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AHA SCIENTIFIC STATEMENT

Considerations on the Management of Acute Postoperative Ischemia After Cardiac Surgery: A Scientific Statement From the American Heart Association

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ABSTRACT: Acute postoperative myocardial ischemia (PMI) after cardiac surgery is an infrequent event that can evolve rapidly and become a potentially life-threatening complication. Multiple factors are associated with acute PMI after cardiac surgery and may vary by the type of surgical procedure performed. Although the criteria defining nonprocedural myocardial ischemia are well established, there are no universally accepted criteria for the diagnosis of acute PMI. In addition, current evidence on the management of acute PMI after cardiac surgery is sparse and generally of low methodological quality. Once acute PMI is suspected, prompt diagnosis and treatment are imperative, and options range from conservative strategies to percutaneous coronary intervention and redo coronary artery bypass grafting. In this document, a multidisciplinary group including experts in cardiac surgery, cardiology, anesthesiology, and postoperative care summarizes the existing evidence on diagnosis and treatment of acute PMI and provides clinical guidance.

Key Words: AHA Scientific Statements ■ myocardial ischemia ■ thoracic surgery

Acute postoperative myocardial ischemia (PMI) after cardiac surgery is an infrequent event but can evolve rapidly and become a potentially severe complication. The diagnosis of acute PMI (defined here as myocardial ischemia occurring any time from arrival in the intensive care unit to discharge) is more complex than the diagnosis of ischemia in the nonpostoperative setting because of potential confounders related to the recent surgery and postoperative treatments. Once acute PMI is suspected and identified, treatment includes a spectrum ranging from conservative strategies to percutaneous coronary intervention (PCI) to redo coronary artery bypass grafting (CABG). Therapeutic decisions must take into account a number of factors ranging from the patients' baseline characteristics to the cause and acuity of acute PMI, as well as logistical and local considerations. In this document, a multidisciplinary group including experts in cardiac surgery, cardiology, anesthesiology, and postoperative care summarizes the existing evidence on the diagnosis and treatment of acute PMI and provides clinical guidance.

INCIDENCE OF ACUTE PMI AFTER CARDIAC SURGERY

Although the criteria defining nonprocedural myocardial ischemia are well established, there are no universally accepted criteria for the diagnosis of acute PMI.[1] Acute PMI after cardiac surgery is usually asymptomatic because patients are often incapable of reporting classic ischemic indicators in the early period due to the effects of postoperative pain management (anesthesia and

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analgesia), as well as the masking effect of symptoms related to surgery (pain, nausea, fatigue).[2] The workup is generally based on observational data adopting variable definitions such as the Third Universal Definition of Myocardial Infarction, Fourth Universal Definition of Myocardial Infarction, and the Society for Cardiovascular Angiography and Intervention's definition.[1,3] Furthermore, acute PMI is difficult to distinguish from myocardial infarction. In the Fourth Universal Definition of Myocardial Infarction, there is no consensus on the troponin cutoff points that clearly differentiate cardiac procedural myocardial ischemia from myocardial infarction.[1]

In recent studies, the incidence of myocardial infarction has been reported to range from 0.3% to 9.8% after isolated CABG and from 0.7% to 11.8% after concomitant valvular surgery (Table 1).[4] In other series, the incidence of myocardial infarction after CABG has been reported to be as high as 14% when assessed with cardiac biomarkers

Table 1. Incidence of Postoperative Ischemia After Cardiac Surgery

Author, year	Study period	Type of study	Procedure	No. of patients	Postoperative ischemia, %	Early mortality, %*	Long-term mortality
Rasmussen et al,[5] 1997	1990–1995	Prospective	Isolated CABG	2003	3.5	12.7	23% mortality at median 3-y follow-up
Fabricius,[6] 2001	1999	Retrospective	Isolated CABG	2052	6.4	14.5	NR
Thielmann et al,[7] 2006	1999–2006	Prospective	Isolated CABG	5427	2.2	14.9	No difference in survival at 6.5 y among PCI, redo CABG, and conservative treatment
Tan et al,[8] 2008	1999–2005	Retrospective	Isolated CABG	4301	1.4†	18.4	8.1% death within 4 y after PCI
Yau et al,[9] 2008	2002–2003	Retrospective	Isolated CABG	3014	9.8	6.8	2-y mortality was highest in patients with CK-MB >15 times the URL (9.5%)
Virani et al,[10] 2009	1997–2002	Retrospective	Isolated CABG	1731	1.7	13.0	NR
Karhunen et al,[11] 2010	1988–2007	Retrospective	Isolated CABG	5251	2.1	2.8	NR
Javierre et al,[12] 2012	2004–2009	Prospective	Valve surgery and CABG	2038	Men 11.8 Women 5.5	5.1	NR
Laflamme et al,[13] 2012	2003–2009	Retrospective	Isolated CABG	5598	0.7	15.8	NR
De Mey et al,[14] 2012	2003–2009	Retrospective	Valve surgery and CABG	5988	0.7	NR	NR
Szavits-Nossan et al,[15] 2012	1999–2009	Prospective	Isolated CABG	3000	1.8	30.0	NR
Davierwala et al,[16] 2013	2004–2010	Prospective	Isolated CABG	7461	5.3	7.3	75±2.9% 5-y survival (acute PMI) 86±0.5% 5-y survival (no acute PMI)
Narayan et al,[17] 2014	2011–2012	Retrospective	Isolated CABG	2312	1.1	11.1	NR
Gaudino et al,[18] 2015	2005–2014	Prospective	Valve surgery and CABG	5275	0.7	10.0	At a mean follow-up of 29±26 mo, 6 patients died
Hultgren et al,[19] 2016	2007–2012	Retrospective	Valve surgery and CABG	4446	2.0	7.0	77% 5-y survival (acute PMI) 87% 5-y survival (no acute PMI)
Preußer et al,[20] 2017	2006–2013	Retrospective	Isolated CABG	4028	4.2	10.7	63% 7-y survival (acute PMI) 81% 7-y survival (no acute PMI)
Alqahtani et al,[21] 2019	2003–2014	Retrospective	Isolated CABG	554 987	4.4	5.1	NR
Rupprecht et al,[22] 2019	2008–2015	Retrospective	Isolated CABG	4825	2.2	13.0	NR
Sef et al,[23] 2019	2011–2015	Retrospective	Isolated CABG	1119	3.8	9.0	NR
Laimoud and Qureshi,[24] 2020	2016–2019	Retrospective	Valve surgery and CABG	1869	3.3	36.0	NR
Sharma et al,[25] 2020	1996–2017	Retrospective	Isolated CABG	53 287	0.3	15.6	15.4% (PCI) 29.8% (Redo CABG) 45.5% (Conservative)

CABG indicates coronary artery bypass grafting; CK-MB, creatinine kinase-MB; NR, not reported; PCI, percutaneous coronary intervention; PMI, postoperative myocardial ischemia; and URL, upper reference limit.

*Defined as in-hospital or 30-day mortality among patients diagnosed with acute PMI.

†Includes only patients who underwent PCI or redo CABG within 72 hours of the index procedure.

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and electrocardiographic findings such as new Q waves or left bundle-branch block and 20% to 30% with cardiac magnetic resonance (CMR) imaging.[3] For isolated mitral valve replacement and mitral valve repair, the incidence is 1.7% and 2.2%, respectively, and is often related to circumflex artery injury.[26] Other factors that have been associated with acute PMI include cytokine release, endothelial dysfunction and atherosclerotic plaque instability, hypercoagulability and diminished fibrinolytic activity, hemodynamic changes, and sympathetic nervous system activity associated with anesthesia and postsurgical pain.[27]

CAUSES

A multitude of factors may lead to acute PMI after cardiac surgery, and they differ by type of surgical procedure performed (ie, coronary surgery versus noncoronary cardiac surgery; Figure 1).

Graft-Related Factors

Graft-related factors represent the most common cause of acute PMI after CABG.[4] A meta-analysis of 9 studies and 1104 patients with acute PMI after CABG showed that, among 1056 patients who underwent subsequent angiography, 62.1% had acute graft failure (95% CI, 56.6%–67.6%), 6.1% had incomplete revascularization (95% CI, 0.4%–12.5%), and 3.5% had new native coronary artery lesions (95% CI, 1.4%–5.7%).[28] Notably, 31.7% of patients did not have any abnormal findings on angiography (95% CI, 25.6%–37.8%). In a retrospective series of 1119 patients with isolated CABG of whom 43 (3.8%) underwent coronary angiography for suspected acute PMI, graft failure was present in 31 patients (72%) and was most frequently due to graft thrombosis/occlusion or anastomotic stenosis, with the left anterior descending artery being the most frequently affected coronary artery target.[23]

Technical Factors

Acute graft failure may also result from other technical issues such as twisting or kinking of the graft, traction due to insufficient length, or graft spasm. The reported incidence of acute graft failure attributable to graft spasm detected on coronary angiography ranges between 1.6%[16] and 11.4%,[11] and it may be relieved by systemic or local administration of nitrates.[11] Harvesting approaches, graft selection, optimal preservation conditions, and anastomotic techniques must be considered to avoid early graft failure and resulting acute ischemia.[29,30] The use of transit time flow measurement and intraoperative graft assessment strategies should be considered to minimize the incidence of acute PMI.[31] The evidence for an association between transit time flow measurement and improved early graft patency is particularly robust for arterial grafts, specifically the internal thoracic artery to left anterior descending artery. High-frequency epicardial ultrasound may also be considered to probe grafts intraoperatively in conjunction with transit time flow measurement, particularly for intramyocardial targets.[31]

Competitive Flow

Competitive flow between an arterial graft and the native coronary artery circulation is more frequently associated with chronic graft failure and rarely leads to an acute clinical event.[32,33]

Non–Graft-Related Factors

Compared with patients undergoing isolated cardiac surgical procedures, the incidence of non–graft-related ischemia is higher in patients undergoing combined surgical procedures. These patients are also at high risk for myocardial ischemia resulting from inadequate myocardial protection during cardioplegic cardiac arrest and acute global ischemia/reperfusion injury associated with cardiopulmonary bypass.[30,34] A recent retrospective study of 53 287 patients undergoing cardiac surgery included 180 patients (0.34%) who underwent angiography for acute PMI within 3 weeks after surgery; the majority of procedures were performed within 48 hours of surgery.[25] A total of 81 patients (45%) had acute graft failure; 30 patients (16.7%) had new native coronary artery occlusion or thrombosis; and 69 patients (38.3%) had no evidence of graft or native coronary artery pathology. Similar findings have been reported in other series.[16,18]

Suture Entrapment/Coronary Distortion

Coronary artery injury leading to acute ischemia is an infrequent yet well-recognized complication of valve surgery and may result from distortion or direct injury related mostly to suture placement. The left circumflex (or a large wrap-around right posterolateral branch) runs in close proximity to the mitral valve annulus and is susceptible to injury during mitral valve procedures, especially in the case of a codominant or left-dominant coronary system.[26,35] In patients with anomalous left circumflex artery originating from the right coronary artery and coursing posterior to the aortic root, there is a high risk of damage during aortic valve replacement.[36] Similarly, the right coronary artery can be injured or occluded during tricuspid valve repair,[16] most frequently in the segment from the acute margin to the crux, where the artery is closely related to the tricuspid annulus.[37] Meticulous placement of sutures in the annulus as opposed to the atrial wall minimizes the risk of coronary artery injury.

Other Factors

Other causes of myocardial ischemia after noncoronary surgery include obstruction of coronary ostia,[38] distal

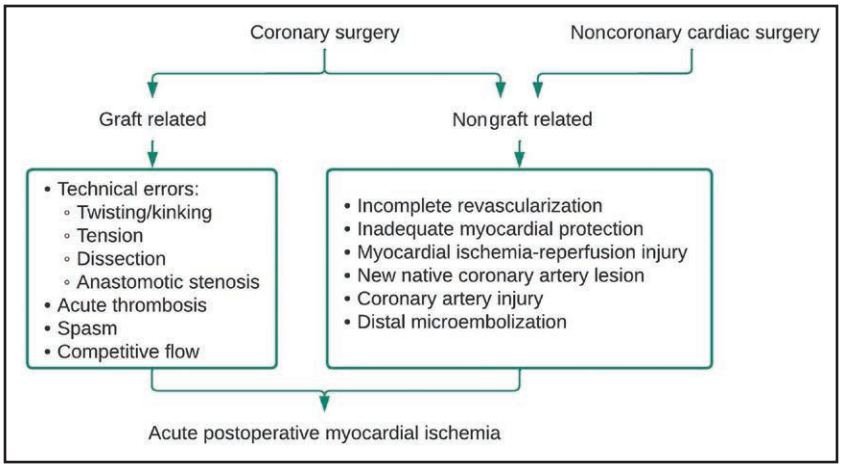

Figure 1. Mechanisms of postoperative myocardial ischemia after cardiac surgery.

embolization of calcified fragments in calcific aortic stenosis during valve replacement,[39] and external compression or distortion of the reimplanted coronary ostium during aortic root replacement.[40]

DIAGNOSIS

Clinical Presentation

Clinical diagnosis of acute PMI in patients undergoing cardiac surgery is challenging.[32] The reasons for this are multifactorial: Sedation may mask ischemic symptoms; there is distracting pain from either sternotomy or thoracotomy incisions; and hemodynamics after extracorporeal circulation frequently require support with inotropic and vasoconstrictor infusions even in patients without myocardial injury. Diagnosis may be further confounded by other early postoperative complications such as tamponade due to pericardial effusions compromising myocardial function. In a patient with failure to wean from cardiopulmonary bypass, new or unexpectedly high requirements for vasoactive support or mechanical circulatory support (MCS), postcardiotomy shock, low cardiac output syndrome, or chest pain out of proportion to usual incisional pain, further evaluation for acute PMI is warranted (Table 2).

Electrocardiogram

Serial ECGs are integral to the diagnosis of acute PMI.[41] New pathological Q waves, dynamic S- segment and T-wave changes, arrhythmias, and new left bundle-branch block that correlate with clinical presentation in patients without left ventricular (LV) hypertrophy and bundle-branch block at baseline indicate acute ischemia.[1,42] Of note, ST-segment and T-wave changes may be less specific for myocardial ischemia after cardiac surgery,[3] whereas ST-segment elevation with reciprocal changes is a more reliable indicator.[1] Interpretation of the ECG is frequently limited by postoperative paced rhythm or pericarditic changes.

Cardiac Biomarkers

Cardiac troponin (cTn) and creatine kinase-MB have traditionally been used as diagnostic and prognostic biomarkers in patients with suspected myocardial ischemia.[1,43,44] In modern practice, high-sensitivity cTn has become the biomarker of choice because of its earliest detection and more frequent association with coronary artery–related events.[45–47] Meanwhile, the creatine kinase-MB assay is no longer widely available; this represents a major practical limitation because the elimination kinetics are much faster with creatine kinase-MB (hours) than cTn (several days), which makes assessment of repeated events based on cTn value challenging.[44] cTn is expected to be elevated above the upper reference limit (URL) after all cardiac procedures. According to the Fourth Universal Definition of Myocardial Infarction, CABG-related (type 5) myocardial infarction is defined within 48 hours after surgery as increases of cTn >10 times the 99th percentile of the URL in patients with normal baseline cTn or by >20% in those whose cTn levels are elevated but stable, plus new electrocardiographic, angiographic, or imaging evidence of myocardial infarction or new loss of viable myocardium.[1] According to the Academic Research Consortium-2 expert consensus document, myocardial infarction was defined as increases in high-sensitivity cTn >35 times the URL with new evidence of ischemia or 70 times the URL as a standalone criterion.[48]

However, the large VISION cardiac surgery study (Vascular Events in Surgery Patients Cohort Evaluation) found that 30-day all-cause mortality was associated with much higher rises in high-sensitivity cTn.[49,50] Specifically, among patients who underwent isolated CABG or aortic valve replacement, a threshold high-sensitivity cTn level of 5670 ng/L (218 times the URL) on postoperative day 1 and 1522 ng/L (59 times the URL) on day 2 or 3 was associated with 30-day mortality. This threshold was 12981 ng/L (499 times the URL) on day 1 and 2503 ng/L (96 times the URL) on day 2 or 3 after other types of cardiac operations.[49] Thresholds for clinically

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Table 2. Summary of Diagnostic Criteria for Myocardial Ischemia After Cardiac Surgery

	Clinical	ECG	Biomarkers	Echocardiography	CMR
Criteria for diagnosis	Chest pain out of proportion to usual incisional pain, new or unexpectedly high inotropic support or MCS, failure to wean from cardiopulmonary bypass	New pathologic Q waves, dynamic ST-segment and T-wave changes, arrhythmia, and new LBBB that correlate with clinical presentation ST-segment elevation with reciprocal changes most reliable	Universal definition (type 5 MI): increases of cTn >10 times the 99th percentile URL in patients with normal baseline cTn or by >20% in those whose cTn levels are elevated but stable, plus new electrocardiographic, angiographic, or imaging evidence of MI or new loss of viable myocardium Academic Research Consortium-2 expert consensus: high-sensitivity cTn >35 times the URL with new evidence of ischemia or 70 times the URL as a standalone criterion*	New RWMAs in a territory dependent on a graft or a major ungrafted vessel, concomitantly with clinical presentation and typical cTn changes	New LGE corresponding to cTn changes LGE during first 2 wk diagnostic of type 5 MI
Pros	Integrates well with diagnostic tests	Serial ECGs integral to diagnosis of MI	Isolated and marked cTn rises are prognostic.	Practical and readily available	Extent of LGE well correlated with cTn elevation, nonrecovery myocardium, clinical presentation and sequelae of MI, and death
Cons	Ischemic symptoms masked by sedation, distracting pain Hemodynamic deterioration masked by presence of inotropic support	ST-segment and T-wave changes less specific after cardiac surgery Interpretation of the ECG limited by paced rhythm or pericarditic changes	cTn is elevated after all cardiac procedures. Actual thresholds are specific to assay and surgery type, as well as type of MI.	RWMAs nonspecific and may reflect pericarditis, hypertrophic cardiomyopathy, and inflammation Septal dyskinesis may occur with temporary epicardial pacing	Impractical and infrequently used in the acute postoperative setting

CMR indicates cardiac magnetic resonance; cTn, cardiac troponin; LBBB, left bundle-branch block; LGE, late gadolinium enhancement; MCS, mechanical circulatory support; MI, myocardial ischemia; RWMA, regional wall motion abnormality; and URL, upper reference limit.

*A recently published large cohort study[32] suggested that the actual cTn thresholds in association with all-cause 30-day mortality were higher and were dependent on procedure, biomarker assay, and type of myocardial injury.

important acute PMI are likely specific to biomarker type, the type of surgery (including off-pump versus on-pump CABG),[51,52] and whether the injury is primary versus from noncoronary occlusive events (type 2). The temporal rise and fall of cTn levels need to be correlated closely with the clinical presentation, intraoperative events, and other diagnostic findings.

Echocardiography

Echocardiography provides simultaneous cardiac structure and function assessment and is the most readily available imaging modality in the postoperative setting. A hallmark of ischemia is new regional wall motion abnormalities appearing immediately after onset in a territory dependent on a graft or a major ungrafted vessel, concomitantly with typical cTn changes.[3] Postoperatively, the quality of transthoracic images may be reduced by the presence of positive-pressure ventilation, bandages, and pleural and pericardial effusions.[48,53,54] Transesophageal imaging, the use of contrast agents to improve endocardial border definition, and tissue Doppler or speckle tracking can be effective strategies to overcome these challenges.[55–57] Echocardiography alone may often be nondiagnostic in the postoperative setting.[3,14] Intraoperative regional wall motion abnormalities on transesophageal echocardiography do not predict graft failure after CABG,[14] and new regional wall motion abnormalities may reflect noninfarct ischemic conditions and nonischemic conditions such as pericarditis, hypertrophic cardiomyopathy, and inflammation.[1,3] In addition, new septal wall dyskinesis commonly occurs after bypass and in the presence of temporary epicardial pacing and thus is nonspecific for ischemia.[58] In short, echocardiography is useful in correlation with the overall clinical picture and cardiac biomarker changes and can rule out nonischemic causes of chest pain, aortic dissection, and mechanical complications of myocardial infarction.[1]

Cardiac Magnetic Resonance

CMR provides high-resolution assessment of suspected ischemia and can detect small areas of subendocardial ischemia, characterizing the chronicity of injury and distinguishing between ischemic and nonischemic scars.[59] It may also be feasible in patients with temporary epicardial pacing wires.[60,61] However, CMR is impractical and infrequently used in the immediate postoperative setting.[53,54,62] New late gadolinium enhancement on CMR during the first 2 weeks after surgery is diagnostic of type 5 myocardial infarction.[63] The extent of late gadolinium enhancement is generally well correlated with the magnitude of cTn elevation, nonrecovery myocardium, and occurrence of major adverse cardiovascular events, nonfatal

myocardial infarction, hospital admission for unstable angina or heart failure, ventricular arrhythmia, and death.[47,64] Hence, CMR is useful prognostically but cannot be practically performed in the acute postoperative period.

CORONARY ANGIOGRAPHY

In patients with suspected acute PMI, coronary angiography is the diagnostic gold standard. The indication for angiography should be defined in consultation with the entire heart team (ie, the surgeon, interventional cardiologist, and acute cardiac care intensivist or anesthesiologist) given all the above-mentioned diagnostic challenges. Different variables such as coronary anatomy, valvular and ventricular function, current hemodynamics, aberrant rhythm (ventricular tachycardia/fibrillation), type of cardiac surgery, presence of complications during surgery, and bleeding risks should be considered. A time interval of >30 hours between the index surgery and urgent angiography has been associated with higher in-hospital mortality and long-term mortality.[16] Therefore, clinicians should be encouraged to have a high degree of suspicion and a low threshold for early action.

TREATMENT

An algorithm for the treatment of acute PMI is proposed in Figure 2.

Mechanical Support

Patients with acute PMI and low cardiac output syndrome or cardiac arrest after cardiac surgery may require MCS to ensure stability and to treat the underlying ischemic mechanism. The first line of treatment for low cardiac output syndrome includes volume status optimization and vasoactive agents. In the short term, these strategies can improve cardiac output but at the expense of potential volume overload or an increase in cardiac oxygen consumption and have been associated with poor outcomes.[65] Therefore, the use of MCS should be considered in a timely fashion when such first-line treatment fails. Although there are no high-quality data on the optimal timing for MCS implantation, retrospective studies have suggested that intraoperative MCS implantation can improve outcomes compared with postoperative implantation.[66] The selection of MCS type depends on the underlying condition, patient characteristics, and center experience. In patients with mild to moderate LV dysfunction, temporary devices such as an intra-aortic balloon pump or a microaxial percutaneous LV assist device can be considered. For patients with severe LV, right ventricular, or biventricular dysfunction, venoarterial extracorporeal membrane oxygenation may be needed. However, extracorporeal membrane oxygenation is known to increase afterload, and LV venting may be required.[67] Ultimately, patients needing long-term LV, right ventricular, or biventricular support may benefit from central ventricular assist devices. In patients requiring MCS, careful consideration must be given to the risks and benefits of systemic anticoagulation. In high-risk patients, the simplest and lowest level of support device (intra-aortic balloon pump) may be selected because of the high bleeding risk. In certain patients, current MCS devices can also be used safely for a limited time without anticoagulation, allowing time for stabilization of the patient and further diagnostic workup and treatment while minimizing bleeding risk. Stabilization by MCS does not eliminate the urgent need to determine the underlying mechanism of acute PMI and address it.

Indications for Urgent Angiography

Retrospective studies have shown that urgent coronary angiography is safe and allows diagnosis of the underlying cause in most cases of acute PMI.[16,18] Indications for urgent angiography may include the following:

- Chest pain with ongoing signs of myocardial ischemia[1]
- New electrocardiographic changes
  - ST-segment elevation or depression
  - T-wave inversion, particularly deep or dynamic
  - New left bundle-branch block
  - New Q waves
- Cardiac imaging abnormalities
  - New regional wall motion abnormality
- Cardiac rhythm abnormalities
  - Ventricular fibrillation, cardiac arrest, or >1 episode of sustained ventricular tachycardia
- Low cardiac output syndrome despite typical postoperative pressor support
  - Mean arterial pressure <60 mm Hg, cardiac index ≤2 $L \cdot min^{-1} \cdot m^{-2}$, central venous oxygen saturation <60%, lactate >2 mmol/L, or urinary output <0.5 mL/kg per hour for 6 hours.[68]
- Significant elevation in cardiac biomarkers as detailed previously

Indications for Reoperation Without Angiography

In general, patients should not be taken to the operating room without a clear diagnosis of the cause of acute PMI. Reoperation without angiography leaves the surgeon essentially blind to the cause of the clinical problem and greatly limits treatment options. Two important exceptions to this rule are the inability to establish percutaneous mechanical support in patients in cardiogenic shock and the presence (or suspicion) of bleeding or mechanical surgical complications. In these scenarios, patients should ideally be taken to a hybrid operating room with imaging capabilities to allow repeat angiography once the patient has been stabilized.

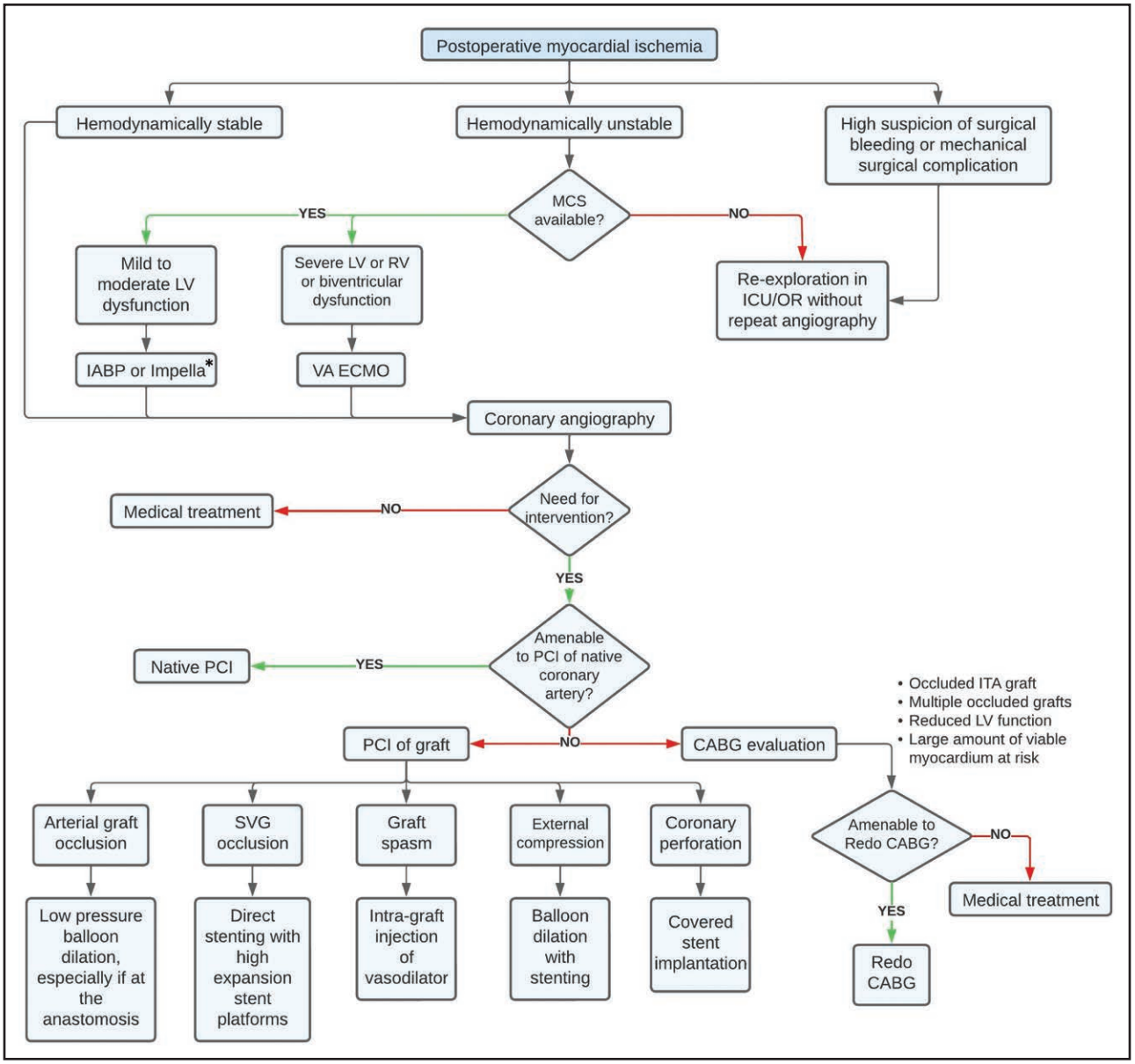

Figure 2. Algorithm for the treatment of postoperative myocardial ischemia after cardiac surgery.[4]
CABG indicates coronary artery bypass grafting; IABP, intra-aortic balloon pump; ICU, intensive care unit; ITA, internal thoracic artery; LV, left ventricular; MCS, mechanical circulatory support; OR, operating room; PCI, percutaneous coronary intervention; RV, right ventricular; SVG, saphenous vein graft; and VA ECMO, venoarterial extracorporeal membrane oxygenation.

*The device listed here serves only to illustrate an example of this type of device. This is not intended to be an endorsement of any commercial product, process, service, or enterprise by the American Heart Association.

Technical Details of Redo CABG

The key to the success of CABG for acute PMI is fast and effective reperfusion of the ischemic zone. Every consideration related to long-term outcomes (use of arterial grafts) or surgical technique (off-pump versus on-pump CABG) is secondary to a quick and effective re-establishment of the flow in the ischemic region. In general, the saphenous vein graft is the ideal conduit because of its almost universal availability, easy and fast harvesting, and anastomotic technical simplicity. Although there is no evidence that technical variations of the CABG procedure (sequential, composite grafts, type of cardioplegia) may affect clinical outcomes, surgeons should use their standard technique to minimize the risk of technical failures. In patients undergoing CABG, performing a new anastomosis distal to the previously implanted graft(s) is generally safer and more effective than redoing previous anastomoses, but comparative data are lacking.

Technical Details of Urgent PCI

The need for PCI after CABG is relatively low and is seen in 1% to 4% of patients undergoing CABG.[21] In patients who had CABG, the main indications for PCI are graft failure and incomplete revascularization.[21] In cases of graft failure, the

technique may vary and may include treatment of the native vessel, particularly if noncomplex, or of the graft. In the case of saphenous vein grafts, embolic protection devices may be considered if failure is due to graft thrombotic occlusion. Because of the larger lumen diameters and different tissue characteristics of saphenous vein grafts compared with arterial grafts, stent platforms with high expansion limits and direct stenting are preferred.[69] In arterial grafts, more commonly at the site of anastomosis of the left internal thoracic artery to the left anterior descending artery, a conservative approach with low-pressure balloon dilation rather than stenting should be considered (Table 3). In the case of proximal left subclavian artery stenosis, angioplasty and stenting can be performed.[69] When failure is related to graft spasm, intragraft vasodilators can be injected. PCI for incomplete revascularization is more frequently performed by native vessel treatment[16] and may include extra support techniques and calcium modification devices.

In patients who underwent valvular surgery, the underlying mechanism can be related to coronary entrapment by a suture, coronary perforation, endothelial damage–causing thrombosis, vascular distortion, or external compression.[70] In the case of an encircling suture, wiring can be challenging, and balloon dilation with stenting is often necessary, whereas in the case of perforation, stenting with a covered stent can be performed. Moreover, balloon dilation with or without stenting can be considered if the mechanism is related to endothelial lesion and thrombosis. Intracoronary imaging (eg, intravascular ultrasound and optical coherence tomography) can be useful for identifying the underlying mechanism and for PCI optimization.

Even more than the technical challenges of PCI, there are tremendous challenges with adjunctive pharmacology given the high bleeding risks in the early postoperative period, inability for oral drug intake or impaired gastrointestinal absorption, and even more acute problems with bleeding when an evolving shock or a hemodynamically unstable condition is present. The use of reversible intravenous agents, for example, unfractionated heparin, can be empirically advisable; these infusions may have to continue until intake of medications with predictable absorption is possible.

OUTCOMES

Short-Term Outcomes

Short-term postoperative morbidity and mortality are higher among patients with acute PMI. For patients undergoing CABG, 30-day mortality rates among acute PMI patients range from 5.1% to 24%, with a meta-analysis of 9 studies showing 8.9% mortality (95% CI, 6.7%–11%),[28] which was substantially higher than rates for patients without PMI (1.8%).[71] The increased mortality associated with acute PMI does not significantly differ by treatment modality (12% for PCI, 20% for redo CABG, and 14.8% for conservative therapy).[4] Patients with acute PMI also have higher rates of kidney injury and stroke ($P<0.001$),[23] with an acute decline in LV ejection fraction that appears to recover at follow-up ($P<0.001$).[7]

Long-Term Outcomes

Longer-term data on outcomes after acute PMI are variable.[72] One study found a significantly higher 5-year mortality rate after acute PMI (86% for PMI versus 75%; $P<0.001$), but after the impact of in-hospital mortality was removed, the difference was no longer significant ($P=0.09$). More recently, a retrospective study of >50 000 patients showed progressively worse outcomes depending on the presentation of acute PMI, with patients presenting with postoperative ST-segment–elevation myocardial infarction/non–ST-segment–elevation myocardial infarction having the lowest 1-year mortality (13.5%), those with ventricular tachycardia/fibrillation having worse mortality (28.1%), and those with hemodynamic instability having the worst mortality (38.1%; $P=0.001$ for all). Notably, peak cTn level was predictive of 1-year mortality (area under the curve, 0.74 [95% CI, 0.65–0.84]; $P<0.001$) but did not predict abnormal coronary findings at angiography. Those who underwent PCI did better than those who received either redo CABG or conservative therapy, although a treatment allocation bias is likely.[25]

Disparities in Outcomes

Disparities in outcomes may relate to preoperative characteristics that have been associated with increased risk of acute PMI after CABG, for example, smaller coronary anatomy among female patients.[73,74] Although there are limited data quantifying differences in acute PMI by race and ethnicity, a report from the Society of Thoracic Surgeons database demonstrated significantly worse 30-day mortality among Black patients compared with White patients, with multiple studies highlighting the substantial contribution of hospital quality to these disparities.[64,75,76] Challenges associated with providing timely and appropriate treatment for acute PMI at lower-quality hospitals may stem from lower nursing ratios and constrained resources, as well as clinician quality and mistrust.[77] Urgent attention to ensuring prompt detection and treatment of acute PMI at all hospitals offers an impactful target for reducing disparities in outcomes after cardiac surgery.

SUMMARY AND CONCLUSIONS

Acute PMI is an uncommon but potentially serious complication of cardiac surgery that may have different causes. It is important to maintain a high index of suspicion for acute PMI in all patients undergoing cardiac surgery because timely diagnosis and treatment are key to a good clinical outcome. A high level

Table 3. Causes of Acute Postoperative Ischemia and Indications for PCI After Cardiac Surgery

Surgery type	Cause	Technical considerations
CABG surgery		
Graft failure		If the native vessel has noncomplex anatomy, consider PCI of the native vessel.
Arterial graft		
LITA to LAD	Thrombotic or technical occlusion at the site of the anastomosis	Low-pressure balloon dilation preferred over stenting
Proximal left subclavian artery stenosis	Thrombotic occlusion at the site of the anastomosis with or without calcium	Angioplasty and stenting
Graft spasm	High catecholamines or inflammatory levels and physical manipulation of the graft	Intragraft vasodilators
Venous graft	Proximal thrombotic occlusion	Consider the use of embolic protection devices. Stent platforms with high expansion limits and direct stenting are preferred.
Incomplete revascularization	More common in nondominant diseased RCA, a small or severely calcified target vessel	PCI of the native vessel Extra support techniques and calcium modification devices may be necessary.
Non–graft related	New coronary artery occlusion	If the vessel has noncomplex anatomy, consider PCI of the vessel.
Valvular surgery	More common in aortic and mitral surgery, but there are described cases of tricuspid and less frequently pulmonary valve	According to the underlying mechanism Intracoronary imaging can be useful for stent optimization.
Aortic valve	Thrombus and calcium embolization; left main or proximal LAD or LCx involvement or both	Prompt flow restoration with balloon angioplasty If thrombus is present, aspiration can be attempted. If calcium, stenting should be considered.
Mitral and tricuspid valve	Coronary entrapment by a suture, coronary perforation, endothelial damage–causing thrombosis, tissue retraction, and vascular distortion and external compression	Mitral valve anatomy is related to the LCx and tricuspid to RCA. Wiring can be challenging. If feasible, stent implantation is reasonable to use. In case of perforation, covered stents are preferred.
Aortic root surgery with coronary reimplantation	Damage during instrumentation and delivery of cardioplegia and entrapment with sutures to the coronary button Distortion of the left main/RCA in aortic root surgery from suboptimal ostial implantation	Prompt flow restoration with balloon angioplasty and stenting Intraoperatively, consider surgical revision.
Ablation of arrhythmias	Transient injury or spasm (occasionally permanent injury) of the RCA from radiofrequency or cryoablation lesions in the right atrium from the ablation lines to the tricuspid valve annulus for atrial fibrillation	Prompt flow restoration with balloon angioplasty and stenting

CABG indicates coronary artery bypass graft; LAD, left anterior descending artery; LCx, left circumflex artery; LITA, left internal thoracic artery; PCI, percutaneous coronary intervention; and RCA, right coronary artery.

of collaboration among intensivists, surgeons, cardiologists, imaging experts, and the postoperative team at large is required.

The current evidence on acute PMI after cardiac surgery is sparse and generally of low methodological quality. These observations underscore the urgent need for rigorous studies to define optimal diagnostic criteria and treatment algorithms.

ARTICLE INFORMATION

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

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*Modest.

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*Modest.

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