M.D., PETER BLOCK, M.D.,
AND ADOLPH M. HUTTER, JR., M.D.

SUMMARY
Tachyarrhythmias occur in about one third of patients with acute myocardial infarction (MI), and may precipitate serious consequences when they arise. Mechanisms of arrhythmogenesis in MI are imperfectly understood, but five categories of factors contributing to ectopic tachycardias are discussed. These include metabolic, anatomic, autonomic, hemodynamic, and iatrogenic causes. Each of the atrial, junctional, and ventricular tachyarrhythmias is briefly considered, and therapy is outlined. Prevention of tachyarrhythmias and prompt treatment when they occur have substantially reduced mortality in acute MI, and constitute a primary objective of coronary care. However, better understanding of mechanisms of arrhythmogenesis, better systems of automated monitoring, and better modes of therapy, especially antiarrhythmic drugs, are urgently needed.

Additional Indexing Words:
Ectopic tachycardias      Electrocardiographic monitoring
Antiarrhythmic drugs

DEVELOPMENTS IN CORONARY care specifically aimed at preventing death from arrhythmias in acute myocardial infarction (MI) constitute one of the major medical advances of the past decade. The widespread application of constant electrocardiographic monitoring to patients with acute MI has revealed an incidence of arrhythmias previously wholly unsuspected. Indeed, some disturbance of rate, rhythm, or conduction is detected in the vast majority of closely monitored patients within the first week after infarction.1, 2, 3 Although arrhythmias in MI can be categorized in many ways, a convenient classification is one which simply divides them into those in which the heart rate is either too slow (bradyarrhythmias) or too fast (tachyarrhythmias). This paper concerns itself with tachyarrhythmias complicating acute MI.

Ectopic tachycardias, whether arising in the atria, atrioventricular (A-V) junction, or ventricles, may precipitate serious and even fatal consequences for the patient in whom they occur. The rapid heart rate often leads to a sharp fall in cardiac output with hypotension and to diminished perfusion of vital organs, one of the most important of which is the heart itself. Similarly, tachycardia may cause congestive failure, particularly when the
deleterious effects of the rapid heart rate are
superimposed upon a myocardium whose
function is already compromised by infarction.
The accelerated heart rate increases the
demand of the myocardium for oxygen at a
time when it can least afford it. Thus, a
cardinal role of the coronary care unit is the
prevention of tachyarrhythmias and their
termination should they occur.

Causes of Tachyarrhythmias

Although much attention has been directed
toward the treatment of infarct arrhythmias,
considerably less has been paid to those
factors which give rise to the disordered
rhythms. We would like to consider briefly
five broad categories of arrhythmogenic fac-
tors in MI: (1) metabolic; (2) anatomic;
(3) autonomic; (4) hemodynamic; and (5)
iatrogenic.

Metabolic

Metabolic factors are clearly among the
most important causes of MI arrhythmias, but
precise mechanisms of arrhythmogenesis are
imperfectly understood at present. Beck et al.
found that arrhythmias in experimental myo-
cardial infarction appeared to arise in the
interzone between infarcted and noninfarcted
myocardium. They postulated that the dif-
ference in tissue oxygen tensions between
ischemic and well-perfused regions of the
ventricles created currents leading to ventricu-
lar ectopic activity, and consequent ventricu-
lar tachycardia or fibrillation. However, a
number of subsequent studies designed to test
this hypothesis have failed to establish the
arrhythmogenic importance of the oxygen
differential, and it is likely that this plays little
if any role.5

Undoubtedly much more important are the
profound metabolic changes occurring in and
around the infarcted myocardium. The death
of heart muscle results in a massive escape of
intracellular contents into the area of infar-
tion, especially potassium. Harris has shown
that this localized hyperkalemia results in a
partial depolarization and increased excitabil-
ity of myocardial cells. He also has postulated
that the elevated potassium produces an
injury potential that forms currents which
extend locally into the region containing the
hyperexcitable cells, the summation of these
effects combining to produce ectopic ventricu-
lar activity.6 In addition to potassium, magne-
sium moves out of infarcted cells, while
sodium and calcium move in.7 Myocardial
carcinosis also results in profound localized
acidosis as well as release of many other
substances into the area of infarction known
to have negative inotropic and chronotropic
effects, such as adenosine, certain amino acids,
and enzymes.8

How these many disturbances lead ultim-
ately to fatal ventricular fibrillation is also
still uncertain. However, the most commonly
held theory for the development of fibrillation,
either atrial or ventricular, is the “wavelet
hypothesis” proposed by Moe.9 According to
this theory, fibrillation does not occur in a
heart that repolarizes uniformly. However,
when there is nonuniformity of recovery of
either the atria or the ventricles fibrillation can
occur primarily by reentry extension of
circulating wavefronts from areas of myocar-
dium still depolarized to zones which have
recovered. This effect is further enhanced if
nonuniform recovery is combined with in-
homogeneous spread of depolarization cur-
rents through the heart. Han and colleagues
have identified many factors which contribute
to nonhomogenous ventricular repolarization
such as bradycardia, sympathetic stimulation,
and simple ectopic beats.10–12 Mandel et al.
have demonstrated that ischemia results di-
rectly in differences in refractory periods
between ischemic and nonischemic tissue.13
Immediately after experimental coronary oc-
cclusion, refractory periods in ischemic tissue
were shorter than in nonischemic tissue. After
24–72 hours, the refractory periods were
significantly longer in the ischemic zones.
Once again, this discrepancy in refractory
periods could set the stage for the develop-
ment of reentry tachycardias.

Active arrhythmias may also be initiated by
enhanced automaticity of the specialized cells
in the sinoatrial (SA) node, atrium, A-V
junction, and ventricular His-Purkinje system
which possess pacemaker properties, that is, the ability to depolarize spontaneously in diastole. A number of factors have been identified which increase the rate of spontaneous firing of these cells, including hypoxia, excessive stretch, mechanical trauma, hypercarbia, and acidosis. Furthermore, it has been shown that a weak, constantly applied electric current will increase the slope of phase-4 diastolic depolarization, thereby increasing automaticity. Such a current may exist in MI, as noted above.

Recently speculation has arisen that the elevation of free fatty acids noted in the plasma after MI might be related to the induction of ventricular arrhythmias, heart block, and sudden death. This elevation is presumably effected through lipolysis of adipose stores by endogenously secreted catecholamines. It is postulated that these effects of free fatty acids may be mediated in two ways: (1) an increase in oxygen requirement of tissues; and (2) a detergent effect on cell membranes and possibly on enzyme systems. Soloff found that the rapid infusion of long-chain saturated fatty acids resulted in arrhythmias, but not the slow infusion of the same fatty acids or the rapid infusion of monounsaturated and polyunsaturated fatty acids. The arrhythmias so produced were generally passive, consisting of A-V and intraventricular block and sinus arrest. It is still unclear what if any role fatty acids play in the genesis of active arrhythmias in MI.

Respiratory alkalosis or acidosis produced by abnormalities in ventilation may predispose to arrhythmias, which may be terminated simply by correcting the respiratory problem.

**Anatomic**

Arrhythmias may arise as a result of compromise of the circulation to those structures in the heart responsible for the origin and propagation of the cardiac impulse. It has been largely the outstanding studies of James which have elucidated these important relationships. Two arteries are of primary importance in this regard: (1) the artery to the sinus node; and (2) the artery to the A-V node.

The artery to the sinus node arises from the right coronary artery approximately 55% of the time, and from the proximal left circumflex coronary artery 45% of the time. Occlusion of the sinus-node artery can lead to ischemia, and even actual infarction, of this important structure. The result may be instability of the sinus mechanism, even sinus arrest. If bradycardia occurs, the vulnerability of the atrium to tachyarrhythmias is increased. In addition, the sinus-node artery is usually a major source of blood to the atrial myocardium. Thus, compromise of its flow may result in ischemia or infarction of the atrial musculature, with consequent electrical instability.

The artery to the A-V node arises from the right coronary artery in 90% of human hearts, and from the left circumflex coronary artery 10% of the time (the latter more frequently in men than in women). It is usually a distal branch of the major artery supplying the posterior wall of the heart so that heart block is considerably more frequent with inferoposterior infarction than with anterior infarction. Ischemia of this area may enhance automaticity of the A-V junction, leading to junctional tachycardias. Bradycardia from heart block produced by ischemia of the A-V node may also predispose to the development of tachyarrhythmias by contributing to nonuniform repolarization. James has postulated that still another reason for bradycardia in patients with inferoposterior infarction is ischemic stimulation of parasympathetic ganglia and nerve endings which lie in the lower posterior portion of the interatrial septum between the ostium of the coronary sinus and the posterior margin of the A-V node.

Still another potential factor related to tachyarrhythmias is pericarditis. The sinus node is a very superficial structure, and as such is vulnerable to the irritative effects of pericarditis. In addition, the atria may be irritated by pericarditis resulting in supraventricular arrhythmias.

It should be appreciated that ventricular arrhythmias bear no relationship to the
anatomy of the coronary circulation and may be the result of infarcts located anywhere.

Autonomic

Attention has already been directed to the enhanced vagal tone accompanying inferoposterior infarction. Vagotonia may also be produced simply by pain accompanying acute MI as well as by medications used to treat pain, especially morphine.

Several groups have demonstrated an increase in catecholamine secretion and excretion in patients with acute MI. Generally, such increases have paralleled severity of infarction. However, a few patients have been identified without significant pump failure with high catecholamine levels who have developed active arrhythmias. The high sympathetic tone may be due to anxiety. Although it seems possible that excessive sympathetic stimulation may occasionally contribute to arrhythmias, this area needs further definition. Nevertheless, adequate sedation and relief of anxiety in patients with MI are important.

Hemodynamic

In general it can be stated that the more severe an infarct in its hemodynamic consequences the higher are both the absolute frequency of arrhythmias and the incidence of life-threatening arrhythmias. Lown has emphasized the importance of left ventricular failure in the genesis of supraventricular tacharyrhythmias. He classifies these as "pump-failure" rhythms, resulting from acute left ventricular failure with left atrial hypertension and distension.

Hypotension accompanying acute MI may also be a factor in the development of arrhythmias by reducing coronary perfusion.

Iatrogenic

A small number of arrhythmias in MI are the result of administered drugs. Morphine with its vagotonic effect has already been mentioned in this regard. Antiarrhythmic agents, atropine, sympathomimetic amines, and numerous other agents also may contribute to cardiac arrhythmias. It has long been postulated that the infarcted heart is more sensitive to the toxic effects of digitalis than the noninfarcted heart. Experimental proof of this has recently been demonstrated by Morris et al., who produced myocardial infarction in pigs. They showed that the doses of digitalis glycosides necessary to produce toxic ventricular tachycardia were reduced by approximately one third after infarction. Furthermore, whereas gastrointestinal toxicity was evident before cardiac toxicity in the majority of animals prior to infarction, the converse was true after infarction.

Electrocardiographic Monitoring

At present, primary emphasis in coronary care is placed upon constant monitoring of the electrocardiogram. Marriott and Fogg have emphasized the value of a selective modified precordial electrode (MCL 1) which closely resembles lead V-I, the lead which they consider of greatest value in detecting disturbances of rhythm and conduction. In this system, the positive electrode is positioned in the usual V-I location (fourth right interspace at the right sternal edge), while the negative electrode is positioned just inferior to the outer quarter of the left clavicle. The ground electrode is placed inferior to the right clavicle. This lead system not only provides a more favorable electrocardiogram for monitoring, but removes the electrodes from the areas of primary auscultation on the precordium, permitting easier physical examination.

For the most part, coronary care units rely upon prominent visual display of the electrocardiograms on monitors located at the bedside and throughout the facility. These monitors are randomly viewed by the unit personnel, who are also alerted by alarms triggered by the heart rate deviating above or below preset limits. Some units have employed specially trained technicians to observe central banks of monitors. A useful addition to many monitoring systems has been the incorporation of a "memory loop," consisting of a magnetic tape that is constantly recording and erasing the patient's electrocardiogram. With an alarm, the eraser is lifted from the tape, preserving the electrocardiogram prior to the

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alarm violation. Although originally envisioned as mainly a teaching device, the memory system has proven to be highly useful in patient management by permitting recall and subsequent analysis of transient electrocardiographic events.

Although monitoring systems have been extremely reliable, most alarm violations are triggered by artifacts, such as loose electrodes and patient movements. Occasionally, artifacts have been recorded which strikingly resemble actual cardiac events and could lead to serious errors in therapy if they are not recognized as such (fig. 1).

There is no doubt that more automated means of arrhythmia detection are desirable. Although computer programs are being designed to detect and interpret arrhythmias, computer monitoring of arrhythmias is still in an early stage of development. Since one of the most important functions of the coronary care unit is detection and suppression of ventricular premature beats (VPBs) relatively simple computers have been designed for this purpose, and are commercially available.

These instruments monitor the R-R interval of successive beats, and detect shortening from the patient’s average R-R interval of more than 20%. The additional incorporation into the system of monitoring for QRS widening adds a further dimension to the usefulness of these systems. Hence, R-R interval and QRS duration can be monitored either separately or in combination, depending on the circumstances. For example, they might be used together for the detection of VPBs in a patient with sinus rhythm and normal QRS width. For the detection of any premature beat (atrial, junctional, or ventricular) or in the presence of bundle-branch block, only the R-R interval might be sensed, whereas monitoring of only the QRS duration might be of value in detecting VPBs in the presence of atrial fibrillation, where the R-R interval of successive beats varies greatly. These instruments can also be programmed to record the number of ventricular premature beats in a given period of time on a simple digital counter, or graphically as a trend recorder, which registers heart rate and frequency of ectopic beats on a slowly moving chart. Computers have also been used to develop histograms of R-R intervals in a variety of forms, showing the scatter of duration of R-R intervals around the usual R-R interval. Such displays have been very useful in assessing the effects of antiarrhythmic agents on an irritable heart.

Figure 1

Artifact caused by scratching near a precordial electrode recorded on the central-station electrocardiogram of a 59-year-old man with acute myocardial infarction. The artifact closely resembles ventricular flutter. However, careful observation reveals normal QRS complexes continuing through the artifact. The apparent blocked sinus beat at the end of the lower panel is caused by recycling of the memory loop.
Whether these somewhat expensive refinements actually contribute to improved survival in the coronary care unit is still undetermined.

One of the major problems related to computer monitoring of arrhythmias, in addition to the cost, is difficulty in accurately defining P waves. The use of an electrocardiographic signal obtained from transvenous electrodes positioned in the cardiac chambers may be very helpful in this regard.

Intracardiac electrodes have actually proven to be very useful in the interpretation of complex arrhythmias. Such electrodes can be directed into the right atrium at the bedside using electrocardiographic monitoring, and intracavitary electrocardiograms recorded from them usually clearly distinguish the electrical activity of the atrium from that of the ventricle (fig. 2). Although esophageal electrodes give similar information, transvenous electrodes provide a more stable electrocardiogram and are less bothersome to the patient.

**Atrial Tachyarrhythmias**

Atrial tachyarrhythmias are estimated to occur in approximately one fourth of patients with acute MI. The primary mechanisms responsible for the origin of atrial arrhythmias have been previously discussed. In brief, the two major factors appear to be (1) compromise of the circulation of the artery to the sinus node, with resultant ischemia and/or infarction of the sinus node or the portion of the atrium supplied by the vessel; and (2) “pump failure,” with atrial tachyarrhythmias being the result of left ventricular failure and acute left atrial hypertension and distention (fig. 3). Each of the atrial arrhythmias will be considered briefly.

**Sinus Tachycardia**

Strictly speaking, sinus tachycardia is not a true dysrhythmia, but brief reference is made to it because it is frequently included in reviews of arrhythmias complicating acute MI. About one third of patients with infarction demonstrate sinus tachycardia. Lown has classified sinus tachycardia as a “pump-failure” rhythm. Although it is true that, in the absence of A-V block, it is distinctly uncommon to have pump failure without sinus tachycardia, there are many other causes of...
sinus tachycardia in acute MI besides pump failure. These include fever, anxiety, pericarditis, and cardioaccelerator drugs. Sinus tachycardia per se is rarely a cause of any important cardiac dysfunction, but since heart rate is an important determinant of myocardial oxygen consumption, tachycardia of any type is undesirable in the setting of acute MI. Note has been made of the higher mortality in patients with sustained sinus tachycardia in MI, a reflection of the fact that when it is sustained it is often an indication of a serious complication of the infarct. However, the treatment of sinus tachycardia is almost never
directed at the rhythm itself, but rather at the underlying cause of the accelerated heart rate.

**Atrial Premature Beats**

Atrial premature beats have been detected in approximately 15–30% of continuously monitored patients with MI. They are not of consequence except that they are frequently the harbinger of atrial tachyarrhythmias, which are commonly initiated by an ectopic atrial beat (fig. 4). In general, atrial premature beats do not warrant suppressive therapy.

**Paroxysmal Atrial Tachycardia**

Paroxysmal atrial tachycardia (PAT) is uncommon, occurring in a reported 1–7.5% of patients with MI. In our coronary care unit the incidence is 2%. Episodes may be either short or sustained and are sometimes episodic. Occasionally, the arrhythmia is the result of digitalis toxicity. This should be particularly suspect if PAT in a patient on digitalis is also associated with block. In rare instances, the tachycardia may take the form of a multifocal atrial tachycardia.

When PAT does occur in MI, it is not uncommonly in the setting of pump failure, and its development may result in serious clinical deterioration. Therapy is dictated by the urgency of converting the arrhythmia. In a patient who remains stable during the tachycardia, the usual vagal maneuvers such as carotid sinus pressure and induced gagging may terminate the attack. A Valsalva maneuver is also sometimes effective. In a patient not previously receiving digitalis glycosides, digitalis is frequently successful, particularly if combined with a repeat of vagal maneuvers after administration of the drug. With MI our preference is for a short-acting preparation.

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**Figure 4**

Onset of atrial fibrillation initiated by an atrial premature beat (lower panel) in a 74-year-old man with an acute diaphragmatic infarct with sinus tachycardia and Wenckebach second-degree A-V block. The tracing also shows another occasional manifestation of an atrial tachyarrhythmia in the presence of high-grade A-V block, namely, the paradox of slowing of the ventricular rate coincident with onset of the rhythm disturbance. This is presumably the result of concealed conduction of fibrillary waves, increasing the refractoriness of the A-V junction, which is already slowed in its ability to conduct.

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administered intravenously, such as ouabain (0.25–0.5 mg), lanatoside C (0.6–1.2 mg), or digoxin (0.5–0.75 mg). We have terminated rare attacks of PAT in the coronary care unit with edrophonium bromide (Tensilon), in doses of 5–10 mg intravenously. However, this drug must be used with considerable care in MI, as its parasympathomimetic effects may produce hypotension or postconversion bradycardia.

Although sympathomimetic drugs such as phentolamine and metaraminol which produce transient hypertension are commonly used to terminate PAT, we have been reluctant to use them for PAT in MI because of the abrupt increase in afterload and cardiac work which they produce.

With sustained attacks refractory to either vagal or pharmacologic maneuvers, cardioversion may be necessary. Cardioversion should be applied urgently if PAT precipitates serious hypotension, failure, or ischemic pain.

In the rare instance of atrial tachycardia due to digitalis intoxication, potassium replacement and the use of antiarrhythmic agents, in particular diphenhydantoin in doses of 100–300 mg intravenously, may terminate the arrhythmia. Propranolol, 1–3 mg given slowly intravenously, may also be useful if there is no pump failure. Cardioversion is contraindicated if there is good reason to suspect that the arrhythmia is the result of digitalis.

**Atrial Flutter**

Atrial flutter is encountered in less than 5% of patients with acute MI. Most frequently, it appears with 2:1 A-V block and a ventricular rate generally in the vicinity of 150 beats/min. With atrial flutter, some semblance of an atrial-ventricular contraction sequence is maintained, but the rapid ventricular rate may result in hemodynamic deterioration especially if cardiac function is already compromised before the attack begins. Atrial flutter is occasionally difficult to recognize on the standard electrocardiogram, and the use of intracardiac electrodes have sometimes been extremely valuable in demonstrating the arrhythmia (see fig. 2).

Atrial flutter may be a very stubborn arrhythmia to treat pharmacologically. It is often difficult to achieve adequate control of the ventricular rate with even large quantities of digitalis. Our approach to the therapy of atrial flutter is generally directed more at terminating the arrhythmia than at controlling the ventricular rate. Assuming that the patient is otherwise stable and not previously receiving digitalis, about half of the usual digitalizing dose of a short-acting glycoside is administered, for example, 0.25 mg of ouabain or 0.5 mg of digoxin. In approximately one fifth of individuals so treated, digitalis alone will terminate the attack. However, if the attack persists more than an hour after digitalis administration, or as initial therapy if the flutter is producing serious compromise of cardiac function, DC cardioversion using a synchronized shock is undertaken. Atrial flutter is extremely responsive to such therapy and can almost invariably be terminated, often with relatively small shocks in the range of 25 to 50 joules. Cardioversion can be performed safely with the small amounts of digitalis recommended above. On rare occasions we have terminated atrial flutter by rapid stimulation of the atrium by way of a transvenous pacing wire. This may convert flutter either to sinus rhythm or atrial fibrillation. In the latter case the ventricular rate can be more easily controlled with digitalis if it does not revert spontaneously to sinus rhythm.

If atrial flutter is recurrent and cannot be suppressed, it may become necessary to control the ventricular rate by pushing digitalis. In the absence of left ventricular failure, the addition of propranolol to digitalis may also help control the ventricular rate and may even convert the arrhythmia. Propranolol is usually given in doses of 1–3 mg intravenously every 3–4 hours, or 10–30 mg orally every 4–6 hours.

**Atrial Fibrillation**

This arrhythmia is about twice as common as atrial flutter in the setting of MI and is detected in approximately 10% of patients with MI. Although studies of the hemodynamic
effects of atrial fibrillation are lacking in patients with acute MI, loss of the atrial kick together with the accelerated ventricular rate has generally been associated with a substantial reduction in cardiac output, particularly when there is already diminished left ventricular performance.\textsuperscript{55}

In contrast to atrial flutter the very rapid atrial rate in atrial fibrillation often results in more concealed conduction into the A-V node and greater A-V-nodal refractoriness. Hence, the ventricular rate in atrial fibrillation can usually be controlled more adequately by administration of digitalis glycosides. Indeed the ventricular rate is a useful guide to the adequacy of digitalization. Occasionally, the addition of propranolol to digitalis in the absence of ventricular failure may aid in slowing the ventricular rate. Since this arrhythmia is often intermittent in MI and commonly reverts spontaneously, cardioversion is usually not undertaken unless atrial fibrillation either occurs in an individual who is already in serious pump failure, or unless the dysrhythmia is the cause of pump failure.

On occasion, in a patient who is stable, an attempt may be made to convert atrial fibrillation with quinidine. However, it is important not to endanger the patient by the administration of large quantities of quinidine, as the arrhythmia is usually easy to convert electrically if it does not convert spontaneously. We usually set 1.6 gm of quinidine as a maximum total dose to be given in any 24-hour period.

**Suppression of Atrial Arrhythmias**

As indicated, atrial tachyarrhythmias in MI are frequently recurrent (fig. 5). They are usually a phenomenon of the early days of MI and rarely persist after the first week or 10 days. Indeed, it is very rare that an atrial tachyarrhythmia which had its inception during acute MI is present when the patient leaves the hospital. It is generally our policy to place patients who have experienced a supraventricular tachycardia on quinidine, 0.8–1.2 g daily in four divided doses. Alternatively,

**Figure 5**

Lead VI. Recurrent short bursts of atrial fibrillation occurring in a 64-year-old man with acute anterior myocardial infarction. Aberrant ventricular conduction is also noted, especially in the upper panel.

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procainamide in doses of 250–500 mg every 3–4 hours may be effective in patients who are sensitive to quinidine. Optimally, both of these drugs should be administered by monitoring blood levels. For quinidine, levels of 3–6 mg/liter are usually adequate, whereas 4–8 mg/liter is considered a good therapeutic range for procainamide. Despite such therapy, the arrhythmias may continue to recur until the arrhythmogenic factors of acute MI have waned. When the recurrent arrhythmias reflect pump failure, therapy directed at improving myocardial function helps abolish them. No good guidelines have been developed concerning the discontinuation of antarrhythmic agents. It is generally our policy to stop them shortly before the patient is discharged from the hospital if the rhythm is stable. However, one of the digitals glycosides is usually given in maintenance doses for several weeks after the infarct.

**Junctional (Nodal) Tachyarrhythmias**

Junctional dysrythmias in acute myocardial infarction are of two types: (1) accelerated junctional rhythm and (2) paroxysmal junctional tachycardia (PJT). The first type consists of an acceleration of a junctional pacemaker focus to rates of between 60 and 100 beats/min. This benign rhythm, occurring usually as an escape rhythm in association with sinus bradycardia, is analagous to accelerated idioventricular rhythm, and is considered further in that section.

Paroxysmal junctional tachycardias, usually manifesting rates of between 120 to 180 beats/min, are seen on rare occasions in MI. Their significance is analagous to that of atrial arrhythmias, in that they may occur spontaneously or as a consequence of pump failure and, rarely, are also due to digitalis toxicity. Junctional tachycardias can be diagnosed with virtual certainty in the presence of QRS complexes of normal duration and either constant retrograde atrial capture or A-V dissociation (fig. 6). In the presence of bundle-branch block, difficulty may arise in distinguishing PJT from ventricular tachycardia. The consequences of junctional tachycardia are analagous to those of PAT, and therapy is similar, although vagotonic maneuvers are less likely to be successful with PJT as opposed to PAT. On occasion, profound clinical deterioration caused by the arrhythmia dictates immediate termination by cardioversion unless it is a result of digitalis toxicity.

**Ventricular Tachyarrhythmias**

It is primarily through the elimination of sudden fatal ventricular arrhythmias that coronary care units have achieved the greatest impact in reducing mortality in acute myocardial infarction. Although reference has been made previously to some factors contributing to ventricular arrhythmias in MI, further comments about their genesis are in order.

Wiggers and Wegria first demonstrated the existence of a so-called "vulnerable phase" of the heart—a point in the cardiac cycle at which a stimulus of proper magnitude produces ventricular fibrillation. This corresponds generally to the ascending limb and apex of the T wave of the electrocardiogram. Clinically, Smirk and Palmer first pointed out the hazard of spontaneously occurring VPBs

![Figure 6](https://circ.ahajournals.org/)

**Figure 6**

*Lead II showing a dissociated paroxysmal junctional tachycardia in a 58-year-old man with anterior myocardial infarction. The sinus rate is 125 beats/min and the ventricular rate is 140. The arrhythmia terminated spontaneously several minutes later.*

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which fell in this phase, a manifestation to which they gave the name “R on T” phenomenon. An example of this is shown in figure 7. Lown et al. have recently summarized factors important in the genesis of ventricular irritability in MI. They point out that it requires extremely high energy levels (in the order of 40,000 µjoules) to produce ventricular fibrillation by stimulating the heart during the vulnerable phase in normal animals. In contrast, only 1 µjoule applied to the endocardium is necessary to produce a ventricular premature beat. They postulate four possible mechanisms, based upon their work and that of others, as to why a ventricular premature beat can precipitate ventricular fibrillation in MI. First, in the zone of the infarction, there is a reduction of between 50 and 75% in the threshold for ventricular fibrillation produced by vulnerable-phase stimulation. Second, in the presence of ischemia, there is greater temporal dispersion of recovery of the ventricles and a reduction of ventricular-fibrillation threshold within the spatial perimeter of a premature beat, a phenomenon we have previously discussed. Third, a sequence of closely occurring premature beats may progressively lower the vulnerable-period threshold. Fourth, ventricular tachycardia precipitated during the vulnerable phase in MI frequently predisposes to ventricular fibrillation. Lown has called this rhythm VT_v (ventricular tachycardia of the vulnerable phase). This rhythm has been shown to occur spontaneously after coronary artery ligation in animals, and may appear for periods of up to 1 week after occlusion. About 25% of the time the rhythm terminates.

![Figure 7](image-url)

**Figure 7**

Onset of ventricular fibrillation following a ventricular premature beat falling in the vulnerable phase in a 49-year-old woman with an acute diaphragmatic myocardial infarction. This arrhythmia occurred an hour after onset of symptoms and was not preceded by any other VPBs. (Bottom panel) The ventricular fibrillation is terminated by an external shock of 400 w-sec.
spontaneously, but in the remaining 75% it deteriorates into ventricular fibrillation. The same rhythm has been recognized clinically in man as an important prefibrillatory rhythm in MI (fig. 8).

Although much emphasis has been placed on VPBs as a precursor to ventricular tachycardia or fibrillation, these serious and potentially fatal arrhythmias undoubtedly can arise without any premonitory ectopic activity. Lawrie et al. have shown that ventricular fibrillation is much more likely to occur without warning in the first 48 hours after MI. When it developed after 48 hours, premonitory arrhythmias were usually seen.40

Clearly, the incidence of potentially fatal ventricular arrhythmias decreases markedly in the initial week after acute MI. Indeed, Pantridge and Geddes have estimated that the dangers of developing ventricular fibrillation are some 15 times greater in the first 4 hours after onset of acute MI than they are between the fourth and twelfth hours, and 25 times greater than the risk between the twelfth and twenty-fourth hours.41 Thus the importance of administering coronary care to patients at the earliest point following the onset of symptoms of acute MI cannot be overemphasized.

**Ventricular Premature Beats**

Ventricular premature beats (VPBs) occur in at least 80% of constantly monitored patients during acute MI.25, 40 The hazards of VPBs, particularly when they occur in the vulnerable phase of the cardiac cycle, have already been emphasized. Lown has called attention to the importance of the ratio of the time interval between the Q wave of the normally conducted beat and the R wave of the VPB compared to the Q-T interval of the normally conducted beat (Q-R'/Q-T ratio).42 He has determined that VPBs which occur with a Q-R'/Q-T ratio of 0.60–0.85 sec are likely to precipitate ventricular tachycardia or ventricular fibrillation (fig. 9). However, quite clearly repetitive ventricular beating may be
initiated by ectopic beats outside the vulnerable phase (fig. 10).

The generally accepted criteria for the suppression of VPBs in the setting of MI are as follows: (1) when they occur with an absolute frequency of greater than five beats/min; (2) when they fall in the vulnerable phase of the cardiac cycle; (3) when they are multifocal in origin; and (4) when they are coupled or occur in salvos. On the other hand, discretion should be used in the application of these guidelines. In a patient who has had ventricular tachycardia or ventricular fibrillation at the onset of MI, it is advisable to suppress all ventricular irritability if possible for a period of 48 hours. Conversely, occasional patients have a degree of ventricular irritability that is refractory to virtually all antiarrhythmic agents, used singly or in combination. If ventricular tachycardia or ventricular fibrillation does not occur, it may be preferable to accept a certain degree of ventricular ectopy rather than precipitate serious toxicity from inordinately large doses of antiarrhythmic agents.

For the suppression of ventricular ectopic beats, lidocaine is generally the most effective and least toxic agent, and in ordinary therapeutic doses causes minimal depression.
of ventricular contractility. It can be administered rapidly intravenously in a dose of 1 mg/kg of body weight, or between 50 and 100 mg in the average adult. Kimball and Killip suggest an additional bolus of 1 mg/kg every 3–5 min until either the arrhythmia is abolished or a total dose of 5 mg/kg has been administered in a period of 15–20 min. Occasionally, side reactions necessitate stopping short of this total amount. For long-term suppression, lidocaine can be administered as a continuous intravenous drip. Gianelly et al. showed that doses above 20 µg/kg/min are usually necessary to achieve an antiarrhythmic effect, whereas doses above 50 µg/kg/min are prone to be associated with toxic side reactions. These values correspond to roughly 1–4 mg/min in an average adult.

Lidocaine has proved to be safe and effective. Side reactions take the form of central nervous system depression or excitation, ranging from obtundation, acute agitation, tinnitus, and visual scintillations to frank seizures. Occasional hypotension and A-V block may occur. However, in general, lidocaine does not alter QRS complexes or P-R or Q-T intervals.

Lidocaine is metabolized rapidly in the liver, its effect lasting for only 15–20 min, but should be used cautiously in the presence of hepatic dysfunction. The short duration of action of lidocaine is advantageous in that any side reactions are quickly dissipated. On the other hand, the short action has the disadvantage that arrhythmias may not be suppressed for long if there is a tendency toward recurrence. The use of intramuscular lidocaine in doses in the range of 300 mg is currently under investigation. This amount has been reported to produce therapeutic levels of lidocaine for periods up to 1 hour.

For patients sensitive to lidocaine or exhibiting persistent ventricular irritability as lidocaine is tapered, one of the longer-lasting antiarrhythmic agents may be used. Our preference is for procainamide, given as a loading dose of 750 mg to 1 g followed by 250–500 mg every 3–4 hours. Koch-Weser and Klein have suggested total doses of 50 mg/kg/day divided into three hourly doses. Generally, the drug is administered hourly but can be given intramuscularly if necessary. Although procainamide is associated with a high incidence of side reactions when administered over a long period of time, side effects with short-term administration are negligible. However, high blood levels may be associated with hypotension and depressed myocardial contractility, especially if some degree of pump failure is already present.

Quinidine in doses of 200–300 mg every 4–6 hours may also be useful in suppressing ventricular irritability. There is one effect of quinidine which must be kept in mind when it is used. Of all of the antiarrhythmic agents, quinidine causes the greatest prolongation of the Q-T interval, and this may pose a potential hazard to patients with ventricular irritability if the irritability is not suppressed. As the Q-T interval lengthens, the T wave—ergo, the vulnerable phase—may be moved into the VPBs, occasionally triggering ventricular tachyarrhythmias. This is very likely the mechanism for the occasional paradoxical increase in ventricular irritability associated with quinidine. The doses of both procainamide and quinidine should be reduced in the presence of hepatic or renal dysfunction.

Diphenylhydantoin has been widely used in the therapy of ventricular irritability, but our experience with it has been rather disappointing, coinciding with that recently reported by Stone et al. However, it does appear to have specific usefulness in arrhythmias due to digitalis toxicity. When used to abolish ventricular ectopic beats or ventricular tachycardia, it is usually given intravenously in doses of up to 500 mg at the rate of 100 mg every 5 min. The intravenous preparation is exceedingly caustic and should be introduced through a rapidly running intravenous solution. For continued suppression, diphenylhydantoin is administered orally in doses of 100 mg three or four times daily. Plasma levels of 10–18 mg/liter are usually sufficient to control arrhythmias, and higher levels may be associated with toxic reactions. Side effects consist
primarily of central nervous system manifestations, and include lethargy, vertigo, dysarthria, nausea, and nystagmus. Rare instances of cardiac arrest precipitated by diphenylhydantoin have been reported, and like any other antiarrhythmic agent it should be used with extra caution in MI.

Beta-adrenergic-receptor blocking agents, of which propranolol is the only one presently available for clinical use in this country, have also been used to treat ventricular irritability, but have no particular advantage over other antiarrhythmic drugs. Indeed, of all antiarrhythmic agents, propranolol appears to depress myocardial contractility the most relative to its antiarrhythmic action. In addition, beta-adrenergic blockers also slow the sinus rate more than any other agent, an effect that can sometimes be deleterious.

Recently, Bacaner has reported the efficacy of bretylium tosylate in treating ventricular irritability. Day and Bacaner, using this drug in MI, have recommended a loading dose of 600 mg intramuscularly, with incremental doses of 200 mg every 1–2 hours until either the arrhythmia is controlled or a total of 2 g has been given. Maintenance doses of 5 mg/kg every 6–8 hours are suggested. Terry et al. have also reported favorably on its efficacy in treating refractory ventricular arrhythmias in MI. The most important side reaction of bretylium is hypotension, especially postural, but bradycardia, nausea, and vomiting also arise sometimes. Although it can be given orally its absorption is very unpredictable. At present bretylium is still considered experimental and its precise role in the armamentarium of antiarrhythmic drugs remains to be determined.

**Ventricular Tachycardia**

Ventricular tachycardia is defined as three or more successive beats of ventricular origin occurring at a rate of greater than 120 beats/min. Of all the cardiac tachyarrhythmias, ventricular tachycardia produces the most serious compromise of cardiac performance. The incidence in acute MI is approximately 10%. The very high incidences of 25–30% reported in early studies of arrhythmias in MI probably included cases of accelerated idioventricular rhythm.

Ventricular tachycardia is of two types: extrasystolic and parasystolic. Extrasystolic ventricular tachycardia is initiated by a VPB and is by far the most common type of ventricular tachycardia encountered in MI.

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**Figure 11**

Ventricular tachycardia with intermittent exit block in an elderly patient with an acute myocardial infarction. The mechanism of the arrhythmia is shown in the laddergram. The sinus rate is 100 beats/min and there is a dissociated slightly irregular ventricular tachycardia at a rate of about 150 beats/min. The occasional blocked ventricular beats are diagrammed.
Ventricular tachycardia with A-V dissociation revealed by an intraatrial electrocardiogram. (Upper panel) Lead AVL, shows broadened QRS complexes at 145 beats/min. P waves are not clearly visible. (Lower panel) Right atrial electrocardiogram shows large P waves (downward deflections P) at a rate of 100 beats/min with dissociated QRS complexes (upward deflections R) at a rate of 145 beats/min. (Reprinted from Brest,56 by permission.)

Figure 12

(see figs. 9, 10). When it occurs paroxysmally, there is usually a fixed interval coupling the initiating VPB to the normally conducted beat. Parasystolic ventricular tachycardia is caused by a “protected” ectopic focus. This focus discharges continuously, activating the ventricles whenever they are not made refractory by a conducted supraventricular impulse. At the same time, the ectopic focus is not depolarized by these supraventricular impulses, thus maintaining its own rhythmicity over long periods of time. The interval between the ventricular complexes can be calculated as a multiple of the basic cycle length. Parasystolic rhythms are rare in MI and generally are rather benign.54 On occasion, ventricular tachycardia in MI is associated with an exit block from the ectopic focus (fig. 11).

Electrocardiographically, the two classical hallmarks of ventricular tachycardia include (1) evidence of A-V dissociation with widened QRS complexes and (2) the presence of capture or fusion beats. Although retrograde atrial activation may be present in ventricular tachycardia and is frequently seen in ventricular tachycardia that is artificially induced by rapid stimulation of the endocardium with pacing electrodes,55 its frequency in spontaneously occurring ventricular tachycardia in patients with heart disease is not known. It is probably uncommon. If a careful search for P waves in the routine electrocardiogram is unrevealing, an intraatrial electrocardiogram may be extremely useful (fig. 12).56

Not uncommonly, the diagnosis of ventricular tachycardia is more evident at the bedside than on the electrocardiogram. Physical examination may reveal evidence of A-V dissociation. These include (1) intermittent cannon waves in the jugular venous pulses; (2) varying intensity of the first heart sound; (3) variations in systemic peak blood pressure; and (4) extra heart sounds consisting of atrial, ventricular, and summation gallop sounds. All of these signs are the result of random contraction of the atria in relation to the ventricles.56

Therapy of ventricular tachycardia is dictated largely by the patient’s status consequent to the tachycardia. If the arrhythmia is or congestive failure, then intravenous lidocaine can be given as described above. However, if ventricular tachycardia results in serious clinical deterioration or if lidocaine is unsuccessful, cardioversion should be undertaken. We use a synchronized DC shock, with transient amnesia induced with either methohexitol (Brevital) or diazepam (Valium) well tolerated without significant hypotension administered intravenously. Rarely a vigorous
thump on the chest will correct either ventricular tachycardia or ventricular fibrillation.\textsuperscript{57}

**Ventricular Fibrillation**

Ventricular fibrillation can be regarded as either a primary or secondary arrhythmia. Primary ventricular fibrillation is that which occurs suddenly and unexpectedly in patients with little or no pump failure. Conversely, secondary ventricular fibrillation represents the end stage of progressive left ventricular deterioration, usually with cardiogenic shock and/or congestive heart failure. Resuscitation from primary ventricular fibrillation is highly successful, whereas the results are extremely poor with secondary ventricular fibrillation. It is not within the scope of this review to discuss resuscitation from ventricular fibrillation, but it is important to emphasize that the earlier the primary ventricular fibrillation is treated by external countershock the greater the likelihood of successful resuscitation.

**Therapy of Refractory Ventricular Irritability**

In rare instances, ventricular irritability may be refractory to all pharmacologic measures, using combinations of antiarrhythmic agents in maximum amounts. In such cases, rapid cardiac pacing may suppress the irritability.\textsuperscript{58} Pacing electrodes can be positioned either in the right atrium when A-V conduction is intact, or the right ventricle if A-V block is present. In general it can be stated that the slower the basic heart rate before pacing the more likely it is that rapid pacing will be successful in overdriving the arrhythmia. Usually, such pacing is used only as a temporary measure, but rarely we have successfully instituted long-term rapid pacing to suppress ventricular ectopicity after MI.\textsuperscript{59}

The advent of cardiac surgery has opened up further possibilities for aggressive therapy of refractory irritability. There have been recent reports of curing refractory ventricular irritability by resecting ventricular aneurysms, though usually these cases have undergone surgery many months or years after infarction.\textsuperscript{60,61} We have recently resected a ventricular aneurysm 25 days after an acute MI in a 38-year-old man with refractory recurrent ventricular fibrillation, with abolition of the irritability after surgery.\textsuperscript{*}\textsuperscript{†} This area needs further investigation, including the reporting of surgical failures as well as successes!

The role of acute revascularization of the myocardium in the therapy of refractory ventricular irritability after MI also is virtually unexplored. We have performed revascularization using saphenous vein bypass grafts on a 51-year-old man with drug-resistant ventricular irritability which had persisted for approximately 2 months after an attack of ventricular fibrillation following a subendocardial infarct.\textsuperscript{†} Though ventricular irritability was transiently worse immediately postoperatively, it has since disappeared.

At present, since the role of surgery in treating ventricular irritability is ill-defined and the risks are high and the results uncertain, it should be considered a last therapeutic resort. It is possible that further experience may modify this stance in the future.

**Prophylactic Antiarrhythmic Therapy**

The effects of several antiarrhythmic agents administered prophylactically to prevent ventricular tachyarrhythmias in MI have been investigated. Results of these studies using the same drugs are sometimes conflicting, but discrepancies are often due to differences in doses. When used in adequate amounts, quinidine,\textsuperscript{62} procainamide,\textsuperscript{63} lidocaine,\textsuperscript{64} and perhaps bretylium tosylate\textsuperscript{65} have all been shown to reduce significantly the incidence of ventricular ectopic beats and serious ventricular arrhythmias.

It is of interest that in most studies where prophylactic therapy has been shown to reduce arrhythmic events, mortality was usually not significantly different in treated or control groups. The reason obviously is that ventricular irritability was promptly suppressed when it arose in untreated patients. Thus it is difficult to take a stand on whether or not routine prophylaxis should be used in

\textsuperscript{*}The patient's physician was Dr. Peter Yurchak.

\textsuperscript{†}The surgery was performed by Dr. Eldred Mundth.
MI. In coronary care units with adequate monitoring and well-trained personnel, it is probably unnecessary. In units less-well equipped or staffed, routine prophylaxis would seem reasonable if used carefully. Routine prophylaxis should not be administered to patients with hypotension, cardiogenic shock, advanced congestive heart failure, or second- or third-degree A-V block. If any prophylactic drug is used, lidocaine given

Figure 13

Accelerated idioventricular rhythm complicating acute inferior myocardial infarction. Continuous lead II. The first two beats in the upper strip are those of normal sinus rhythm at 75 beats/min. In the two panels shown, there are five short runs of accelerated idioventricular rhythm at a rate of 88 beats/min. The third and twelfth beats in the upper panel are fusion beats, intermediate in configuration between normally conducted and idioventricular beats. (Reprinted from Brest, 66 by permission.)

Figure 14

(A) An accelerated idioventricular rhythm in a patient with an acute inferior myocardial infarction with atrial fibrillation and high-grade A-V block (lead I). The idioventricular rhythm is interrupted by a ventricular premature beat (third QRS complex). (B) A short time later there is a short burst of ventricular tachycardia at a rate of 130 beats/min, beginning with a VPB.
intravenously at doses of 2–3 mg/min for the first 48 hours after MI is probably the simplest and safest.

For almost a decade, the value of so-called "polarizing solution," first introduced by Sodi-Pallares and his associates, has been debated. This is a solution consisting of glucose, insulin, and potassium designed to prevent potassium loss from injured myocardial cells. The argument over its efficacy in preventing arrhythmias in MI is still not settled, but the general sentiment in this country based on several negative reports is against its routine use in MI.

Accurated Idioventricular Rhythm

This generally benign rhythm has been recognized with increasing frequency in MI. Rothfeld et al. report an incidence of 23% in a series of 300 consecutively monitored patients with infarction. This rhythm occurs as an escape rhythm, usually when sinus bradycardia is combined with an accelerated ventricular (or junctional) focus, the latter presumably caused by enhanced automaticity as a consequence of the infarct. When the sinus slows, the accelerated rhythm takes over as the dominant cardiac pacemaker, whereas the sinus again captures the ventricles when it speeds up (fig. 13). Rarely, the rhythm is seen in atrial fibrillation when there is a slow ventricular response (fig. 14).

Rothfeld et al. have described the features of this arrhythmia. Because bradycardia is more common in inferoposterior infarction, accelerated idioventricular rhythm is more commonly associated with infarcts in this location. It is frequently seen during sleep, when the sinus rate slows even more. It begins as an escape rhythm, and fusion beats are common. The rate is usually between 60 and 100 beats/min, occurring usually in brief intermittent salvos of from 4 to 30 beats. It does not adversely affect prognosis, usually disappears spontaneously in 48 hours, and generally does not require treatment.

On the other hand, this arrhythmia does not always occur in pure form. We have seen instances of idioventricular rhythm complicated by premature beats, sometimes apparently from the same focus, sometimes from another focus, degenerating into ventricular tachycardia (fig. 14). In such instances, the rhythm should be suppressed. This can often be accomplished simply by accelerating the slow sinus rate with a cardioaccelerator drug such as atropine, or by pacing at a more rapid rate, although the addition of an antiarrhythmic agent may also be necessary.

The Future Challenges

Although much has been accomplished in the past decade in focusing upon the prevention of death from arrhythmia in MI, much remains to be done. The basic factors responsible for arrhythmogenesis need further clarification. Better automated systems of monitoring need to be developed, perhaps using simple computers which reliably detect and warn of at least potentially serious arrhythmias. Better antiarrhythmic agents must be found, and the role of cardiac surgery in the therapy of refractory ventricular irritability must be established. A great challenge still unmet is how to prevent those 50% of sudden arrhythmic deaths from coronary disease which occur before patients even arrive at the hospital. Finally, the ultimate treatment of any disease lies in its prevention, and the secrets of how to prevent the development of coronary arteriosclerosis must be solved.

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