Critical Care Aspects in the Management of Patients with Acute Coronary Syndromes

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Coronary heart disease is an especially prevalent disease in the United States with an estimated more than 13 million persons affected. Worldwide prevalence of coronary heart disease has been increasing at an alarming rate as western diets and industrialization reaches developing countries with large populations (eg, India and China). Of individuals with coronary heart disease, approximately half eventually have acute myocardial infarctions (AMIs), whereas the other half suffer from angina \cite{1}. There are almost 1.7 million distinct acute coronary syndrome (ACS) discharges from American hospitals annually including approximately 500,000 ST-elevation myocardial infarctions (STEMI) \cite{2}. Additionally, coronary heart disease is the leading cause of death in the United States among both men and women \cite{3}.

The critical care aspects of treatment of the ACS patient are the focus of this article. The American Heart Association (AHA) and American College of Cardiology (ACC) maintain comprehensive evidence-based guidelines for the diagnosis and treatment of ACS. Much of the emphasis of these guidelines is related to early recognition, diagnosis, and treatment of ACS based on principles of prevention of major complications caused by ACS and improvements in survival. Pharmacologic and reperfusion therapies designed to improve myocardial blood flow are the primary focus of the sections

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dedicated to therapeutics. In contrast to these broad guidelines, this article discusses specific situations that may arise during the care of the patient experiencing an ACS that may result in patient instability and require critical care management.

Contained in this article are discussions of common complicating features of ACS with an emphasis on those occurring during AMI. Hemodynamic and electrophysiologic problems are the most common and serious immediate complications faced by providers treating ACS patients. Reperfusion therapy is the widespread standard for the treatment of STEMI. Complications specifically related to fibrinolytic therapy, including major hemorrhages and failure to reperfuse, are also included in this discussion.

**Cardiogenic shock**

**Background**

Cardiogenic shock represents a state of end-organ hypoperfusion and dysfunction secondary to low cardiac output. With the development of coronary care units, cardiogenic shock has surpassed arrhythmias as the leading cause of in-hospital mortality after AMI [4,5]. The incidence of cardiogenic shock complicating MI has remained relatively constant despite marked improvements in revascularization over the past few decades [6–8].

**Epidemiology**

Cardiogenic shock complicates approximately 7% of ACS cases [6,8–10]. Although it is commonly perceived that only a small subset of AMI patients have an initial presentation of cardiogenic shock, the incidence has been reported to be as high as 28% [8,9,11]. Most ACS patients who develop cardiogenic shock during their hospitalizations are experiencing a STEMI. A small percentage patients with non-STEMI also suffer from cardiogenic shock as described in the GUSTO II-B and PURSUIT trials (2.5% and 2.9%, respectively) [7,12]. The onset of cardiogenic shock has been shown to occur later in the course of the AMI in non-STEMI versus STEMI [7,12,13]. Fifty percent of the overall mortality in cardiogenic shock is within the first 10 hours of symptom onset [14,15]. This fact demonstrates the importance of early recognition and intervention by the treating physician. The etiologies of cardiogenic shock from the SHould we emergently revascularize Occluded Coronaries for cardiogenic shockK (SHOCK) Registry are represented in Fig. 1 and are discussed in more detail next.

**Mechanism**

**Left ventricle failure**

Severe left ventricle (LV) dysfunction is responsible for nearly 80% of cases of cardiogenic shock after MI [16–18]. LV dysfunction occurs when greater than 40% of the LV myocardium is affected [19,20]. It is more
commonly associated with large anterior MIs, although it can be seen in other distributions or secondary to recurrent infarction or extension of previous damage [19,21]. Hochman and colleagues [18] found that patients were more likely to develop severe LV dysfunction with cardiogenic shock if they had a prior MI (40.1% versus 29.5%). In the SHOCK trial, 60% of patients with cardiogenic shock were found to have triple-vessel disease and 20% had left main disease as the culprit lesion; the group with left main disease had the highest in-hospital mortality [21].

The myocardial pathology involved in ischemia and infarction helps to explain why patients with multivessel disease or previous infarction are at increased risk for dysfunction and failure. Myocytes adjacent to the infarct are susceptible to expanding ischemia because of decreased coronary perfusion pressure, increased oxygen demand, or propagation of thrombus [22]. Sites remote to the initial infarct may develop systolic dysfunction because of impaired autoregulation, limited vasodilatation, hypotension, or metabolic derangement resulting from the original insult [23]. Myocardial stunning and hibernation are both additional components of nonfunctional myocardium during cardiogenic shock. Myocardial stunning is dysfunction that occurs postinfarction and is eventually reversible with restoration of normal coronary flow [24,25]. The severity of stunning is dependent on the degree of preceding ischemia and is thought to occur because of oxidative stress, disruption of calcium homeostasis, and decreased myocardial calcium responsiveness [25]. Myocardial hibernation does not result from the ischemic insult itself, but from chronically reduced coronary blood flow to an area. This hypoperfused myocardium has reduced contractility that may be recovered with revascularization allowing for overall improved cardiac output [26–28].
Right ventricle failure

Right ventricle (RV) infarction complicates anywhere from 30% to 50% of ACS cases, although it only clinically manifests as RV failure in 10% of cases [29]. It is commonly associated with inferior MI, but also it can be found in posterior and anterior distributions, usually to much lesser degrees. Isolated RV failure is a rare cause of cardiogenic shock after MI, occurring in less than 3% of patients [18,30]. These patients are preload dependent and often require judicious intravenous resuscitation with crystalloid to improve hemodynamics. Patients with RV failure are also reliant on atrial filling and the atrial kick for right-sided cardiac output. Ironically, patients with RV involvement after inferior wall MI are much more likely to develop high-degree atrioventricular (AV) block (48%) than those without RV involvement (13%) [31].

Acute mitral valve regurgitation

Acute mitral valve regurgitation (MVR) is found commonly in patients with AMI. Almost 40% of patients have mild to moderate regurgitation seen on echocardiogram 24 hours after infarction [32,33]. A total of 6% to 7% of patients have severe MVR resulting in hemodynamic compromise and pulmonary edema [33]. Acute MVR is associated more with inferior and posterior MI than in anterior distributions [34]. Acute MVR occurs by several mechanisms [35,36]:

1. Systolic tenting or tethering of the valve
2. Mitral annulus dilation from severe left ventricular dysfunction
3. Ischemia of papillary muscle
4. Ruptured papillary muscle
5. Ruptured chordae tendinae
6. Worsening pre-existing mitral regurgitation

In the setting of acute MVR, afterload reduction improves cardiac output by allowing greater forward flow. Development of severe MVR has been found to have significant prognostic implication with higher prevalence of long-standing heart failure and death [33].

Papillary muscle rupture is a distinct entity causing acute MVR. It is usually seen 2 to 7 days postinfarction. Often the posterior papillary muscle is the site of rupture because of a typical, singular blood supply from the posterior descending branch of a dominant right coronary artery [37]. Immediate surgical repair or valve replacement is indicated in papillary muscle rupture with a mortality rate of 50% within the first 24 hours in patients who do not undergo surgical intervention [38]. Risk factors for the development of acute MVR and papillary muscle rupture are listed previously.

Left ventricle free wall rupture

Incidence of free wall rupture has significantly decreased from 6% to around 1% with the use of angioplasty for AMI [18,39,40]. Rupture occurs within the first 24 hours after infarction in 40% of cases and within 1 week
85% of the time [41]. Patients often complain of persistent or recurrent chest pain, intractable nausea or repeated emesis, or present with syncope or agitation [39]. Paradoxical bradycardia is seen in up to 20% of cases [42,43]. Risk factors for free wall rupture are listed along with the other mechanical causes in Table 1.

**Ventricular septal rupture**

The reported incidence of ventricular septal rupture varies from 0.4% in the GUSTO-I trial to as high as 4% seen in the SHOCK trial [18,44]. Rupture usually occurs within a thin, akinetic area of septal myocardium. Interestingly, ventricular septal rupture more commonly occurs in patients without a history for MI who have complete occlusion of a single vessel [45]. It is thought that chronic stenosis is protective against rupture by allowing scarring and remodeling of the damaged myocardium, which produces a more durable fibrinous architecture [45]. The left anterior descending artery is most frequently the causative vessel. Table 2 lists the risk factors for ventricular septal rupture.

**Diagnostic evaluation**

Clinically, the patient with cardiogenic shock has a systolic blood pressure less than 90 mm Hg; evidence of hypoperfusion (decreased urine output; decreased mental status; cool, clammy skin); cardiac index less than 2.2 L/min/m²; and LV end diastolic pressure or pulmonary capillary wedge pressure greater than 15 mm Hg [17]. Patients with LV failure often have extra heart sounds and rales on lung examination, whereas patients with RV failure often have clear lung fields with jugular venous distention or Kussmaul’s sign (paradoxical increase in jugular venous distention with inspiration). Mechanical etiologies of shock may produce their own discrete physical findings. In the case of LV free wall rupture, the patient has signs

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**Table 1**

<table>
<thead>
<tr>
<th>Risk factors for mechanical causes of cardiogenic shock complicating acute myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute mitral valve regurgitation</td>
</tr>
<tr>
<td>Age &gt; 65 years</td>
</tr>
<tr>
<td>Gender Female</td>
</tr>
<tr>
<td>Multivessel versus single-vessel Multivessel</td>
</tr>
<tr>
<td>Location Inferior-posterior</td>
</tr>
<tr>
<td>History of previous myocardial infarction Yes</td>
</tr>
</tbody>
</table>

of cardiac tamponade with distended neck veins; hypotension with a narrow pulse pressure; and distant, muffled heart sounds. Patients with acute MVR and ventricular septal rupture may have an audible murmur, although they may be clinically silent because of low cardiac output [46].

Ideally, an ECG should be performed expeditiously on all patients with suspected cardiogenic shock. Depending on the etiology, different ECG patterns may be expected. Patients may have tracings consistent with an acute STEMI, Q waves consistent with areas of previous damage, or diffuse ST-depression representing ischemia. Right precordial leads may be useful in patients with clinical signs of right heart failure. Arrhythmias, atrioventricular dissociation, and left bundle branch block may be noted and present additional complications that worsen the patient’s cardiac output.

Echocardiography and right heart catheterization are excellent tools to guide therapy. According to the 2003 guidelines of the ACC, AHA, and American Society of Echocardiography, the use of echocardiography to

Table 2
Hemodynamic profiles in various states of cardiogenic shock

<table>
<thead>
<tr>
<th>Condition</th>
<th>PCWP, cardiac output, systemic vascular resistance</th>
<th>RAP, RAP/PCWP &gt; 0.8, exaggerated RA descent, RV square root sign</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricle failure</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Right ventricle failure</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>Mitral valve regurgitation</td>
<td>Large PCWP v wave</td>
<td></td>
</tr>
<tr>
<td>Ventricular free wall rupture, tamponade</td>
<td>Equalization of diastolic pressures (approximately 20 mm Hg)</td>
<td></td>
</tr>
<tr>
<td>Ventricular septal rupture, tamponade</td>
<td>Large PCWP v wave, oxygen saturation step-up (&gt;5%) from RA to RV</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: PCWP, pulmonary capillary wedge pressure; RA, atrium; RAP, right atrial pressure; RV, right ventricle.


Table 3
Arterial supply to the cardiac conducting system

<table>
<thead>
<tr>
<th>Conduction system component</th>
<th>Vascular supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>SA node</td>
<td>RCA in 60%</td>
</tr>
<tr>
<td></td>
<td>Left circumflex in 40%</td>
</tr>
<tr>
<td>AV node</td>
<td>RCA 90%</td>
</tr>
<tr>
<td></td>
<td>Left circumflex 10%</td>
</tr>
<tr>
<td>His-bundle</td>
<td>Primarily RCA with some septal perforators from the LAD</td>
</tr>
<tr>
<td>Proximal left bundle branch</td>
<td>LAD predominates</td>
</tr>
<tr>
<td>Left anterior fascicle</td>
<td>Proximally AV nodal branch of RCA</td>
</tr>
<tr>
<td>Left posterior fascicle</td>
<td>Distal dual supplied by anterior and posterior septal perforating arteries</td>
</tr>
<tr>
<td>Right bundle branch</td>
<td>Septal perforators ± AV nodal</td>
</tr>
<tr>
<td></td>
<td>LAD perforators ± collateral from RCA/LCX</td>
</tr>
</tbody>
</table>

Abbreviations: AV, atrioventricular; LAD, left anterior descending; LCX, left circumflex; RCA, right coronary artery; SA, sinoatrial.

diagnosis mechanical complications of MI receives a class I recommendation [47]. Echocardiography allows for rapid assessment of myocardial and valvular function, and presence of pericardial effusion from free wall rupture. It also provides an assessment of the RV in possible cases of RV infarction. Subset analysis of the SHOCK trial demonstrated that echocardiographic evaluation of left ventricular ejection fraction and mitral regurgitation performed within 24 hours of infarction could provide more detailed information on prognosis and mortality [32].

Right heart catheterization allows for central hemodynamic monitoring. The patient’s volume status and pulmonary capillary wedge pressure can be evaluated. Table 3 illustrates the hemodynamic profiles associated with each specific clinical scenario [48]. This objective information may be critical in guiding treatment with vasopressors, vasodilators, and inotropes.

**Treatment**

*Inotropes and vasopressors*

The goal of treating patients with cardiogenic shock is tissue perfusion stabilization and optimization, with a goal systolic blood pressure greater than 90, mean arterial pressure greater than 65, and urine output of 0.5 mL/kg/h. Initial intervention may include fluid resuscitation, unless the patient is in frank pulmonary edema. Judicious use of intravenous fluid boluses allows for optimizing preload and filling pressures so that cardiac output is maximized. Patients with RV failure have great dependence on preload and may require more aggressive fluid resuscitation. If hypotension persists despite adequate filling pressures achieved with fluid resuscitation, then inotropes and vasopressors may be necessary. Dobutamine, a selective $\beta_1$-adrenergic receptor agonist, improves myocardial contractility and cardiac output with less effect on heart rate and systemic vascular resistance and is the inotrope of choice in patients with systolic blood pressure greater than 80 mm Hg. Dopamine also acts directly on $\beta_1$-adrenergic receptors and indirectly through norepinephrine. It causes increases in vasoconstriction, heart rate, and myocardial contractility. Because it may increase myocardial demand, dopamine is the inotrope of choice in patients with systolic blood pressure less than 80 mm Hg. If hypoperfusion persists despite the use of dobutamine and dopamine, norepinephrine or phenylephrine may be necessary. Phenylephrine, a pure $\alpha_1$-adrenergic agonist, is useful when tachyarrhythmias limit the use of vasopressors. Care must be taken with the addition of vasopressors because there is a delicate balance between improved coronary perfusion pressures and increasing myocardial demand. Phosphodiesterase inhibitors, such as milrinone, have inotropic actions but also cause vasodilatation. Although they are less arrhythmogenic and chronotropic, they have prolonged half-lives and may cause hypotension, so one must be cautious when using them in patients with acute cardiogenic shock in the setting of ischemia.
Intra-aortic balloon pump counterpulsation

Intra-aortic balloon pump counterpulsation increases diastolic coronary artery perfusion pressures and decreases afterload without increasing myocardial demand. The Thrombolysis and Counterpulsation to Improve Cardiogenic Shock Survival trial randomized patients to thrombolysis with or without intra-aortic balloon pump use and found improved 6-month mortality in patients with Killip class III or IV failure (39% versus 80%, respectively) [49]. Intra-aortic balloon pump use is advocated as a Class IB recommendation in the ACC-AHA guideline as a stabilizing measure when mechanical complications of MI result in cardiogenic shock or when hypotension persists despite other interventions [50]. It should be noted that the placement of an intra-aortic balloon pump by emergency physicians does not represent the standard of care in most communities and that this procedure is usually performed by an interventional cardiologist or a cardiothoracic surgeon.

Fibrinolysis versus revascularization

There are limited data to support the efficacy of fibrinolysis in patients with cardiogenic shock. Thrombolysis has been shown to decrease the incidence of cardiogenic shock in patients with STEMI, but not to improve outcomes in patients presenting with AMI and cardiogenic shock [35,51,52]. It is presumed that decreased coronary perfusion pressures limit the effectiveness of fibrinolytics and that use of intra-aortic balloon pump counterpulsation may improve their efficacy [36].

Revascularization by percutaneous coronary intervention (PCI) or coronary artery bypass grafting allows for improved coronary flow after infarction. In the Primary Angioplasty in Myocardial Infarction trial, PCI was compared with fibrinolysis in high-risk patients (> 70 years, large anterior MI, heart rate > 100) and was found to have significant mortality benefit (2% versus 10.4%, respectively) [53]. GUSTO-I showed a significant decrease in 30-day mortality in patients who underwent aggressive revascularization within 24 hours of shock onset (38%) versus fibrinolysis alone (62%) [9]. Additionally, the SHOCK study showed a nonsignificant decrease in 30-day all-cause mortality between the group undergoing early revascularization versus medical management (46.7% versus 56%) [5]. There was a significant absolute risk reduction for mortality of 13% at 6 months in the early revascularization group, which persisted at 12 months [5]. Based on these data, the ACC-AHA guidelines recommend patients younger than 75 years of age undergo early revascularization as a Class I indication [50].

Complications of fibrinolysis

Fibrinolytic therapy remains a common treatment for STEMI both in the Unites States and abroad. Severe hemorrhage, specifically intracranial hemorrhage (ICH), is the most feared complication of fibrinolytic therapy for
AMI. The failure to re-establish coronary perfusion after administration of a fibrinolytic agent is also a common scenario. These topics are discussed in the following section.

**Hemorrhage**

Hemorrhagic complications after fibrinolytic therapy can range from minor gingival bleeding to ICH. Bleeding after fibrinolysis most frequently occurs at venous access or vascular puncture sites [54–56]. Invasive procedures should be limited after fibrinolysis is given. If bleeding occurs at a compressible site, then direct pressure should be used to minimize bleeding. Gastrointestinal bleeding has been shown in studies to be the most common form of severe spontaneous bleeding [54,56,57]. Blood transfusions should be given to patients with hemodynamic compromise with gastrointestinal bleeding. Only a minority of patients, less than 1% in most studies, who receive fibrinolytic therapy develop ICH [54,58–62]. Several studies have examined patient factors that may indicate a higher risk for ICH after fibrinolysis. Patient characteristics associated with increased rates of ICH after fibrinolytic therapy are as follows [56,59–62]:

- Age > 75 years
- Female gender
- Black race
- History of cerebrovascular accident
- Systolic blood pressure > 160 mm Hg on presentation
- Dyastolic blood pressure > 100 mm Hg on presentation
- Low body weight
- Tissue plasminogen activator dose > 1.5 mg/kg.

Although these risk factors are not contraindications for fibrinolysis, the recognition of their presence allows the emergency department physician to estimate better the risk of ICH.

Any change in neurologic status including focal deficits, seizure activity, or change in mental status occurring during or after fibrinolytic therapy should be presumed to be from ICH until proved otherwise. Fibrinolytic, antiplatelet, and anticoagulant therapy should be discontinued until neuroimaging is performed. If ICH is present, fresh frozen plasma and cryoprecipitate can be used to restore fibrinogen levels. Protamine should be given to reverse the effect of heparin, and platelet transfusions may be necessary if bleeding times are prolonged. ε-Aminocaproic acid is an option for fibrinolysis reversal by inhibiting activation of plasminogen to plasmin. Much consideration must be taken before administration because its use may cause significant systemic thrombosis.

In addition to the management of the fibrinolytic-induced coagulopathy, patients with ICH may also require other medical interventions and neurosurgical consultation. If there are concerns for increased intracranial
pressure, then such methods as elevating the head of bed, optimizing blood pressure, administration of mannitol, and endotracheal intubation with hyperventilation may be necessary.

**Failure of fibrinolysis**

Failure to reperfuse has been defined by the ACC-AHA as absence of at least some normalization of ST-segment elevation 60 to 90 minutes after initiation of fibrinolytic therapy; persistent symptoms (eg, chest pain, diaphoresis, shortness of breath); or electrical or hemodynamic instability [63]. There have been several randomized studies performed to evaluate the benefit of rescue PCI versus conservative therapy or repeat fibrinolysis [64–67]. The largest and most comprehensive trial was the Rescue Angioplasty versus Conservative Treatment or Repeat Thrombolysis trial that showed significantly higher rate of event-free survival with PCI compared with both conservative therapy and repeat fibrinolysis [67]. Results of three meta-analyses performed on rescue PCI trials have shown decreased rates of mortality and decreased need for revascularization, but they have also shown increased rates of thromboembolic stroke [68–70]. At facilities that do not have PCI capabilities, the potential benefits of rescue PCI should be countered with the risk of emergency transportation of a patient with presumed failed fibrinolysis. Patients at higher risk for complications from their AMI, such as those with large anterior infarctions or inferior infarctions with RV involvement, may benefit more from rescue PCI than their lower-risk counterparts. As for risk factors that predispose patients to fibrinolytic failure, a small study by Deepa and Mishra [71], found that patients with more than three risk factors for coronary artery disease were at higher risk of failing fibrinolysis than those with fewer than three risk factors. This was a limited study, but may serve as a catalyst for additional research into high-risk characteristics for failure of fibrinolysis.

**Arrhythmias in acute coronary syndrome**

**Background**

Arrhythmias occur in anywhere from 3% to 40% of patients with AMI [72–75]. Onset of arrhythmias may occur during the acute phase of an ACS or later after AMI. There are multiple mechanisms for the development of arrhythmia in ACS. The interruption of normal electrical impulse transmission through the myocardium creates areas of tissue that are supersensitive to sympathetic stimulation, rendering them more arrhythmogenic [76]. At the cellular level, decreased function of the energy-dependent sodium-potassium-ATPase pump and increased permeability of myocardial cell walls lead to metabolic derangement of the myocardium [77]. These changes affect the resting membrane potential of the myocardium causing increased arrhythmogenicity. Increased levels of metabolic by-products
can also adversely affect the heart raising the risk of arrhythmias [78]. Ischemia can also have significant effects on the action potential of cardiac myocytes to varying degrees depending on their proximity to the area of infarction, and this heterogenic conduction provides further arrhythmogenicity [79]. The categories of arrhythmias seen in ACS can be broken down into bradyarrhythmias, tachyarrhythmias, and reperfusion rhythms.

**Bradyarrhythmias**

Bradyarrhythmias at the simplest level can be divided into sinus bradycardia and heart blocks. Sinus bradycardia is defined as a heart rate less than 60 beats per minute with each QRS complex preceded by a P wave and is seen in up to 40% of patients with AMI. The blood supply to the heart’s conduction system plays a significant role in certain arrhythmias seen in AMI (Table 3). Sinus bradycardia is seen most commonly with inferior MI because of the blood supply of the sinoatrial node [73]. Loss of blood supply to the normal pacemakers of the heart can cause bradycardia secondary to ischemia and relatively increased vagal tone. Sinus bradycardia in the setting of AMI is usually transient and typically resolves within 24 hours. Treatment with vagolytics, such as atropine, is reserved for patients who are suffering from hypotension or ischemia secondary to bradycardia. Temporary pacing can be used if the patient fails to respond to atropine, but permanent pacing is rarely required in these patients [73].

Multiple types of heart block can be seen in the setting of AMI [80]. The basic underlying pathology of these conduction disturbances is caused by decreased upstroke velocity of the action potential in the cardiac myocytes secondary to ischemia [81]. The type of block seen depends on the coronary artery creating ischemia and the portion of the conduction pathway that it supplies. For instance, the right coronary artery supplies the AV node in approximately 90% of patients and either a first-degree AV block, a second-degree type I, or a complete heart block with a narrow QRS complex indicates an infranodal lesion associated in patients with acute right coronary artery infarction. For this same reason, second-degree type 2 AV block, complete heart block with a wide QRS complex, or new bundle branch blocks may be present in a patient suffering from acute left anterior descending occlusion because it supplies most of the blood to the conduction system distal to the AV node.

Atropine should be considered as a first-line agent for use in patients who are symptomatic with signs of hypoperfusion and either second-degree type one or complete heart block with a narrow QRS (ie, lesions at the level of the AV node). The use of atropine is discouraged in patients with high-degree AV block (ie, infranodal block), and asymptomatic patients [82]. In some cases of second-degree heart block, the use of atropine has been reported to cause progression to complete heart block, although conflicting evidence in the literature exists [83].
In cases where atropine is not used or is unsuccessful, temporary pacing may be indicated for symptomatic bradycardia. Transcutaneous pacing is easily done in most emergency department cases, but occasionally transvenous pacing wires may need to be placed. Optimally, this procedure is performed under fluoroscopy, and possibly done in coordination with rapid coronary catheterization for diagnostic and potentially therapeutic purposes.

Tachyarrhythmias

Tachyarrhythmias fall into two major groups: atrial and ventricular tachyarrhythmias. Atrial tachyarrhythmias are commonly associated with AMI and are seen in approximately 6% to 20% of these patients [75]. These arrhythmias are most often seen 3 to 72 hours after an infarction occurs. Atrial fibrillation or atrial flutter associated with rapid ventricular responses in the setting of AMI can significantly increase oxygen demands, exacerbating the already ischemic myocardium. Atrial fibrillation in the setting of AMI does impart a significantly higher short- and long-term mortality demonstrated by the GUSTO-I and III trials and the PURSUIT and TRACE trials [84–87]. In GUSTO-I, the unadjusted mortality rate at 30 days was 14.3% in patients with atrial fibrillation versus 6.2% in those with sinus rhythm [84].

The presence of hemodynamic compromise and ongoing ischemia must be assessed to determine the appropriate intervention in patients with tachycardic atrial fibrillation and atrial flutter. Heart rate control with an AV nodal blocker (eg, β-adrenergic antagonists and nondihydropyridine-type calcium channel blockers) is appropriate in patients without hypotension or cardiogenic shock. Amiodarone is also a reasonable option for slowing or chemically cardioverting rapid atrial fibrillation and atrial flutter. β-Blockers are generally preferred as a first-line agent for rate control because there is the added benefit of decreasing sympathetic stimulation to the heart, further decreasing ischemia. Care must be taken to avoid hypotension in all ACS patients to avoid extending the ischemic penumbra of an AMI.

In those patients with both hemodynamic compromise and ongoing ischemia, synchronized cardioversion should be attempted to treat atrial fibrillation or atrial flutter. Repeated shocks with escalating energy may be required, but the danger of ventricular arrhythmia induction or asystole increases with multiple shocks. Combinations of pharmacologic agents and cardioversion may be effective in recalcitrant cases.

Paroxysmal supraventricular tachycardia is also seen in the setting of AMI, although with a lower frequency. As in atrial fibrillation with a rapid ventricular response, rate-induced ischemia in the ACS patient mandates rapid treatment. Reentrant mechanisms and increased automaticity are the primary mechanisms involved, so the administration of a vagal maneuver including carotid massage, Valsalva, or coughing is initially indicated.
and advanced cardiac life support algorithms suggest the use of adenosine bolus therapy if simple vagal maneuvers fail. Electrical cardioversion is indicated for hemodynamically unstable patients and in those in whom pharmacologic treatment has failed [82].

Ventricular arrhythmias can range from premature ventricular contractions to ventricular fibrillation. Ventricular arrhythmias are common in the setting of AMI with incidence ranging as high as 20% for ventricular fibrillation, up to 40% for ventricular tachycardia, and as high as 90% for premature ventricular contractions [72], although more recent studies in the era of fibrinolysis indicate significantly lower incidence. Premature ventricular contractions are typically asymptomatic and acutely have no bearing on short- or long-term morbidity and mortality. Treatment aimed at suppression of these beats is not helpful and in some cases may even be harmful [88]. Nonsustained ventricular tachycardia is defined as ventricular tachycardia that spontaneously resolves in less than 30 seconds. The significance of nonsustained ventricular tachycardia depends on its temporal relationship to AMI. Specifically, nonsustained ventricular tachycardia early in course of AMI defined as within 48 hours of AMI onset was relatively benign and did not require treatment unless associated with concurrent hemodynamic decompensation. If a patient becomes hypotensive or has angina during recurrent nonsustained ventricular tachycardia, then a rate-controlling medication, such as intravenous amiodarone or a β-blocker, may be used [82]. Sustained ventricular tachycardia is divided into two distinct categories: stable or unstable. The unstable varieties are further subdivided into polymorphic or pulseless monomorphic, which are treated with unsynchronized electrical defibrillation, and monomorphic ventricular tachycardia with hemodynamic instability, which is treated with synchronized cardioversion [82]. Stable ventricular tachycardia may be treated initially medically with amiodarone, or procainamide before cardioversion if a patient can temporarily tolerate the increased heart rate of the ventricular tachycardia. The final ventricular tachydysrhythmia is ventricular fibrillation. This rhythm is almost universally terminal if left untreated and usually deteriorates into asystole within a few minutes time. Prompt recognition and intervention in the form of electrical defibrillation is crucial. Ventricular fibrillation that does not respond to initial defibrillation should be treated following current ACLS guidelines.

Reperfusion arrhythmias

Ventricular arrhythmia and ectopy following the administration of fibrinolytic therapy in the setting of STEMI are termed “reperfusion arrhythmias.” Premature ventricular contractions, ventricular tachycardia, ventricular fibrillation, and accelerated idioventricular rhythms all may be observed when reperfusion occurs. The hypothesized etiologies underlying the development of reperfusion are numerous and include cell injury,
swelling, and necrosis, and myocardial stunning [89]. Accelerated idioventricular rhythms is seen in up to 50% of patients with AMI, but is not considered a sensitive or specific marker for reperfusion [89–91]. Pulse rates with accelerated idioventricular rhythms in the 60 to 100 range are usually associated with improved cardiac output and systemic perfusion. Accelerated idioventricular rhythms should be observed unless the patient becomes hemodynamically compromised and it generally resolves spontaneously. The use of antiarrhythmic agents in stable patients with accelerated idioventricular rhythms may present risk of harm without significant benefit.

Summary

ACS and associated ACS complications are highly prevalent among emergency department patients. The spectrum of ACS and ACS complications are very treatable diseases and modern aggressive therapy has greatly reduced patient morbidity and mortality. The maintenance of specific critical care management skills in the recognition and treatment of ACS complications is essential for the practicing emergency physician.

References


