Revascularization procedures (percutaneous coronary intervention [PCI] and coronary bypass graft surgery [CABG]) are performed in more than 1.7 million patients with ischemic heart disease in the United States; more than 2.2 million PCIs alone are performed worldwide on an annual basis. With technological advances in coronary intervention over the past 3 decades, procedural complications and long-term outcomes have significantly improved, yet periprocedural myocardial infarction (MI) remains common. We review the current state of knowledge of the incidence, risk factors, prognosis, and prevention of periprocedural MI after revascularization procedures, including PCI and CABG.

**Clinical Classification of Periprocedural MI**

The definition of MI after revascularization procedures has evolved with better understanding of the etiology and prognostic significance of these events. Historically, the World Health Organization definition of MI required 2 of 3 criteria, including clinical symptoms, ECG abnormality, and creatine kinase (CK) elevation (\( \geq 2 \times \text{the upper limit of normal [ULN]} \)). The World Health Organization biomarker threshold was then adopted to define periprocedural MI, irrespective of clinical syndrome or ECG findings. With increasingly sensitive serological biomarkers and imaging techniques, minute amounts of myocardial necrosis have become detectable. Although any level of periprocedural myonecrosis may be termed periprocedural MI, the prognostic implications are highly variable, depending on the biomarker thresholds applied and clinical circumstances. As such, it is essential to understand the relationship between periprocedural MI, causality, and prognosis.

Varying diagnostic criteria including the type, extent, and timing of biomarker release, symptoms, ECG, and imaging criteria have been proposed in the Universal Definition of Myocardial Infarctions, which has been endorsed by most academic societies and regulatory bodies (Table 1). PCI-related MI (type 4) is distinguished from spontaneous MI (type 1), secondary MI (type 2), and those associated with sudden death (type 3) or CABG (type 5) (Table 1). After PCI among patients with a normal baseline troponin value, elevations of cardiac biomarkers above the 99th percentile of the upper reference level (URL) are indicative of periprocedural myocardial necrosis. By convention, increases of biomarkers greater than \( 3 \times 99\text{th percentile URL} \) have been designated as defining PCI-related MI (type 4a). A subtype of PCI-related MI related to documented stent thrombosis is also recognized (type 4b). In patients with normal baseline troponin values undergoing CABG, elevations of cardiac biomarkers above the 99th percentile URL are also indicative of periprocedural myocardial necrosis, and by convention, increases of biomarkers greater than \( 5 \times 99\text{th percentile URL} \) with either new pathological Q waves or new left bundle-branch block, or angiographically documented new graft or native coronary occlusion, or imaging evidence of new loss of viable myocardium define CABG-related MI (type 5) (Table 1). Myocardial reinfarction is defined by a new rise of \( \geq 20\% \) in serum biomarkers over and beyond the last nadir, which meets criteria (\( \geq 3 \text{ SD difference of the variance of the measure} \)) for differences in analytic values.

The task force for universal definition as well as regulatory bodies including the US Food and Drug Administration further recommend reporting biomarkers based on multiples of the 99th percentile URL of the biomarker, as well as by defined subtype (Table 1) thereby displaying the severity of the varying types of MIs. For practical purposes, the ULN of the local laboratory is often substituted for the 99th percentile URL and has comparable thresholds and implications.

**Serum Biomarkers**

Cellular proteins are released into the circulation after myocyte damage including myoglobin, lactate dehydrogenase, CK, and cardiac troponin (cTn) T and I, among others, each with characteristic release kinetics and greater or lesser cardiac sensitivity. The most thoroughly validated biomarker for postprocedural MI is the CK-MB isoenzyme (CK-MB) measured by mass assay. The most sensitive and specific biomarker of myonecrosis with the longest time window for elevation is troponin (T or I). Although troponins have not been as extensively validated in the context of revascularization as CK-MB. Serial samples of both troponin and CK-MB should be performed at baseline and every 6 to 10 hours to

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From Yale University School of Medicine (A.J.L.), New Haven, Conn; and Columbia University Medical Center and the Cardiovascular Research Foundation (G.W.S.), New York, NY.

Correspondence to Alexandra J. Lansky, MD, Yale University School of Medicine, 300 George St, Suite 759, New Haven, CT 10022. E-mail alexandra.lansky@yale.edu

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identify a characteristic rise exceeding the 99th percentile of URL with subsequent fall for the diagnosis of MI.

Predictors and Mechanisms of MI After Revascularization Procedures

Revascularization procedures resulting in direct instrumentation and manipulation of the coronary arterial vasculature (whether CABG or PCI) predispose patients to ischemic events that can lead to myocardial necrosis.

After PCI, a number of factors have been associated with periprocedural MI, which can broadly be categorized as (1) patient-related factors; (2) lesion-related factors; and (3) procedure-related factors (Figure 1). Patient-related factors, including multivessel disease, evidence of systemic atherosclerosis, reduced left ventricular ejection fraction, diabetes mellitus, older age, and chronic kidney disease, increase the risk of postprocedural CK-MB release by 1.3- to 1.8-fold. Systemic inflammation on presentation including elevated hs-C-reactive protein correlates with postprocedural CK-MB elevation, as does an elevated admission white cell count (>9.5×10⁹/L). The clinical syndrome on presentation also affects risk, with enzyme negative patients with acute coronary syndromes (ACS) having up to a 40% incidence of post-PCI enzyme elevations and enzyme-positive ACS patients having even more frequent and larger postprocedural MIs (Table 2). Lesion-related factors such as device selection, in particular, atherectomy; aggressive stent expansion resulting in plaque extrusion; side branch occlusion; and angiographic complications including distal embolization, coronary dissection, no-reflow, vasospasm, and unsuccessful procedures, are all associated with periprocedural MI.

In totality, these risk factors identify patients with increasing atherosclerotic disease burden, increased thrombotic risk, and neurohormonal activation that predisposes to either macrovascular complications (side branch occlusion or macroembolization) or microvascular obstruction (distal embolization of microparticles), unifying the pathophysiologic basis of myocardial necrosis after PCI (Figure 1). At issue is whether periprocedural biomarker elevations are independent predictors of subsequent mortality (implying causality) or merely represent underlying comorbidities and diffuse atherosclerosis (not all of which may be presently measured and accounted for).

Prevalence and Prognosis of Periprocedural MI

The prevalence of periprocedural MI varies markedly, based on the biomarker and threshold used and the clinical syndrome on

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**Table 1. Clinical Classification of Different Types of Myocardial Infarction**

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>Spontaneous MI related to ischemia caused by primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection</td>
</tr>
<tr>
<td>Type 2</td>
<td>Secondary MI caused by ischemia resulting from either increased oxygen demand or decreased supply, for example, coronary artery spasm, anemia, arrhythmias, hypertension, or hypotension</td>
</tr>
<tr>
<td>Type 3</td>
<td>Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischemia, associated with presumably new ST elevation, or new left bundle-branch block, or evidence of fresh thrombosis in a coronary artery by angiography and/or autopsy, but death occurring before blood samples could be obtained or at a time before the appearance of cardiac biomarkers in the blood</td>
</tr>
<tr>
<td>Type 4a</td>
<td>MI associated with PCI</td>
</tr>
<tr>
<td>Type 4b</td>
<td>MI associated with stent thrombosis as documented by angiography or autopsy</td>
</tr>
<tr>
<td>Type 5</td>
<td>MI associated with CABG</td>
</tr>
</tbody>
</table>


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**Figure 1.** Risk factors and mechanisms of biomarker release after PCI.
presentation (Tables 2 and 3). The distinction between spontaneous and periprocedural MI is essential because for any given CK-MB level, the absolute risk from spontaneous MI is higher than from periprocedural MI. In the large-scale ACUITY trial, in a time-updated, covariate-adjusted, multivariable analysis, spontaneous MIs (using a sensitive definition of any troponin elevation) were an independent predictor of mortality (hazard ratio [HR], 7.49; 95% confidence interval [CI], 4.95 to 11.33; $P<0.001$), whereas periprocedural MIs (CK-MB $>3\times$ ULN after PCI or $>5$ to $10\times$ ULN after CABG) were not ($P=0.22$). Preprocedural biomarker positivity (spontaneous MI) should be a controlled confounder when assessing the prognostic significance of subsequent periprocedural MI, a risk factor that has rarely been adjusted for in prior studies.

### Impact of Periprocedural CK-MB Elevations After PCI

The prognostic implications of CK-MB elevations after PCI have been highly debated. Early studies demonstrated a graded mortality risk with each increment of CK-MB release after PCI, with even minor CK-MB elevations (1 to $3\times$ ULN) having prognostic significance for up to 10 years. As a continuum, CK has been shown to independently predict a 1.05 (1.03 to 1.08) relative risk of mortality for every 100-U/L increment of CK above ULN and CK-MB a 1.06 (1.01 to 1.11) relative risk of mortality per CK-MB ratio unit increase above $1\times$ ULN. However, most studies that have assessed the independent risk of CK-MB by categorical thresholds have consistently attributed an independent mortality risk to large periprocedural MIs (those with biomarker elevations $>5$ to $8\times$ ULN), with increased mortality risk of up to 3- to 5-fold in the short and long term. Intermediate CK-MB (3 to $5\times$ ULN) levels detected after the procedure have been independently linked to a modest increase in mortality risk in some but not all studies (Table 2). However, the importance of low levels of enzymatic release after PCI (CK-MB 1 to $2\times$ ULN or troponin elevations in the absence of CK-MB elevation) is frequently (and appropriately) dismissed because there is currently little or no evidence for an independent risk of

<table>
<thead>
<tr>
<th>Stable CAD</th>
<th>n</th>
<th>PCI Type</th>
<th>Prevalence by Cardiac Marker Type</th>
<th>Follow-Up Time and Outcome Measure</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jeremias et al$^{34}$</td>
<td>5850</td>
<td>Stent</td>
<td>CK-MB $&gt;3\times$ ULN: 21.3% CK-MB $&gt;3\times$ ULN: 8.7%</td>
<td>1-year death</td>
<td>Nonpredictive</td>
</tr>
<tr>
<td>Cleveland Clinic$^{47}$</td>
<td>3478</td>
<td>Stent</td>
<td>CK-MB $&gt;3\times$ ULN: 24% CK-MB $&gt;3\times$ ULN: 9.8%</td>
<td>2-year death</td>
<td>CK-MB: OR, 95% CI $&gt;3\times$ ULN: 1.96 (1.29-2.77)</td>
</tr>
<tr>
<td>Ellis et al$^{12}$</td>
<td>8409</td>
<td>PCI</td>
<td>CK-MB $&gt;3\times$ ULN: 17.2%</td>
<td>4-month death</td>
<td>Max CK-MB: T Stat 3.24, $P&lt;0.001$</td>
</tr>
<tr>
<td>Stone et al$^{25}$</td>
<td>7147</td>
<td>Stent/athero/PoBA</td>
<td>CK-MB $&gt;3\times$ ULN: 17.9%</td>
<td>2-year death</td>
<td>CK-MB: HR, 95% CI $&gt;8\times$ ULN: 2.2 (1.6-3.0)</td>
</tr>
</tbody>
</table>
| Ioannidis et al$^{44}$ | 23230 | PCI | CK-MB 1–5× ULN: 19% CK-MB >5× ULN: 6% | 6- to 34-month death | CK-MB: RR, 95% CI 1–3× ULN: 1.5 (1.2–1.8) 3–5× ULN: 1.8 (1.4–2.4) 
\>5× ULN: 3.1 (2.3–4.2) |
| Tardiff et al$^{18}$ | 2341 | PCI | CK-MB 1–3× ULN: 13.8% CK-MB 3–5× ULN: 3.6% CK-MB 5–10× ULN: 3.7% CK-MB >10: 2.9% | 6-month death | CK-MB 1–3×; 3–5×; 5–10×; >10 associated with increased risk |
| Emory$^{45}$ | 15637 | PCI | CK 1–2× ULN: 4.6% CK 1–3× ULN: 1.1% CK >3× ULN: 1.6% | 10-year death | CK >3× ULN: HR, 95% CI 1.84 (1.41–2.41) |

ACUS indicates coronary artery disease; DES, drug-eluting stent; and POBA, balloon angioplasty; $T$ stat, $T$ statistic; OR, odds ratio; CI, confidence interval; RR, relative risk; HR, hazard ratio.
reduced survival from these small biomarker elevations once confounding factors are taken into account.\textsuperscript{9,12,25,47} Moreover, several large studies have failed to demonstrate an independent risk of periprocedural MI (even large infarcts) when confounders are accounted for such as an unsuccessful procedure\textsuperscript{34} or MI on presentation.\textsuperscript{42}

Thus, detection of any degree of CK-MB release, even small, identifies patients with cardiovascular comorbidities (greater burden of atherosclerotic disease and cardiac risk factors), and is thereby associated with a mortality increment over time. However, small enzymatic leaks do not carry an independent mortality risk.\textsuperscript{48} In the future, a comprehensive assessment of patient risk may require high-resolution imaging to define the full extent of myonecrosis and to identify the threshold beyond which biomarker elevations and microinfarcts become clinically meaningful.

### Table 3. Prevalence of Periprocedural Myocardial Infarction Based on Troponin and Clinical Syndrome Presentation

<table>
<thead>
<tr>
<th>PCI Type</th>
<th>Prevalence by Cardiac Marker Type</th>
<th>Follow-Up Time and Outcome Measure</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
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<tr>
<td>Stable CAD</td>
<td></td>
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</tr>
<tr>
<td>Bertinchant et al\textsuperscript{84}</td>
<td>105 POBA/stent cTn-I &gt; ULN: 22% cTn-T &gt; ULN: 18% CK-MB &gt; ULN: 11.4% Myoglobin &gt; ULN: 7.6%</td>
<td>16-month death</td>
<td>cTn-I nonpredictive</td>
</tr>
<tr>
<td>Fuchs et al\textsuperscript{31}</td>
<td>1129 PCI/stent cTn-I 1–3× ULN: 15.0% cTn-I &gt;3× ULN: 15.5%</td>
<td>8-month death</td>
<td>cTn-I nonpredictive</td>
</tr>
<tr>
<td>Garboz et al\textsuperscript{32}</td>
<td>109 Stent cTn-I &gt; ULN: 27% CK &gt; ULN: 15%</td>
<td>8-month death</td>
<td>cTn-I nonpredictive</td>
</tr>
<tr>
<td>Gruberg et al\textsuperscript{26}</td>
<td>116 Stent in CKD (Cr ≥ 1.8 mg/dL) cTn &gt; ULN: 43.1%</td>
<td>12-month death</td>
<td>cTn &gt; ULN: OR (95% CI) 2.26 (1.07–4.77) CK-MB &gt; 3× ULN: OR (95% CI) 2.43 (1.16–5.08)</td>
</tr>
<tr>
<td>Nallamothe et al\textsuperscript{23}</td>
<td>1157 PCI/stent cTn I 1–3× ULN: 16% cTn 3–5× ULN: 4.6% cTn 5–8× ULN: 2.0% cTn &gt;8× ULN: 6.5%</td>
<td>1-year death</td>
<td>cTn &gt;8× ULN: HR, 95% CI 2.4 (1.2–5.0)</td>
</tr>
<tr>
<td>Ricciardi et al\textsuperscript{21}</td>
<td>286 PCI/stent cTn &gt; ULN: 13.6%</td>
<td>1-year MACE</td>
<td>cTn continuous; RR (95% CI) 1.05 (1.02–1.07)</td>
</tr>
<tr>
<td>Kini\textsuperscript{46}</td>
<td>2873 PCI/stent CK-MB vs cTn &gt; ULN: 16.1% vs 38.9% 1–3× ULN: 12.2% vs 16.4% 3–5× ULN: 2.3% vs 8.4% &gt;5× ULN: 1.6% vs 14.1%</td>
<td>1-year death</td>
<td>CK-MB &gt;5× ULN: HR (95% CI) 6.7 (1.9–22.9) cTn nonpredictive</td>
</tr>
<tr>
<td>Natarajan et al\textsuperscript{87}</td>
<td>1128 PCI/stent cTn &gt; ULN: 17%</td>
<td>1-year death</td>
<td>cTn nonpredictive</td>
</tr>
<tr>
<td>Cavallini et al\textsuperscript{88}</td>
<td>3494 PCI/stent CK-MB vs cTn &gt; ULN 16% vs 44.2%</td>
<td>1-year death</td>
<td>cTn nonpredictive</td>
</tr>
<tr>
<td>ACS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prasad et al\textsuperscript{42}</td>
<td>5487 Stent cTnT &gt; ULN: 43% CK-MB &gt; ULN: 21%</td>
<td>28-month death</td>
<td>cTnT nonpredictive</td>
</tr>
<tr>
<td>Fuchs et al\textsuperscript{33}</td>
<td>132 PCI cTn &gt;1× admit level 38.6%</td>
<td>6-month death</td>
<td>cTn reelevation &gt;1× admit level: OR, 95% CI 6.2 (1.5–25.8)</td>
</tr>
<tr>
<td>Cantor et al\textsuperscript{46}</td>
<td>230 Stent cTn &gt; ULN: 48% CK-MB &gt; ULN: 29% (includes pre-PCI + enzymes)</td>
<td>90-day death/MI</td>
<td>Continuous: HR, 95% CI 1.02 (1.01–1.03)</td>
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</table>

CAD indicates coronary artery disease; POBA, balloon angioplasty; and CKD, chronic kidney disease; OR, odds ratio; CI, confidence interval; RR, relative risk; HR, hazard ratio; Cr, creatinine.

### Prognosis of Periprocedural Troponin Elevations

Widespread adoption of troponin levels, which allow detection of smaller amounts of myonecrosis than CK-MB,\textsuperscript{4,49} has by itself increased the incidence of spontaneous and periprocedural MIs by 40% to 50%,\textsuperscript{30} principally by enabling the diagnosis of microinfarcts (Table 3). Although elevated troponin levels may provide equivalent or stronger prognostic
value than CK-MB in patients presenting with spontaneous MI not related to revascularization procedures, the prognostic significance of isolated postprocedural troponin-defined MIs is not as well characterized. In a large study of 5487 patients undergoing PCI, preprocedural cTnT >ULN independently predicted death at 28 months (HR, 1.79 [1.35 to 2.39]; P<0.001) but postprocedure cTnT >ULN was not predictive (P=0.5). These findings were corroborated in a study of 2353 patients wherein only preprocedural but not postprocedure cTnT elevations predicted long-term mortality. From a meta-analysis of 20 studies including 15,581 patients, troponin elevation after PCI occurred in 32.9% of cases and was weakly associated with increased mortality (odds ratio, 1.35; 95% CI, 1.13 to 1.60) at 3- to 67-month follow-up (Figure 2). Most studies, however, have failed to demonstrate an independent relationship of postprocedure troponin with long-term mortality, with the exception of high-risk patients with chronic kidney disease, post-PCI reinfarction in enzyme-positive ACS patients, or in those with very high levels of periprocedural troponin (Table 3). Thus, although troponins have supplanted measurement of CK-MB in many hospitals for their expediency of measurement and lower cost, the scenarios and thresholds for which they are correlated with subsequent mortality is largely unknown. For these reasons, the use of even intermediate biomarker positivity (troponin or CK) has little relevance in quality benchmarking.

**Biomarkers and Anatomic Measures of Infarct Size**

Cardiac MRI has become the gold standard for quantification of ventricular infarct size. The use of contrast-enhanced (CE)-MRI allows quantification of <1 g of infarcted myocardium and has demonstrated that even small elevations in biomarkers represent true myonecrosis. In a study by Ricciardi et al, among 14 patients undergoing PCI, 9 had a postprocedural CK-MB elevation (median, 21 ng/mL; range, 12 to 93 ng/mL) or 2.3× ULN, CE-MRI detected discrete hyperenhancement within 4 weeks of PCI with a median estimated mass of myonecrosis of 2 g (range, 0.7 to 12.2 g). Hyperenhancement persisted in all but 1 patient within 3 to 12 months on follow-up MRI. Another study identified new hyperenhancements by CE-MRI (mean mass, 6±5.8 g) in all patients with a postprocedure increase in cTnl (range, 1.0 to 9.4 μg/L), with a strong correlation between cTnl level and infarct size measured early (r=0.84, P<0.001) and late (r=0.71, P<0.001). A strong correlation has also been demonstrated between cTnT and CE-MRI infarct size. This provides evidence of the anatomic consequence of injury associated with biochemical markers after PCI, suggesting that even small troponin or CK-MB elevations do represent irreversible discrete microinfarctions and that the magnitude of injury correlates with the extent of enzyme elevation. The threshold at which such microinfarctions begin to significantly affect prognosis, however, is unknown, although few question that no myonecrosis is preferred to some. Patients with left ventricular systolic dysfunction before revascularization may be especially at risk after further periprocedural myonecrosis.

**Prevention of Biomarker Elevation After PCI**

Therapeutic strategies that have been found in selected studies to reduce periprocedural MI have included statins, antithrombotic and antiplatelet agents, and devices to protect against distal embolization.

**Pharmacology to Reduce Periprocedural MI**

The benefit of high-dose statin therapy among patients undergoing PCI is well documented, and those not receiving statins are at increased risk of periprocedural CK-MB release. Statin therapy initiated before PCI has also been associated with a significant reduction in CK-MB rise as well as a mortality benefit. This hypothesis was validated in the modest-sized prospective, randomized Atorvastatin for Reduction of Myocardial Damage during Angioplasty (ARMYDA) trial, which demonstrated a significant reduction in periprocedural MI with high-dose statin administration before PCI. The acute benefits of high-dose statin therapy may be mediated through their acute antithrombotic and anti-inflammatory actions rather than their lipid-lowering effects; in one study, the most benefit was seen in patients with elevated hs-C-reactive protein. A large, multicenter, randomized trial is required to confirm the beneficial effect of statins for this application.

Dual antiplatelet therapy with adequate preloading and maintenance of thienopyridines has resulted in significant reductions in periprocedural MI. Specifically, the use of clopidogrel 600 mg loading at least 2 hours before intervention or the use of prasugrel 60 mg has reduced periprocedural MI rates by approximately 20% and 30%, respectively, in patients with ACS. The Evaluation of IIb/IIIa Inhibitor for Stenting (EPISTENT) demonstrated that the use of the GPIIb/IIIa inhibitor abciximab reduces periprocedural CK-MB elevations and possibly mortality after stenting, and in a pooled analysis of abciximab PCI trials, 18% of the mortality benefit of abciximab was CK-MB-related. The benefits of GPIIb/IIIa inhibitors may be mediated in part by improvements in perfusion of the
distal microvasculature but may also be mediated through their anti-inflammatory effects. In addition to being a potent inhibitor of platelet aggregation and thrombus formation, abciximab binds to the vitronectin receptor on endothelial, smooth muscle, and inflammatory cells and to the αMβ2 receptor on leukocytes. Abciximab has equipotent affinity for the vitronectin receptor of endothelial and smooth muscle cells as well as for the Mac-1 integrin found on monocytes and neutrophils, responsible for the inflammatory response to vessel injury. Whether small-molecule GPIIb/IIIa inhibitors such as tirofiban and eptibatide, which lack these nonplatelet effects, are equally as effective in suppressing myonecrosis has never been appropriately tested in a large-scale, comparative, randomized trial, although abciximab was shown to be more effective than low-dose tirofiban in preventing peri-PCI myonecrosis in patients with ACS in the TARGET trial.

More recently, it has been recognized that the clinical benefits of GPIIb/IIIa inhibitors are offset by their propensity to increase bleeding and thrombocytopenia, complications strongly associated with subsequent mortality. The direct thrombin inhibitor bivalirudin has demonstrated noninferior suppression of ischemic complications compared with administration of heparin plus GPIIb/IIIa inhibitors with significant reductions in bleeding in patients with ACS, resulting in reduced all-cause and cardiac mortality in patients with ST-segment elevation–MI undergoing primary PCI. GPIIb/IIIa inhibitors may also not be more effective in decreasing myonecrosis than high-dose clopidogrel in patients with stable ischemic heart disease undergoing PCI with unfractionated heparin procedural anticoagulation.

### Periprocedural MI After CABG
Myocardial ischemia and necrosis may occur after CABG as the result of direct cardiac trauma from manipulation, reperfusion injury, incomplete revascularization, hypotension, bleeding, ventricular arrhythmias, acute graft closure, inadequate myocardial protection, and cardioplegia. Most studies have shown similar mortality risk associated with post-CABG enzyme elevations as seen after PCI. The Guard During Ischemia Against Necrosis (GUIDIAN) study in 2332 CABG patients demonstrated the strongest mortality risk associated with large CK elevations (those above 10× ULN) at 6-month follow-up. Another registry of 3667 CABG patients with 5-year follow-up showed a graded reduction in survival associated with post-CABG CK-MB (CK-MB < ULN: 80%; CK-MB 1 to 3× ULN: 78%; CK-MB > 3× ULN: 73%; P=0.0008). Every unit increment of CK-MB was independently associated with mortality (odds ratio; 95% CI, 1.04 [1.009 to 1.062]; P=0.0007). As has been shown after PCI, CE-MRI can detect enzyme-related infarcts after CABG. Among 23 patients studied, 18 (73%) showed some evidence of infarction on CE-MRI with good correlation with post-CABG CK-MB measures (Figure 3).

### Conclusions
Improvements in revascularization technologies and technique have resulted in treatment of increasingly complex patients and lesions with both PCI and CABG. Although procedural and long-term outcomes have improved over the last several decades, periprocedural MI remains frequent, partly because of the adoption of more sensitive biomarkers. High-resolution imaging techniques such as CE-MRI have established that even small biomarker elevations represent myonecrosis. There is little controversy surrounding the prognostic consequences of large periprocedural MIs, but the clinical significance of smaller enzymatic MIs continues to be controversial and of little relevance for quality benchmarking.
References


11. Ellis SG, Ahmose JA, Anandikizadowski M, AstraZeneca, Eli Lilly, and Bristol Meyers Squibb. Dr Lansky has received honoraria from Abbott Vascular and Boston Scientific and is a consultant to The Medicines Company, AstraZeneca, Eli Lilly, and Abbott Vascular.


73. Schwarz M, Nordi T, Bode C, Peter K. The GP IIb/IIIa inhibitor abciximab (c7E3) inhibits the binding of various ligands to the leukocyte integrin Mac-1 (CD11b/CD18, alphaMbeta2). Thromb Res. 2002;107:121–128.


Periprocedural Myocardial Infarction: Prevalence, Prognosis, and Prevention
Alexandra J. Lansky and Gregg W. Stone

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