Ventricular arrhythmias, ranging from premature ventricular contractions (PVCs) to ventricular fibrillation (VF), occur frequently in patients after acute myocardial infarction (AMI) [1]. The prognostic significance of these arrhythmias as well as their optimal management is largely dependent on several factors (Table 18.1): time from AMI, type of ventricular arrhythmia, and extent of myocardial dysfunction. In this chapter, we discuss specific ventricular arrhythmias observed in patients after MI and their significance, the impact of current medical therapy on the incidence of sudden cardiac death, and the role of antiarrhythmic drugs and implantable cardioverter-defibrillators in the management of post-MI patients.

Specific Arrhythmias

Premature Ventricular Contractions

Premature ventricular contractions are seen in most patients after AMI. In the thrombolytic era, predischarge 24-hour ambulatory electrocardiographic recordings demonstrate some degree of ventricular ectopy in up to 64% of patients [1]. When observed >48 hours after an AMI, frequent PVCs (>10/h) appear to be associated with adverse prognosis [1, 2]. In the GISSI-2 trial, PVCs were an independent predictor of overall and sudden mortality within the first 6 months after AMI, with a relative risk (RR) of 1.62 and 2.24, respectively [1].

Accelerated Idioventricular Rhythm

Accelerated idioventricular rhythm (AIVR) is characterized by a wide QRS complex, with a regular rate higher than the atrial rate and lower than 100 beats per minute (bpm) [3]; this can represent an escape rhythm or an accelerated ectopic focus from the ventricle. Although AIVR is frequently found in the first 12 hours of AMI, it does not appear to be a risk factor for the development of VF [4]. In patients who receive thrombolitics, AIVR has been shown to be a
marker for both myocardial necrosis and reperfusion of the infarct vessel. In fact, the QRS morphology of the AIVR may be useful for the noninvasive identification of the infarct vessel [5].

**Nonsustained Ventricular Tachycardia**

Nonsustained ventricular tachycardia (NSVT) is defined as $\geq 3$ consecutive ventricular ectopic beats at a rate of $>100$ bpm lasting $<30$ seconds and not accompanied by hemodynamic collapse [6]. Its incidence after AMI ranges from 1% to 7% [7, 8]. In the early postinfarct period, electrical instability caused by abnormal automaticity within surviving Purkinje fibers, triggered activity arising from Purkinje fibers, or reentry involving either the Purkinje fibers or within the ischemic myocardium is the likely mechanism [9]; NSVT that occurs later is likely due to reentry involving a fixed substrate [6]. The prognostic significance of NSVT depends on its time of occurrence with respect to the AMI. Likely benign in the early peri-infarct period, its association with early mortality increases as time progresses, becoming significant at 13 hours after infarction and plateauing at approximately 24 hours (relative risk 7.5) [9].

**Sustained Ventricular Tachycardia**

Sustained ventricular tachycardia (VT) is defined as a regular, wide-complex tachycardia of ventricular origin lasting $\geq 30$ seconds or accompanied by

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**Table 18.1.** Ventricular arrhythmias in AMI.

<table>
<thead>
<tr>
<th>Time</th>
<th>AMI 24 hrs</th>
<th>48 hrs</th>
<th>40 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathophysiology</td>
<td>↓ATP, local electrolyte imbalance $\rightarrow$ abnormal automaticity, triggered activity (transient) $\rightarrow$ abnormal gap junctions in border zone of healing infarct and viable myocardium $\rightarrow$ slow, inhomogeneous conduction $\rightarrow$ reentry (substrate starts to form within 2 weeks after MI and remains indefinitely)</td>
<td>PVCs, AIVR, VF</td>
<td>PVCs, NSVT, monomorphic VT</td>
</tr>
<tr>
<td>Dominant ventricular arrhythmia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Therapy</td>
<td>Revascularization, beta-blockers, ACE-inhibitors, statins</td>
<td>Rule out/ treat ischemia</td>
<td>Beta-blockers, ACE inhibitors, statins, ICDs (if sustained arrhythmia)</td>
</tr>
</tbody>
</table>