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# 44

## How Does One Diagnose and Manage Acute Myocardial Ischemia in the Intensive Care Unit?

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p0250 Intensivists face several challenges when critically ill patients present with myocardial ischemia or infarction. Patients may be admitted to the intensive care unit (ICU) with a primary diagnosis of cardiac injury. In other hospitalized patients, underlying atherosclerosis and noncardiac stresses such as hemorrhage, mechanical ventilation, and sepsis can precipitate a myocardial insult. A meta-analysis of patients in the ICU found elevated troponin levels in 12% to 85% of critically ill patients with a median frequency of 43%.<sup>1</sup> Although the incidence of myocardial injury, defined by elevations in troponin levels, is high, it is often unrecognized.<sup>1-3</sup> This chapter reviews the diagnosis, assessment, and management of critically ill patients with myocardial ischemia or infarction.

### s0010 **DIAGNOSIS: BIOMARKERS**

p0255 The diagnosis of myocardial infarction has been recently redefined to emphasize etiology and requires a troponin elevation above the 99th percentile of normal with at least one of the following criteria: ischemic ST- and T-wave changes, new left bundle branch block, new Q waves, percutaneous coronary intervention (PCI)-related marker elevation, or imaging suggestive of a new loss of viable myocardium.<sup>4</sup> In the setting of sudden death, myocardial infarction is diagnosed without an elevated troponin if ST-segment elevation, new left bundle branch block, evidence of fresh thrombus at angiography or autopsy, or new loss of viable myocardium occurs. Increases in biomarker levels of 3 × 99th percentile for PCI and 5 × 99th percentile for coronary artery bypass graft (CABG) also characterize myocardial infarction (Table 44-1).<sup>4,5</sup> The definition of myocardial infarction does not include myocyte necrosis from mechanical injury, which may occur in the setting of CABG or from myocardial cell death from etiologies such as sepsis, chest trauma, or cardioversion.<sup>4</sup>

p0260 Ischemia results when there is inadequate oxygen supply from coronary artery blood flow to satisfy the oxygen demands of the myocardium. Myocardial infarction can occur in the setting of coronary artery thrombus,

inflammation, dissection, and plaque erosion or rupture.<sup>5</sup> Electrocardiogram (ECG) changes and historical symptoms such as angina, dyspnea, diaphoresis, nausea, syncope, and jaw, upper extremity, and epigastric discomfort may characterize myocardial ischemia. Ischemia often is accompanied by a failure in myocardial contractility that results from myocardial necrosis, stunning, or hibernation. A stunned myocardium occurs after coronary occlusion and produces regional wall motion abnormalities for hours or days despite reperfusion. Hibernation is an adaptive response to chronically reduced coronary blood flow and describes decreased myocardial contractility, a “self-preserving” mechanism to minimize ischemia or necrosis. After prolonged ischemia, myocardial infarction occurs, and an elevation in cardiac troponin will be seen.

When myocardial necrosis occurs, proteins such as cardiac troponins T and I, creatine phosphokinase (CPK), myoglobin, and lactate dehydrogenase (LDH) are released into the circulation. Because of their sensitivity and specificity, the rise and fall of troponin levels are the preferred biomarkers for the evaluation of myocardial injury. If troponin testing is not available, CK-MB measurements are the best alternative biomarker.<sup>4</sup> Troponin levels should be drawn at the onset of symptoms and 6 to 9 hours later to evaluate the enzyme’s rise and fall. Occasionally, the patient may require a blood sample 12 to 24 hours later if the initial troponin evaluation was normal and the clinical suspicion for cardiac ischemia was high.<sup>4</sup> Troponin T and I are generally equivalent, except in patients with chronic kidney disease, in which troponins may stay elevated from impaired clearance. In patients with end-stage renal disease, an increase in troponin T without evidence of myocardial necrosis is more common than an increase in troponin I. Nevertheless, an increase in troponin T in the setting of renal failure is associated with an increased morbidity.<sup>5</sup>

In critically ill patients with and without acute coronary syndromes, an elevated troponin level has been associated with increased mortality.<sup>1,6-9</sup> A rise in troponin values, however, does not indicate the mechanism of

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t0010

**Table 44-1 Clinical Classification of Different Types of Myocardial Infarction**

**TYPE 1**

- Spontaneous myocardial infarction related to ischemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection

**TYPE 2**

- Myocardial infarction secondary to ischemia due to either increased oxygen demand or decreased supply, such as coronary artery spasm, coronary embolism, anemia, arrhythmias, hypertension, or hypotension

**TYPE 3**

- Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischemia, accompanied by presumably new ST elevation, new left bundle branch block, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood

**TYPE 4A**

- Myocardial infarction associated with percutaneous coronary intervention

**TYPE 4B**

- Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy

**TYPE 5**

- Myocardial infarction associated with coronary artery bypass graft

From Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. *J Am Coll Cardiol.* 2007;50:2173-2195.

injury. Therefore, in the absence of ischemic features, elevations in troponin levels should prompt clinicians to search and examine their patients for nonischemic etiologies of myocardial injury. Many disease processes, such as sepsis, tachycardia, congestive heart failure, renal failure, pulmonary embolism, pulmonary hypertension, chemotherapy, burns, extreme exertion, and stroke are associated with an increase in troponin.<sup>4</sup> In sepsis, altered myocyte permeability may release troponin into the circulation and increase intracellular calcium. Direct myocardial injury from cytokine-mediated responses have been implicated in sepsis-induced myocardial depression.<sup>10-13</sup> Clinically, myocardial depression from sepsis is a reversible process that does not require revascularization.

p0275 An elevated troponin is often a result of myocardial injury, and its presence can provide the practitioner with insight into the severity of a patient's illness. Troponin levels have emerged as a marker of outcome in the ICU. In the ICU, however, it is critical to distinguish between elevated troponins as a marker of acute myocardial injury and as a reversible, treatable, and independent predictor of outcome.

p0280 Measurement of brain natriuretic peptide (BNP) in acute coronary syndrome (ACS) may have prognostic

value. An elevation in plasma BNP after ACS is associated with recurrent myocardial infarction, worsening heart failure, and death.<sup>14</sup>

**DIAGNOSIS: ELECTROCARDIOGRAPHY**

s0015

Ordering an ECG is essential in patients with suspected cardiac ischemia or infarction. ECG findings such as the evolution of ST-segment abnormalities and Q waves can provide essential information regarding the duration, size, and location of injury. Characteristic features of myocardial ischemia and infarction are listed in Tables 44-2 and 44-3. When inferior myocardial infarction is suspected, a right-sided ECG should be recorded to evaluate right ventricular infarction.

ECGs should be interpreted in the context of troponin values because patients in the ICU may have conditions such as early repolarization, pericarditis, myocarditis, ventricular hypertrophy, hypokalemia, cholecystitis, tachycardia, and digitalis effect that may cause ECG changes without biomarker evidence of ischemia.<sup>4</sup>

Ordering an echocardiogram in the setting of myocardial ischemia provides diagnostic and prognostic information and detects complications. The diagnostic use of echocardiography is recommended in cases in which acute ischemia is not detected by standard means despite

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**Table 44-2 Electrocardiographic Manifestations of Acute Myocardial Ischemia in the Absence of Left Ventricular Hypertrophy and Left Bundle Branch Block**

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**ST ELEVATION**

- New ST elevation at the J-point in two contiguous leads with the cut-off points:  $\geq 0.2$  mV in men or  $\geq 0.15$  mV in women in leads V2 to V3 and/or  $\geq 0.1$  mV in other leads

**ST DEPRESSION AND T-WAVE CHANGES**

- New horizontal or down-sloping ST depression  $\geq 0.05$  mV in two contiguous leads and/or T inversion  $\geq 0.1$  mV in two contiguous leads with prominent R wave or R/S ratio  $> 1$

From Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. *J Am Coll Cardiol.* 2007;50:2173-2195.

**Table 44-3 Electrocardiographic Changes Associated with Prior Myocardial Infarction**

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- Any Q-wave in leads V2 to V3  $\geq 0.02$  sec or QS complex in leads V2 and V3
- Q-wave  $\geq 0.03$  sec and  $\geq 0.1$  mV deep or QS complex in leads I, II, aVL, aVF, or V4 to V6 in any two leads of a contiguous lead grouping (I, aVL, V6; V4 to V6; II, III, and aVF)\*
- R wave  $\geq 0.04$  sec in V1 to V2 and R/S  $\geq 1$  with a concordant positive T wave in the absence of a conduction defect

\*The same criteria are used for supplemental leads V7 to V9 and for the Cabrera frontal plane lead grouping.  
From Thygesen K, Alpert JS, White HD. Universal definition of myocardial infarction. *J Am Coll Cardiol.* 2007;50:2173-2195.

a high suspicion. The presence of left ventricular (LV) dysfunction or mitral regurgitation after myocardial infarction is an adverse prognostic finding. After myocardial infarction, echocardiography can detect complications such as residual ischemia, ventricular septal defects, papillary muscle rupture or dysfunction, free wall rupture, regurgitant lesions, LV thrombus, or tamponade.<sup>15</sup> Radio-nuclide ventriculography, myocardial perfusion scintigraphy (MPS), and magnetic resonance imaging (MRI) are techniques that assess the viability of myocardial tissue and can characterize the extent of injury.<sup>4</sup>

p0300 Mechanically ventilated patients, particularly those who are chronically critically ill, often will undergo prolonged periods of ventilator weaning. A small prospective study found that myocardial ischemia detected by continuous ECG monitoring is common in patients requiring prolonged mechanical ventilation. Further, evidence of ischemia increased the risk for remaining ventilator dependent.<sup>16</sup> In mechanically ventilated patients with risk factors for coronary artery disease, myocardial ischemia detected by ST-segment analysis was noted in 24% of patients. In this patient population, the interruption of sedation was not associated with an increased occurrence of myocardial ischemia.<sup>17</sup> Therefore, ST-segment monitoring is encouraged during weaning from mechanical ventilation and a reduction in sedative infusions in patients with coronary artery disease is not contraindicated if needed to facilitate liberation from mechanical ventilation.

p0305 Diagnosing myocardial ischemia and infarction amenable to coronary intervention in the ICU can be difficult and challenging. Critically ill patients often are intubated and sedated and unable to communicate regarding ischemic symptoms. In addition, analgesia and sedation may mask symptoms of ischemia.<sup>18</sup> Because troponin levels may be elevated in ICU patients for a variety of reasons other than myocardial ischemia, biomarker evaluation is uncertain. In addition, imaging studies such as transthoracic echocardiography may be inadequate in the setting of positive-pressure ventilation, obesity, chronic obstructive pulmonary disease, chest tubes, and thoracic bandages. The use of contrast echocardiography, harmonic imaging and transesophageal echocardiography can improve the adequacy images obtained in critically ill patients.<sup>19</sup>

#### s0020 **MANAGEMENT: PATHOPHYSIOLOGIC BASES**

p0310 The management of myocardial ischemia in critically ill patients is guided by the multifaceted nature of the problem and involves amelioration of inciting conditions such as hypoxemia, anemia, pain, fever, stress, and increased work of breathing. Supportive measures, hemodynamic control, and interventions to improve or restore myocardial perfusion are the mainstays of therapy. The clinician should search for reversible problems such as anemia and surgical or gastrointestinal bleeding, undertake corrective measures, and consider cardiology consultation for possible revascularization.

p0315 Maintenance of adequate oxygenation is essential. In patients with respiratory distress or hypoxemia,

supplemental oxygen should be administered to maintain oxygen saturation at more than 90% (American College of Cardiology and American Heart Association [ACC/AHA] 2007 Non-ST-elevation myocardial infarction [NSTEMI] guidelines, class I, level B recommendation).<sup>5</sup> Oxygen saturation above this level may reduce heart rate. Although there is no clear evidence that oxygen helps in the absence of respiratory compromise, it is a low-risk intervention that minimizes the potential for under-recognized hypoxemia. Assisted ventilation may be required to improve gas exchange and to decrease work of breathing in patients with respiratory distress.

p0320 Anemia leads to diminished oxygen delivery and increases myocardial work and oxygen demand. It has been associated with the development of ischemia as well as with a higher incidence of adverse cardiac events or death in patients with unstable angina and myocardial infarction. In observational studies, the risk increases with the progressive decrease of hemoglobin below 10 to 12 g/dL.<sup>20</sup> Control of bleeding and prevention of anemia are one important aspect of the management of ischemia. Blood transfusion, however, has been linked to increased risk for death.<sup>21</sup> A large randomized controlled trial in critically ill patients has suggested that restrictive transfusion practices decrease cardiopulmonary complications and improve outcomes.<sup>22</sup> In the absence of good randomized controlled trials specifically addressing the relationship among the degree of anemia, transfusion threshold, and outcomes in patients with ischemia, the optimal transfusion targets remain unclear. Patients in the ICU without significant cardiac disease are likely to benefit from a restrictive transfusion threshold of 7 to 8 g/dL of hemoglobin. A higher target for hemoglobin may be considered in patients with preexisting cardiac disease. Given the problems associated with both anemia and transfusion, clinicians should try to limit blood loss (especially unnecessary blood draws) in ischemic and anemic patients.

#### s0025 **MANAGEMENT: PHARMACOLOGIC APPROACHES**

p0325 Adequate analgesia plays important role in the management of ischemia in the ICU. Narcotics have been used to control angina as well as pain from other sources that may precipitate ischemia. Morphine also decreases the heart rate, peripheral vascular resistance, and myocardial wall stress by reducing left ventricular preload and can be beneficial in the setting of pulmonary edema. Animal and human studies have suggested a role for morphine in ischemic preconditioning and reduction in infarct size. The effects of morphine on hemodynamics, however, are variable, and significant hypotension and bradycardia may worsen myocardial perfusion.<sup>23</sup> Additional compromise can be caused by respiratory depression. A large observational study in patients with acute coronary syndrome demonstrated increased unadjusted and adjusted rates of death and myocardial infarction with the use of intravenous morphine.<sup>24</sup> No randomized controlled trials are available to guide the use of morphine in ischemia, and the ACC/AHA guidelines support its use for ST-elevation myocardial infarction (STEMI)<sup>25</sup> but advise

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caution in patients with unstable angina or NSTEMI (ACC/AHA 2007 NSTEMI guidelines, class IIa, level B recommendation).<sup>5</sup>

p0330 Nonsteroidal anti-inflammatory drugs (NSAIDs), with the exception of aspirin, and selective cyclooxygenase-2 inhibitors should not be used in patients with ischemia because of the increased risk for thrombotic events, reinfarction, and death<sup>26,27</sup> (ACC/AHA 2007 NSTEMI guidelines, class III, level C recommendation).<sup>5</sup>

p0335 Nitroglycerin decreases left ventricular preload and left ventricular wall stress. At higher doses, it also reduces afterload. Nitroglycerin dilates coronary arteries, both those with stenosis and the collateral vessels to the ischemic regions. Nitrates should not be administered during absolute or relative hypotension. If ischemia is not improved through sublingual or transdermal nitroglycerin, intravenous infusion can be started. Nitrate use in ischemia is based on observational data, but randomized trials demonstrate only a varying mortality benefit in myocardial infarction.<sup>28</sup> Meta-analysis of the trials supports some clinical utility.<sup>29</sup> Subsequent randomized controlled trials of therapies in ischemia have not reaffirmed the strength of the evidence for the use of nitrates. In the absence of well-designed study of the effects of this class of medications, nitroglycerin continues to be a first line of therapy of ischemia (ACC/AHA 2007 NSTEMI guidelines, class I, levels B and C recommendation).<sup>5</sup>

p0340  $\beta$ -Blockers decrease heart rate, contractility, and blood pressure, thus reducing cardiac work and oxygen consumption. The reduction in cardiac events and mortality with early administration of  $\beta$ -blockers during unstable angina and myocardial infarction has been supported by some randomized trials and meta-analyses and questioned by others.<sup>30–33</sup> Pooled analysis of several studies indicates that  $\beta$ -blockers reduced short-term mortality in patients with acute coronary syndromes who had PCI.<sup>34</sup> However, in patients with hemodynamic instability or with risk factors for heart failure such as low output state, low blood pressure, high heart rate, or older age,  $\beta$ -blockers may increase the risk for cardiogenic shock and should be used judiciously or avoided.<sup>35</sup> This class of medications should be used cautiously in patients with reactive airway disease and are best avoided in asthmatics<sup>36,37</sup> as well as in patients with significant atrioventricular (AV) block. Delayed administration of  $\beta$ -blockers as a secondary prevention measure in patients with LV dysfunction reduces mortality and reinfarction rate. A recent randomized controlled trial of carvedilol compared with placebo in addition to standard medical therapy in patients with LV dysfunction 3 to 21 days after myocardial infarction demonstrated reduced all-cause mortality and nonfatal myocardial infarction without significant increase in the incidence of shock.<sup>38</sup> During noncardiac surgery,  $\beta$ -blockers have the potential to reduce perioperative cardiac events as suggested in cohort studies, randomized controlled trials, and meta-analyses; however, recent trials and systematic reviews have challenged the presence and the magnitude of these effects.<sup>39–41</sup> The benefits appear significant mostly in high-risk patients. ACC/AHA 2007 NSTEMI guidelines classify use of oral  $\beta$ -blockers as class I, level B recommendation and intravenous use as class IIa, level B recommendation.<sup>5</sup>

Calcium channel blockers include agents with varying hemodynamic effects. The evidence from trials and meta-analyses supports the use of drugs with negative chronotropy such as verapamil and diltiazem.<sup>42,43</sup> In the setting of ischemia, they reduce myocardial oxygen demand by decreasing heart rate, contractility, and afterload along with arterial dilation resulting in improvement of coronary blood flow. Calcium channel blockers control tachyarrhythmias and are helpful when  $\beta$ -blockade is contraindicated. However, they should be avoided in patients with LV dysfunction and used cautiously in patients with decreased AV conduction. Short-acting dihydropyridines (e.g., nifedipine), on the other hand, have been associated with increased adverse events, especially when used without concomitant  $\beta$ -blockade.<sup>44</sup> ACC/AHA 2007 NSTEMI guidelines classify use of nondihydropyridine calcium channel blockers as a class I, level B recommendation in lieu of  $\beta$ -blocker and as a class IIa, level C recommendation along with  $\beta$ -blocker.<sup>5</sup>

Angiotensin-converting enzyme (ACE) inhibitors improve blood pressure control and ventricular remodeling and reduce the death rate in high-risk patients with ischemia and myocardial infarction. The benefit is particularly evident in patients with LV dysfunction and heart failure after myocardial infarction.<sup>45</sup> ACE inhibitors should be used orally in the acute setting and avoided in the presence of hypotension. Similar effects are noted with angiotensin-receptor blockers (ARBs), which can be used when ACE inhibitors are contraindicated. ACC/AHA 2007 NSTEMI guidelines classify use of ACE inhibitors and ARBs as a class I, level A recommendation in the setting of LV dysfunction.<sup>5</sup>

Antiplatelet agents such as aspirin, alone or with anticoagulation therapy, reduce the risk for death or myocardial infarction in patients with ischemia and should be administered unless significant bleeding risk from gastrointestinal, intracranial, or other source exists.<sup>46–48</sup> Withdrawal of aspirin therapy, used for primary or secondary prevention in patients with coronary artery disease, has increased the incidence of cardiovascular adverse events. Interruption of drug therapy should be avoided, and it should be restarted as soon as bleeding risk has been reduced, particularly in the perioperative period.<sup>49</sup> The use of aspirin according to the ACC/AHA 2007 NSTEMI guidelines is a class I, level A recommendation.<sup>5</sup> Other antiplatelet agents are the adenosine diphosphate (ADP)-receptor antagonists, the thienopyridines ticlopidine and clopidogrel. They are useful alone or in conjunction with aspirin for the treatment of unstable angina and myocardial infarction and result in decreased cardiovascular death and myocardial infarction<sup>50–52</sup> (ACC/AHA 2007 NSTEMI guidelines, class I, level A recommendation).<sup>5</sup> Clopidogrel has a more favorable side-effect profile than ticlopidine. These agents increase the risk for minor and major bleeding. In critically ill patients, the potential benefits should be weighed against the associated risk, especially in the perioperative setting. Double antiplatelet therapy (DAT) with aspirin and thienopyridine after PCI reduces the incidence of major cardiovascular events. Patients with preexisting coronary stents warrant special consideration. The presence of bare-metal stents requires 1 month of uninterrupted DAT while drug-eluting stents require at least 12 months of such

therapy. Premature discontinuation of therapy has led to a higher rate of stent thrombosis and fatal myocardial infarction. Therefore, therapy should be discontinued only in the case of significant bleeding risk.<sup>53,54</sup> An intravenous glycoprotein IIb/IIIa inhibitor added to a thienopyridine may be beneficial if an invasive coronary intervention is planned (ACC/AHA 2007 NSTEMI guidelines, class I, level A recommendation).<sup>5</sup>

p0360 Anticoagulation is an essential aspect of the therapy for ischemia and myocardial infarction. The available agents range from unfractionated heparin (UFH) and factor Xa inhibitors such as low-molecular-weight heparin (LMWH) and fondaparinux to the direct thrombin inhibitors (DTIs) argatroban and bivalirudin. The initiation of anticoagulation should be strongly considered in critically ill patients with unstable angina and myocardial infarction. However, the benefits should be weighed against the risk for bleeding, particularly in the perioperative setting. No particular anticoagulation regimen has been proved superior, and the choice should be guided by the bleeding hazard related to the drug pharmacokinetics, pharmacodynamics, and reversal ability as well as the risk for heparin-induced thrombocytopenia (HIT).<sup>55-57</sup> DTI are generally reserved for use in patients with HIT. ACC/AHA 2007 NSTEMI and STEMI guidelines classify anticoagulation agents as a class I, level A recommendation.<sup>5,25</sup>

p0365 Statins decrease myocardial infarction, stroke, and cardiovascular mortality. In patients with acute coronary syndromes, early aggressive statin therapy may reduce unstable angina and reinfarction. Therefore, these medications should be considered when PCI is indicated.<sup>58-60</sup> Patients in the ICU may already be taking statins. Prolonged withdrawal should be avoided because adverse effects have been shown in surgical ICU patients.<sup>61,62</sup> Other studies have suggested that statins may or may not be protective against renal, pulmonary, and multiorgan failure.

p0370 Other investigational drugs may prove useful in the future. Atrial natriuretic peptide in a randomized controlled trial decreased infarct size and improved long-term ejection fraction in patients with acute myocardial infarction who had revascularization. However, this drug was associated with a higher incidence of severe hypotension. A trial of the adenosine triphosphate (ATP)-sensitive potassium channel opener nicorandil demonstrated improved long-term ejection fraction but did not affect infarct size.<sup>63</sup> The calcium sensitizer levosimendan had positive inotropic effects and arterial and venous vasodilatory properties. It improved preload, contractility, and afterload without increasing oxygen consumption. In a small randomized controlled trial of patients with or without cardiogenic shock after acute myocardial infarction and revascularization, levosimendan significantly improved hemodynamics and coronary flow reserve compared with placebo or dobutamine.<sup>64,65</sup>

p0375 Severe hyperglycemia worsens outcomes in critically ill patients. In diabetic and nondiabetic patients with acute coronary syndrome, hyperglycemia was associated with cardiovascular complications and increased incidence of mortality, effects consistently seen in the setting of

PCI.<sup>66-68</sup> Tight glycemic control with insulin infusions has become a prevalent intervention in the contemporary ICU and has important benefits in the management of ischemia. The target glucose range at which the maximal benefits are realized while avoiding the risk for hypoglycemia remains to be determined. In a trial of intensive insulin therapy, a protocol used 80 to 110 mg/dL as a target range,<sup>69</sup> whereas others have aimed to keep blood glucose less than 200 mg/dL. Recent multicentered trials have failed to demonstrate the benefits of tight glucose goal of 80 to 110 mg/dL, while finding increased risk for hypoglycemia with this goal.<sup>70-71</sup>

Therapy interventions according to the level of efficacy in the 2007 ACC/AHA NSTEMI<sup>5</sup> and STEMI<sup>25</sup> guidelines are summarized in Table 44-4. p0380

## MECHANICAL SUPPORT AND CORONARY REVASCULARIZATION

s0030

In patients with acute ischemia with hemodynamic instability, intra-aortic balloon pump counterpulsation can be used for circulatory support during revascularization<sup>72</sup> (ACC/AHA 2007 NSTEMI and STEMI guidelines, class IIa, level C recommendation).<sup>5,25</sup> p0385

The decision to proceed with invasive interventions aimed at revascularization in addition to medical management is best done with the assistance of a cardiology consult. In the cardiology setting, patients with unstable angina and NSTEMI who have refractory angina despite optimal medical therapy or patients with hemodynamic instability benefit from early invasive strategy. Survival and quality of life improved with early invasive therapy compared with conservative management in stabilized patients as well.<sup>73,74</sup> ACC/AHA 2007 NSTEMI<sup>5</sup> and STEMI<sup>25</sup> guidelines classify early invasive therapy as class I, levels A and B recommendation. Invasive intervention should be avoided in patients with significant comorbidities in whom the risks for performing such intervention outweigh the potential benefits. Many patients in the ICU are in a state of acute decompensation and therefore may not be suitable candidates for invasive intervention. In the perioperative setting, the bleeding risk often precludes the institution of aggressive anticoagulation needed for revascularization. p0390

## AUTHORS' RECOMMENDATIONS

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- Troponin elevations are common in ICU patients. Although not always due to myocardial ischemia or infarction, such elevations are associated with poor outcome.
- Electrocardiography and imaging studies may further define pathophysiology and assist in prognosis.
- Pharmacologic therapy of myocardial ischemia and infarction includes  $\beta$ -blockade, statin therapy, and aspirin.
- Although acute coronary revascularization may occasionally be performed, in most ICU patients, comorbidities, contraindications, and instability usually preclude acute CABG or PCI.

t0025 Table 44-4 Recommendation for Interventions According to the 2007 American Heart Association and American College of Cardiology Non-ST Elevation and ST Elevation Myocardial Infarction Guidelines

Class I (benefit significantly outweighs risk; intervention is indicated and should be done)	Oxygen in hypoxia Nitroglycerin sublingual or intravenous β-Blocker orally Long-acting nondihydropyridine calcium channel blocker as β-blocker alternative ACE inhibitor or ARB orally with LV dysfunction Discontinue NSAID Aspirin Antiplatelet agents: thienopyridine (±glycoprotein IIb/IIIa inhibitor) Anticoagulation
Class IIa (benefit outweighs risk; additional focused studies are needed; intervention is reasonable and can be beneficial)	Oxygen in all ischemia Morphine β-Blocker IV Long-acting nondihydropyridine calcium channel blocker in addition to β-blocker alternative ACE inhibitor or ARB orally without LV dysfunction IABP Glycoprotein IIb/IIIa inhibitor Anticoagulation
Class IIb (benefit may outweigh risk; further studies are needed and may be considered; effectiveness is uncertain)	Extended-release nondihydropyridine calcium channel blocker Immediate-release dihydropyridine calcium antagonists with adequate β-blockade
Class III (risk outweighs benefit; not recommended and may be harmful)	Nitrates in hypotension, tachycardia or bradycardia, or along with phosphodiesterase inhibitor for erectile dysfunction Immediate-release dihydropyridine calcium antagonists without adequate β-blockade ACE inhibitor IV NSAID

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; IABP, intra-aortic balloon pump; LV, left ventricular; NSAID, nonsteroidal anti-inflammatory drug.

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