SCAI Consensus Statement on Best Practices for the Cardiac Catheterization Laboratory Management of Patients with Suspected STEMI Referred for Primary PCI

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INTRODUCTION

ST-elevation myocardial infarction (STEMI) is a cardiac emergency that requires rapid diagnosis and timely treatment, with primary percutaneous coronary intervention (PCI), recognized as the preferred mode of reperfusion [1]. Over the last three decades there have been considerable advancements in the treatment of patients with STEMI, beginning with fibrinolytic therapy, the implementation of primary PCI, and subsequent focus on improving reperfusion times through the coordination of systems of care [2]. The American College of Cardiology (ACC) and American Heart Association (AHA) publish clinical practice guidelines that address the management of STEMI [3, 4]. While these guidelines provide a robust and well-established clinical framework to manage patients with STEMI that is largely informed by randomized clinical trials and meta-analyses, they are not designed to address the procedural and technical aspects of the management of STEMI, nor the implementation of newer approaches. Likewise, the evidence-base informing the guidelines are primarily derived from patients with suspected atherosclerotic plaque disruption (type 1 MI), and therefore are often unable to guide clinicians on specific circumstances such as the management of patients with non-atherosclerotic etiologies of STEMI. The purpose of this consensus statement is to provide a summary of best practices for managing patients with STEMI, focusing on the cardiac catheterization laboratory (CCL) management and the technical aspects of the procedure, and address special circumstances, anatomical subsets, and non-atherosclerotic causes of STEMI.

METHODOLOGY

This statement has been developed according to the Society for Cardiovascular Angiography & Interventions (SCAI) Publications Committee policies for writing group composition, disclosure, and management of relationships with industry, internal and external review, and organizational approval [5].

The writing group was organized to ensure diversity of perspectives and demographic characteristics and appropriate balance of relationships with industry. Relevant author disclosures are included in (Supplementary Table S1). Before appointment, members of the writing group were asked to disclose financial and intellectual relationships from the 12 months prior to their nomination. A majority of the writing group disclosed no relevant, significant financial relationships. The work of the writing committee was supported exclusively by SCAI, a nonprofit medical specialty society, without commercial support. Writing group members contributed to this effort on a volunteer basis and did not receive payment from SCAI.

Literature searches were performed by group members designated to lead each section, and initial section drafts were authored primarily by the section leads in collaboration with other members of the writing group. Consensus statements on the various aspects of CCL management were discussed and agreed upon by the full writing group using a modified Delphi process, which required 75% agreement among authors for a consensus. The draft manuscript was peer reviewed via a public comment period in ____ 2024, and the document was revised to address pertinent comments. The writing group unanimously approved the final version of the document. The SCAI Publications Committee and Executive Committee endorsed the document as official society guidance in ____ 2024.

The SCAI statements are primarily intended to help clinicians make decisions about treatment alternatives. Clinicians also must consider the clinical presentation, setting, and preferences of individual patients to make judgments about the optimal approach.

CARDIAC CATHETERIZATION LABORATORY TEAM READINESS AND INITIAL ASSESSMENT

STEMI systems of care must accomplish rapid transition of acutely ill patients from the field to the CCL [6]. Prompt treatment is associated with improved clinical outcomes [7] and time represents an overall priority to streamline care at every level of the STEMI system (Figure 1). Hospitals should develop and maintain protocols and quality-improvement assessments to achieve these goals, as STEMI teams are often activated outside of usual working hours (Table 1). Pre-hospital activation of the STEMI team by the Emergency Medical Services (EMS) or the emergency department (ED) allows team members time to prepare the CCL during working hours, and time to travel to the hospital after working hours. This has been associated with improved time-to-treatment and outcomes [8]. A single page and/or telephone call activation to a list of STEMI team members is efficient as a mechanism to alert the proper parties, either through the hospital operator or via an electronic application-based system [9, 10]. For transferred patients, location and estimated time of arrival are valuable information to include in the communication. Transmission of the 12-lead electrocardiogram (ECG) is helpful to allow the team an opportunity to review the findings, concur with the diagnosis, and anticipate potential diagnostic or therapeutic interventions [11]. ECG transmission may also alert the team about cases with equivocal ECG findings or uncertain clinical presentations that may require clinical evaluation prior to CCL transfer. While ECG transmission to the CCL team members can be

useful, transmission should not delay the patient's transportation. CCL team members should be expected to arrive at the CCL within 30 minutes and ideally 20 minutes after notification.

Once a STEMI is confirmed on the initial ECG and there are no circumstances precluding emergent revascularization, the patient should be directly transported to the CCL. All patients should be loaded with aspirin, a P2Y12 inhibitor, and given a bolus of intravenous heparin, followed by direct transfer to the CCL. When feasible, patients presenting via EMS should go directly to the CCL, bypassing the ED. This process known as "ED bypass" has been associated with shorter times to treatment and better outcomes [12]. The definition of ED bypass varies across centers. In some institutions, the ED is literally bypassed, and the patient is brought straight to the CCL from the ambulance. Alternatively, some centers make a brief stop in the ED for registration, identification of a proxy and a brief review of the ECG and to ensure the patient has a working IV. In all cases, transport of the patient to the CCL should occur once laboratory staff are prepared to receive the patient. Unstable patients may require further stabilization, including intubation and mechanical ventilation in the ED prior to transport to the CCL.

Some patients with acute coronary occlusion do not always manifest ST elevation on ECG (i.e. acute left circumflex occlusion) or may have subtle ST elevation not meeting the defined diagnostic criteria for STEMI. When there is a high index of suspicion, the acquisition of additional ECGs including leads V7-V9, or the use of bedside point of care ultrasound (POCUS) can be helpful. In the appropriate clinical setting, patients with ECG features concerning for an acute coronary occlusion but not meeting the STEMI criteria may require the same management pathway as patients with definite STEMI.

While time to reperfusion is essential, this should not preclude a careful evaluation including a focused history and physical exam, as some concomitant conditions or situations may

warrant specific precautions (e.g.: severe peripheral vascular disease, prior coronary artery bypass graft surgery, use of oral anticoagulants, acute or chronic kidney disease, severe aortic stenosis, pregnancy), or influence the decision to proceed with an invasive approach (e.g.: stroke or altered mental status, severe anemia, advanced age, code status and advanced directives) or prompt additional interventions to stabilize the patient before proceeding with PCI (e.g.: respiratory failure requiring airway protection and/or intubation, unstable arrhythmias requiring treatment, cardiogenic shock (CS)). Caution is warranted in late presenters (>12 hours) who are at risk for mechanical complications, in which case, prompt POCUS can facilitate evaluation. Importantly, in cases of futility (e.g. advanced dementia and/or advanced directives against resuscitation), discussion with the patient and/or family members is critical to ensure patient goals of care are respected prior to the procedure.

A detailed evaluation is also important in cases with equivocal clinical presentation or uncertain ECG findings which can lead to false STEMI activations due to interpretation errors, technical issues, or STEMI mimics (such as pericarditis or myocarditis, early repolarization, Brugada syndrome, altered electrolytes, Takotsubo, or ventricular aneurysms). This can result in potentially unnecessary invasive procedures and complications, as well as delays to treatment of other conditions requiring timely management [13]. A high rate of false activations can also have downstream consequences with respect to availability of STEMI staff for other simultaneous activations, resource utilization including risk of staff fatigue and burnout, financial costs, and time management. False activation rates can be reduced with educational efforts and training on ECG interpretation. In unclear clinical scenarios, POCUS may enhance clinical decision making.

Consensus Key Points Regarding Cardiac Catheterization Laboratory Team Readiness

- **Prehospital notification and ECG transmission streamlines care.**

- **When feasible ED Bypass should be implemented.**
- **A focused history and physical exam should be performed by a member of the cardiovascular team.**

CARDIAC CATHETERIZATION LABORATORY EQUIPMENT

Hospitals offering primary PCI should ensure that the CCL is equipped with the necessary tools required to complete angiographic assessment and facilitate PCI. Prior statements have outlined the minimal procedural requirements and qualifications of CCL staff to perform PCI [14], but do not focus on necessary equipment to care for patients with STEMI. While there may be some differences in operator preferences, there are key categories of devices (beyond the standard PCI equipment) that should be available for any CCL offering primary PCI (Table 2).

OPTIMAL TECHNIQUES FOR ANGIOGRAPHY AND INTERVENTION

Arterial Access

Transradial access is endorsed as a class I recommendation in preference to femoral access to reduce the risk of access-site bleeding, vascular complications, and death [1]. Radial access is an important bleeding avoidance strategy for high-risk patients such as those who have received thrombolytics, glycoprotein IIb/IIIa receptor inhibitors (GPI), or are on oral anticoagulants [15]. Radial access should be the preferred approach in STEMI (Figure 2). When performing radial artery access, careful attention to procedural technique is needed to allow for timely access, reduce complications, and maintain the artery for future access [16] (Table 3). Femoral access represents an alternative access route that should be reserved for patients in whom the radial artery cannot be used due to technical, clinical, or anatomical reasons, or cases in which a femoral approach may be preferred such as in patients with CS who require large bore

mechanical circulatory support device or those with prior coronary artery bypass graft (CABG) surgery in whom a left radial approach may not be facile in the acute setting. Optimal access technique in the common femoral artery is associated with lower risk of bleeding [17, 18], and requires certain key steps (Table 3). Irrespective of the access route, ultrasound guidance is a key component of contemporary vascular access techniques that is beneficial to reduce the number of attempts and time to access. Its use in both radial and femoral access is supported by randomized trials and several meta-analyses [19, 20].

Consensus Key Points Regarding Arterial Access

- **Transradial access is the preferred route for coronary angiography and PCI**
- **When femoral access is necessary, use of contemporary techniques including the routine ultrasound and fluoroscopy are advised.**

Diagnostic Assessment

Interventional cardiologists often have different styles for performing diagnostic angiography and PCI that may have advantages and disadvantages. Complete angiography of both the left and right coronary systems, as well as bypass grafts (if present), should be routinely performed. The timing of complete angiography (ie before or after PCI) will depend on the clinical circumstances and operator preference. Performing complete diagnostic angiography prior to PCI helps identify the culprit lesion in cases in which the infarct vessel is ambiguous, determines the extent and severity of non-infarct artery disease, allows for assessment of collateral blood flow, and may help inform guide selection. Alternatively, when the ECG is non-ambiguous, an upfront guide catheter to assess the coronary circulation and facilitate PCI, with subsequent completion of diagnostic angiography after the PCI can reduce treatment delays [21]. In most cases, the added knowledge of the non-infarct anatomy is often unlikely to alter the

revascularization approach. One caveat to this approach is that in RCA-territory STEMI, initial angiography of the left system may identify critical left main or multivessel CAD that can influence the approach to PCI of the infarct artery. In patients with hemodynamic or electrical instability or with CS a full angiogram before proceeding with PCI may be prudent irrespective of the infarct artery location.

Measurement of left ventricular end diastolic pressure (LVEDP) offers an assessment of left-filling pressure and can serve as a useful guide to intra-procedural and post-procedural volume management, as well as provide objective information to help with decisions surrounding the need for diuresis, afterload reduction, or mechanical circulatory support (MCS). An elevated LVEDP is associated with higher short and long-term mortality and the development of congestive heart failure [22]. Instrumentation of the left ventricle (LV) should be avoided in late presenters who are at risk for LV thrombus unless previously excluded. The value of LV angiography in contemporary practice is uncertain, particularly with the wider availability of POCUS. However, it can be useful in cases of CS if a mechanical complication is suspected, or when there are ambiguous clinical circumstances where the infarct artery is uncertain. A left ventricular angiogram can also help establish a diagnosis in patients with non-obstructive or normal coronaries including stress cardiomyopathy.

Consensus Key Points Regarding Diagnostic Angiography

- **A complete diagnostic coronary angiogram should be performed during the index procedure.**
- **Measurement of LVEDP can help guide further treatment.**

MANAGING THROMBUS

Intracoronary thrombus can lead to distal embolization with resultant microvascular obstruction (MVO), impaired tissue perfusion (no reflow), and occasional terminal vessel truncation. Intracoronary thrombus may also result in side-branch compromise during PCI and has been associated with late stent malapposition [23]. The most widely accepted and thorough classification of intracoronary thrombus is the Thrombolysis in Myocardial Infarction (TIMI) thrombus grade (Supplemental Table S2) [24]. Practically, thrombus can be classified as either large thrombus (grade 4-5) or small/no thrombus burden (grade 0-3), as this more specifically informs subsequent steps in PCI. Given that an initial TIMI Thrombus Grade 5, defined as a complete vessel occlusion, may rapidly improve to Grade 0 or Grade 1 once the guide wire has crossed the lesion (or with "balloon dottering"), the determination of thrombus grade that will dictate management should not be made until after the guidewire has crossed the occlusion.

The strategy of thrombus management (Figure 3) invariably begins with attempts to re-establish flow. After initial wiring, a deflated compliant balloon may be passed back and forth across the lesion to re-establish flow. This is referred to as "balloon dottering" and is beneficial as it may allow distal vessel visualization to confirm distal wire position prior to balloon dilatation. Following initial angioplasty, repeat angiography and reclassification of TIMI flow and thrombus burden can then guide the next steps. If the TIMI flow is ≥ 2 and there is absence of a large residual thrombus with adequate visualization of the distal vessel, then PCI can follow. Direct stenting without initial balloon angioplasty is a reasonable strategy but should ideally be guided by intracoronary imaging prior to stenting. In the absence of significant calcification, a strategy of direct stenting is associated with lower corrected TIMI frame count, and a greater degree of ST segment resolution, in addition to shorter procedure time and contrast utilization, and improved medium term outcomes [25, 26]. An important exception to direct stenting is when

significant calcium is identified either fluoroscopically or via intracoronary imaging or when there is tortuosity in the vessel requiring lesion modification prior to stenting.

If after initial wiring and passing of a deflated balloon, there is TIMI ≤1 flow, or there is a large thrombus burden, additional thrombus management is needed prior to stenting. Available options can be categorized as those that exclude, extract, or dissolve intracoronary thrombus. Thrombus extraction with manual and mechanical aspiration thrombectomy are the most employed treatment strategies for large thrombus burden. Initial trials of manual aspiration thrombectomy demonstrated improved TIMI flow, and myocardial blush grade and improved clinical outcomes with thrombus aspiration [27, 28]. However, the larger Thrombus Aspiration during ST Segment Elevation Myocardial infarction (TASTE) trial [29] and the Trial of Primary PCI with or without Routine Manual Thrombectomy (TOTAL) [30],which collectively enrolled over 18,000 patients, failed to show a reduction in all-cause mortality or cardiovascular events with thrombus aspiration. Additionally, TOTAL reported higher rates of stroke in the group of patients randomized to thrombus aspiration. An individual patient level meta-analysis confirmed these findings with no difference in cardiovascular death but a strong trend toward a higher rate of stroke [31]. In this report, a subgroup analysis of endpoints demonstrated a consistent "lack of benefit" with thrombus aspiration irrespective of thrombus size. However, there was a higher stroke risk with thrombus aspiration only in the patients with a large thrombus burden. Current guidelines recommend against the *routine* use of thrombus aspiration in STEMI [1]. This recommendation does not apply to patients with a more extensive thrombus burden in whom there may be concern for distal embolization, or when balloon angioplasty is unsuccessful, in which case bail-out thrombus aspiration may be needed.

The Penumbra CAT Rx device is a newer device that allows continuous mechanical aspiration that can be used in cases with large intracoronary thrombus. While a prospective registry of patients with large thrombus burden demonstrated excellent TIMI 3 flow on final angiogram and stroke rates comparable to the control arm of other studies [32], randomized data is not available and therefore a direct comparison of the various aspiration devices cannot be made. Regardless of the device used, optimal thrombectomy technique is imperative and includes avoidance of thrombectomy in severely tortuous arteries, active thrombectomy with initial antegrade advancement, deep seating of the guiding catheter upon withdrawal of the device to minimize the chance of dislodgement of thrombus down a side branch or into the aorta, continuous active aspiration until just before the catheter enters the hemostatic valve at the hub of the guiding catheter (so as not to entrain air) , and "back bleeding" of the hemostatic valve followed by rigorous flushing to ensure any residual thrombus remaining in the catheter has been removed.

For cases with large residual thrombus that persist despite usual measures including thrombectomy, the approach to treatment is more nuanced. The use of GPI either IV or intracoronary, and the use of intracoronary fibrinolytic agents can be considered as adjunctive therapy. In theory, the antiplatelet effects of a GPI may be potentiated by local drug delivery via the intracoronary route resulting in relatively higher concentrations of drug at the site of an occlusive thrombus, however, trials examining intracoronary GPI as compared with IV GPI during PCI for STEMI have reported conflicting results and a meta-analysis reporting on 14 trials of intracoronary GPI, enrolling 3,754 patients showed no difference in long term major adverse cardiovascular events (MACE) [33]. In this study, the use of intracoronary GPI, was associated with improved markers of reperfusion including ST segment resolution, myocardial

blush grade, and infarct size, as well as reduced short-term MACE. Intracoronary fibrinolytic studies have been limited to case reports or series, or smaller trials performed in the lytic era, with absence of contemporary data from randomized trials [34]. Intracoronary GPI or fibrinolytic agents are generally reserved for situations when there is significantly large thrombus or no-reflow despite usual aspiration techniques. If used, delivery of the drug directly to the distal artery using micro-catheters can be useful.

In some cases, when flow has been reestablished but there is a residual large thrombus burden that persists despite the techniques described above, deferral of stenting while treating patients with prolonged intravenous anti-platelet and anti-thrombotic therapy is an alternative strategy to reduce no-reflow risk and infarct size, with planned repeat coronary angiogram and potential staged PCI after the antithrombotic therapy course has been completed. A small, randomized trial demonstrated improved coronary blood flow with this technique [35], but a larger clinical trial and meta-analysis did not report a reduction in clinical events with deferred stenting [36, 37].

Consensus Key Points on Managing Thrombus

- **Angiographic assessment of thrombus burden should be made after the wire crosses the lesion.**
- **Bail-out thrombectomy in selected cases of large thrombus is an acceptable treatment strategy.**
- **Parenteral (or intracoronary) anti-platelet or anticoagulants can be used for refractory thrombus**

MANAGING NO-REFLOW

Coronary no-reflow during primary PCI, defined as the lack of antegrade coronary flow in the absence of epicardial obstruction, is a frequent challenge that is associated with larger

infarct size and increased long term mortality [38]. The primary basis for no-reflow is MVO. The pathologic mechanism underlying MVO includes individual susceptibility, ischemia/reperfusion injury, endothelial dysfunction, microvascular spasm and distal thrombo-embolization [39]. Important risk factors include advanced age, male sex, longer ischemic times, hyperglycemia, leukocytosis, elevated creatinine, and elevated cardiac biomarkers at baseline. Angiographic and hemodynamic predictors include initial TIMI flow, high thrombus burden, and higher Killip class [40].

Prevention and early recognition are the foundations of management of no-reflow. Systems that promote early presentation after symptom onset and shorter ischemic times would likely have a favorable effect on the incidence of no-reflow. Acute glycemic control and pre-procedural statin use have been shown to decrease the incidence of no-reflow, presumably by mitigating endothelial dysfunction and ischemia/reperfusion injury [41], (although pre-procedural glycemic control may not be feasible in cases of primary PCI). Additionally, earlier pre-treatment with oral P2Y12 inhibitors is important as it has been associated with improved pre-procedural TIMI flow [42]. From a technical standpoint, the best treatment of no-reflow is also prevention. Thus, careful assessment for thrombus after initial wiring, and maneuvers to treat thrombus, (see Section *Managing Thrombus*) are associated with greater myocardial blush, and ST segment resolution, which are clinical markers of tissue level reperfusion [27, 33, 35].

Coronary perfusion pressure is an often unappreciated, yet critically important variable contributing to no-reflow. Augmentation of cardiac output and active reduction of LV end diastolic pressure, with afterload reduction, diuresis and at times with the use of MCS can aide in the prevention of no reflow, particularly in larger anterior infarcts [43, 44]. Maximization of distal capacitance with the prophylactic use of intracoronary vasodilators, including adenosine,

nitroprusside, and verapamil, prior to stenting is another important strategy. When used for prophylaxis, these medications should be given before and after each coronary manipulation (i.e. thrombectomy, angioplasty and/or stenting), and may be given via the guide catheter, a microcatheter, or aspiration catheter.

Once no-reflow is noted, the goal of therapy should focus on reversing vasoconstriction and treatment of microvascular thrombosis. Therefore, delivery of medications to the distal coronary bed is necessary. In addition to their prophylactic role, intracoronary vasodilators remain the mainstay of treatment once no-reflow occurs, although evidence is limited. The most employed agents include adenosine, nitroprusside, calcium channel blocking agents, and epinephrine (Table 4). Most of these agents are used anecdotally with limited and conflicting results from clinical studies. Factors to consider before choosing an agent include LV function, LVEDP, mean arterial blood pressure, the presence of conduction delays or significant bradyarrhythmias, and the presence of significant aortic stenosis, or obstructive hypertrophic cardiomyopathy.

Consensus Key Points for Prevention and Management of No-Reflow

- **Pre-procedural loading of P2Y12 agents and high dose statins, and glycemic control are useful options to reduce the likelihood of no reflow**
- **Use intracoronary arteriolar vasodilators to prevent and manage no reflow.**
- **Maximize coronary perfusion pressure with augmentation of mean arterial pressure and reduction of LVEDP, to treat no-reflow.**

INTRACORONARY IMAGING

Intracoronary imaging with intravascular ultrasound (IVUS) or optical coherence tomography (OCT) is recommended as a reasonable option to guide PCI, and in patients with stent thrombosis or in-stent restenosis to assess the mechanism of stent failure [1, 45].

Randomized trials and meta-analyses have demonstrated a reduction in MACE, stent thrombosis, cardiac death, and all-cause mortality in longer term follow-up with the use of intracoronary imaging [46-49]. While robust data supports the use of intracoronary imaging, few patients in these trials were undergoing PCI for STEMI. Registry data comparing intracoronary imaging to angiography-guided PCI in patients with acute myocardial infarction (AMI), including approximately 50% with STEMI, have demonstrated lower MACE and lower adjusted mortality with intracoronary imaging [50, 51]. Future RCTs examining the benefits of IVUS in STEMI patients undergoing PPCI are planned (NCT04775914) and will provide important insight for STEMI patients.

Prior to stenting, intracoronary imaging can play a critical role in deciphering ambiguous culprit lesions in STEMI or to assess the mechanisms of stent thrombosis (Figure 4). In these situations, OCT may be preferred as it provides greater resolution than IVUS, though at the expense of additional contrast use. When feasible, imaging acquisition should be performed before and after stent placement (Figure 5). Morphology, including the assessment of plaque burden, areas of positive remodeling, thrombotic burden and the extent of calcium may influence the approach to PCI. Distal and proximal reference vessel cross-sectional diameters and lesion length are important variables necessary for optimal stent sizing. After intervention, the focus of intracoronary imaging is to assess stent expansion, apposition, geographic miss, and edge dissection.

Consensus Key Points for Intracoronary Imaging

- **Routine use of intracoronary imaging is encouraged to guide PCI.**
- **When there is stent thrombosis or stent failure, leading to STEMI intracoronary imaging should be performed to investigate the mechanism.**
- **Intracoronary imaging can be helpful in assessing ambiguous lesions and delineating the mechanism of STEMI.**

EMERGING APPROACHES TO REDUCE INFARCT SIZE

There are numerous efforts aimed at reducing MVO, reperfusion injury, infarct size, and minimize scar [52]. Several studies have explored various methods at reducing reperfusion injury or infarct size [53-58] but there are limited data to support any of the approaches as a routine first line therapy for STEMI.

Supersaturated oxygen (SSO2) therapy was FDA approved in 2019 to reduce infarct size in patients with anterior STEMI treated with primary PCI within 6 hours of symptom onset, in the absence of shock [59]. The catheter-based system allows for blood that is withdrawn from the side arm of a sheath to mix with aqueous oxygen (via an oxygenator) to provide high levels of dissolved oxygen to the "at-risk" myocardium limiting microvascular and myocardial damage. Studies have shown an average reduction in infarct size of 28% with SSO2 [55, 60] and low rates of net adverse clinical events including death, reinfarction, clinically driven target vessel revascularization, stent thrombosis, and severe heart failure as compared to historical controls [61]. While early iterations used larger femoral sheaths that were associated with more bleeding complications, the latest system allows the use of a smaller 5 French contralateral sheath. Post marketing surveillance studies are ongoing and aim to evaluate outcomes in a larger subset of patients receiving SSO2.

Mild hypothermia (32.0°-35.9°C) reduces reperfusion injury by blunting inflammation, thrombosis, and myocardial metabolism[62, 63]. RCTs with varying protocols for cooling have investigated adjunctive hypothermia as a strategy to decrease infarct size and preserve microvascular integrity [57, 64-68]. While most of the studies demonstrated that cooling was safe, the efficacy endpoints have been largely negative, although there was some suggestion of

benefit in patients with anterior infarct [65, 67]. The European Intracoronary Cooling Evaluation in Patients With ST-Elevation Myocardial Infarction (EURO-ICE) trial is an ongoing clinical trial that will randomize 200 patients with an anterior infarct and TIMI 0 or 1 flow in the infarct artery to a strategy of intracoronary cooling during PCI or PCI alone [69]. The primary endpoint is infarct size as a percentage of LV mass on MRI at 3 months.

Left ventricular unloading is another approach aimed at reducing infarct size. The Door-to-Unload (DTU)-STEMI trial randomized 50 patients with anterior STEMI to a protocol of 30 minutes of LV unloading with the Impella CP (Johnson & Johnson, New Brunswick, NJ) and delayed reperfusion vs immediate primary PCI without unloading [58]. The study was designed as a feasibility trial, with a focus on safety, and demonstrated no differences in MACE or major cerebrovascular events groups between the groups. The STEMI-Door to Unload (STEMI-DTU) trial aims to enroll 668 patients with an anterior infarct and randomize them to a strategy of LV unloading 30 minutes before PCI or immediate PCI without LV unloading and will assess the effects of LV unloading on myocardial infarct size measured with MRI at 3-5 days [70].

SPECIAL CIRCUMSTANCES

Cardiogenic Shock

Patients with CS are at significantly higher risk of mortality which is reduced by prompt revascularization as demonstrated in the Should We Emergently Revascularization Occluded Coronaries for Arteries for Cardiogenic Shock (SHOCK) trial [71]. Emerging data suggests improvement in mortality from CS when algorithmic risk-stratification and treatment protocols are in place [72]. Designating regional centers by level may be useful to facilitate interhospital transfer or EMS triage for primary PCI in the context of CS [6]. This designation as outlined by

the American Heart Association STEMI Systems of Care Policy Statement [6] provides three levels of care for STEMI: Level 1 centers provide a complete range of treatment for STEMI and shock including MCS and surgical support when needed. Level 2 centers provide primary PCI and some level of MCS. Level 3 centers are non-PCI hospitals that can administer fibrinolytic therapy or provide rapid transfer to a PCI hospital for primary PCI. While this designation facilitates triage, it is uncertain whether this case allocation improves outcomes and is not always used in practice.

The management of STEMI complicated by CS requires thoughtful decisions to optimize care and prevent bleeding and vascular events while the patient is in the CCL and subsequently, in the critical care units. A detailed discussion surrounding the assessment and management of CS in the catheterization laboratory is described elsewhere [73, 74]. Notable aspects of catheterization lab management in patients with STEMI complicated by CS are summarized below.

POCUS can be useful in patients with CS to aid in determining the etiology, particularly in patients with delayed presentation. The evaluation should focus on an assessment of biventricular systolic function, and the presence of significant valvular disease, intracardiac shunts, or pericardial effusion. When feasible, a POCUS should be done prior to angiography and is best performed during the time when the CCL is preparing for the patient so that it can provide important information before the start of the procedure while not delaying the time to PCI.

In the absence of known or suspected LV thrombus, LVEDP should be routinely measured in cases of CS (unless right heart catheterization measurements are available at the time of the procedure) as it may help guide management. Additionally right heart catheterization

with measurement of pulmonary capillary wedge pressure, pulmonary artery pressures, cardiac output and index, and calculation of pulmonary artery pulsatility index and cardiac power index can help guide the need for, and type of, MCS.

For patients with SCAI Stage C or D shock an MCS device can help reduce ventricular workload and, in some cases, can allow for ventricular unloading before PCI. The Danish-German Cardiogenic Shock (DAN-GER SHOCK) trial [75] randomized 355 patients with STEMI and SCAI stages C through E CS to a strategy of MCS support using the microaxial flow pump (Impella-CP) or standard care. Approximately 50% of patients received the Impella-CP before PCI. The primary endpoint, death from any cause was lower in the Impella-CP group as compared to standard care (45.8% vs 58.5%, p=0.04). Notably there was a significantly higher rate of adverse events including moderate or severe bleeding, limb ischemia, need for renal replacement therapy and sepsis in the Impella-CP group. These results provide encouraging support for using the Impella-CP in CS but emphasizes the need for careful selection and the use of safe femoral access, and protocols for care to minimize adverse events.

STEMI Treated with Fibrinolytic Therapy

If primary PCI cannot be performed within 120 minutes of first medical contact, fibrinolytic therapy is recommended [3], but this approach carries important limitations including failure of reperfusion or the potential for re-occlusion. For this reason, after treatment with fibrinolytic therapy, current guidelines recommend early transfer to a PCI hospital [1]. Patients with CS, decompensated heart failure, and failed reperfusion (defined by a lack of ST resolution >50% and absence of reperfusion arrhythmias) should undergo immediate angiography and revascularization [1]. In the remaining patients, it is reasonable to transfer with the plans for

coronary angiography and PCI within 3-24 hours after fibrinolysis [1]. In these situations, radial access is valuable to mitigate access site related bleeding complications.

Stent Thrombosis

Approximately 1% of patients experience stent thrombosis in the first 2 years after PCI [76]. Most patients with stent thrombosis present with STEMI. Premature discontinuation of DAPT and stent related factors such as stent under-expansion, stent fracture, mal-apposition or edge dissection, stent gap, residual uncovered plaque, and stent under sizing, are the most common risk factors for stent thrombosis (Figure 4). The management of stent thrombosis has been described [77]. Notable aspects of management of STEMI due to stent thrombosis include the use of intracoronary imaging (OCT or IVUS) to determine the cause of stent thrombosis. In these cases, the higher resolution of OCT may provide better assessments of uncovered struts, mal-apposition, stent fracture, and neo-atherosclerosis. The approach to intervention will depend on the mechanism of stent thrombosis. If under-expansion or mal-apposition is noted, then high pressure balloon inflation with appropriate sized balloon based on intracoronary imaging may be all that is needed. On the other hand, for stent fractures, edge dissections or neo-atherosclerosis, a second drug eluting stent is often warranted. Stent thrombosis frequently presents with large thrombus burden and thoughtful considerations for treatment are needed. (See section *Managing Thrombus*)

Multivessel Coronary Artery Disease

Multivessel coronary artery disease is present in approximately 50% of patients with STEMI and is associated with increased adjusted mortality compared to patients with disease of

the infarct artery only [78]. Multiple clinical trials have demonstrated that PCI of the non-infarct artery after primary PCI is safe and is associated with a reduced risk of MACE [79-83]. Current guidelines recommend staged PCI of a significantly stenosed non-infarct artery [1], however more recent studies have suggested that multi-vessel PCI at the time of primary PCI reduces the risk of recurrent ischemic events when compared to a staged PCI procedure [84, 85]. For this reason, individualized care is needed when determining the indications for, and timing of PCI of the non-infarct artery in STEMI. This will depend on multiple factors including clinical and hemodynamic stability, lesion complexity of both the infarct artery and the non-infarct artery, the extent of myocardium at risk, and the presence of other comorbidities [1]. The benefits of non-infarct artery PCI should not be extrapolated to patients with CS in which multi-vessel PCI at the time of primary PCI is associated with worse outcomes compared with culprit only PCI [86] and is not recommended [1]. In some situations, there may be complex disease of the non-infarct artery disease for which surgery is necessary. In such cases, unless surgery is urgently warranted, PCI and stenting of the infarct vessel should be performed with plans for elective CABG at a later date [1]. In select scenarios when urgent surgery is needed, it may be appropriate to consider promptly restoring flow in the culprit vessel without stenting, followed by urgent coronary bypass to all vessels. The functional assessment of the non-infarct artery to guide the decision for PCI is controversial with conflicting results from randomized trials [87, 88]. The Physiology-guided vs Angiography-Guided Non-culprit Lesion Complete Revascularization for Acute MI & Multivessel Disease (COMPLETE 2) trial (NCT05701358) which plans to enroll 5100 patients with AMI and multi-vessel disease will examine the role of physiology guided PCI of the non-infarct artery in acute infarction.

Consensus Tips for Special Circumstances

- **RHC should be performed in STEMI with CS during the index procedure.**
- **Microaxial flow pumps can be beneficial in patients with STEMI and CS**
- **Immediate catheterization and rescue PCI are essential when fibrinolytic therapy has failed.**
- **In stable patients, early catheterization within 24 hours of fibrinolytic therapy is indicated.**
- **Intracoronary imaging should be routinely performed in cases of stent thrombosis to investigate the mechanism.**
- **Complete revascularization of a significant non-infarct stenosis should be performed.**

ANATOMICAL SUBSETS

Coronary calcification

Moderate and severe calcification of the culprit lesion has been reported in 26% and 6% of STEMI cases respectively, when reviewed by an angiographic core laboratory [89]. The presence of moderate/severe calcification is associated with suboptimal angiographic results, higher procedural complications, and independently predicts increased risk of stent thrombosis and ischemic target lesion revascularization at 1 year [89]. A detailed OCT analysis of a cohort of patients with acute coronary syndrome found three calcified culprit plaque phenotypes: eruptive calcified nodules, superficial calcific sheet, and calcified protrusion [90]. The management of calcified culprit lesions in STEMI has not been prospectively examined, however, reports of rotational atherectomy and intracoronary lithotripsy suggest these techniques can be useful. Until further evidence is available, as in all cases of calcified lesions, the choice of plaque modification strategies should be individualized based on intracoronary imaging guidance [91].

Bifurcation Lesions

Bifurcation lesions are encountered in 10-20% of patients with STEMI, results in longer fluoroscopy times and higher contrast use, but have rates of acute procedural success similar to non-bifurcation lesions. There are no prospective studies examining the approach to patients with STEMI involving a bifurcation lesion and the management of complex bifurcations lesions in a stable patient may not be similarly applicable to a patient with STEMI where propagation of thrombus into the branch vessel can occur. An earlier retrospective study with first generation drug eluting stents showed higher rates of cardiovascular events with the use of two stents [92], however a more recent study addressing patients with anterior STEMI with bifurcation disease of the left anterior descending artery and diagonal artery using current generation drug eluting stents showed similar rates of cardiovascular events at 6 months with a single or two stent strategy [93]. While an individualized approach to management considering the TIMI flow in the side branch, size of the side branch and degree of thrombus, should be considered, a provisional (one stent) approach maybe the preferred option in most cases of bifurcation disease of the infarct artery.

Coronary Aneurysms/Ectasia

A coronary artery aneurysm is a localized vascular dilation ≥ 1.5 times the diameter of the normal adjacent reference segment. Coronary aneurysms are found in up to 5% of patients undergoing coronary angiography, usually in association with connective tissue disorders or prior Kawasaki disease [94]. Aneurysms can also result from iatrogenic wall injury after coronary intervention with stenting or brachytherapy [94]. Giant coronary artery aneurysms leading to thrombosis with STEMI can be difficult to treat due to the potential for distal embolization and the limited options for stent sizing. As such, PCI of an aneurysmal segment in the setting of

STEMI is problematic with higher incidence of no-reflow or distal embolization, and increased rates of adverse cardiovascular events and definite stent thrombosis [95]. The most important goal for managing a STEMI involving a coronary artery aneurysm is to restore flow. This will often require advanced techniques to manage thrombus such as mechanical or manual aspiration thrombectomy or the use of IV GPI (See section *Managing Thrombus*). Once flow is restored, consideration for percutaneous or surgical treatment will depend on several factors including the size of the aneurysm, side branch involvement, and the extent of residual stenosis within the lesion. In the absence of a large side branch, covered stenting and/or stent assisted coiling has been reported with good success [96, 97] . In many cases, traditional covered stents used for coronary arteries maybe sufficient, however when the aneurysm is > 5 cm, peripheral stents are needed [96]. When managing these cases, a 7 French guiding catheter can aid in the delivery of these large devices. For larger aneurysms or those with involvement of a significant side branch, urgent surgery (in the case of ongoing symptoms and slow flow) or delayed surgery should be considered.

Consensus Key Points for Managing Anatomical Subsets

- **When necessary, plaque modification for calcified lesions can be used to facilitate stent delivery and expansion after restoration of blood flow.**
- **A provisional (one stent) strategy in bifurcation lesions is preferred.**
- **The focus of management of a coronary artery aneurysm is to restore flow.**

NON-ATHEROSCLEROTIC CAUSES OF STEMI

Epicardial Vasospasm

Abnormal vasoconstriction often precipitated by a pharmacologic substance (e.g. cocaine/triptans) and/or emotional stress can lead to transient occlusion of one or more epicardial

coronary arteries with accompanying ST elevation. Epicardial coronary artery vasospasm is an uncommon but important, non-atherosclerotic mechanism of acute STEMI [98]. The threshold for suspecting coronary artery vasospasm should be especially high in younger patients (<50 years of age) who do not have cardiac risk factors, when diffuse disease is seen on coronary angiography. Irrespective of the suspicion for spasm, unless hemodynamically contraindicated, intra-coronary nitroglycerin is useful in patients with an acute STEMI at the time of coronary angiography. This important measure can help identify and treat patients with coronary artery spasm while also facilitating the accurate assessment of vessel diameter for those patients undergoing PCI due to atherosclerotic plaque. Patients with myocardial infarction without obstructive coronary artery disease (MINOCA) and suspected epicardial coronary artery spasm may be referred for a comprehensive evaluation for coronary microvascular dysfunction and coronary spasm assessment with intracoronary acetylcholine provocation in the elective setting. Patients with coronary vasospasm are best managed with vasodilators such as nitrates or calcium channel blockers.

Spontaneous Coronary Artery Dissection

SCAD with intimal disruption and intramural hematoma is an increasingly recognized non-atherosclerotic mechanism of STEMI leading to acute coronary occlusion and is often a cause of myocardial infarction in young females. Recognition of SCAD features on coronary angiography is critical as the management is different than the management of STEMI due to atherosclerosis. One should suspect SCAD when managing STEMI in a young woman, a patient with concurrent systemic arteriopathies, and those with few or no conventional cardiovascular risk factors with angiographic characteristics suggestive of SCAD [99]. Angiographic SCAD

652 classification includes the following: Type I: Multiple radiolucent lumens or arterial wall
653 contrast staining; Type II: , Diffuse stenosis that can be varying severity and length; Type III:
654 Focal or tubular stenosis, that mimics atherosclerosis [100]. While intracoronary imaging may
655 confirm the diagnosis of SCAD, vessel instrumentation has risks in patients with SCAD where
656 inadvertent wiring of the false lumen can cause complications. Additionally, the use of OCT can
657 propagate the hematoma due to the need for contrast injections [101]. For this reason,
658 intracoronary imaging should be reserved for those patients in whom the diagnosis is uncertain,
659 if imaging will impact management. PCI for the treatment of SCAD is associated with an
660 increased risk of complications including further extension of the dissection due with wire
661 manipulation, or propagation of the hematoma [102]. Therefore, conservative therapy is often
662 indicated especially in stable patients, or in those with a limited territory of myocardium at risk
663 due to distal disease. PCI (or coronary bypass surgery) may be necessary in the setting of an
664 acute total occlusion of a vessel with a large area of myocardium at risk and refractory symptoms
665 or hemodynamic instability. If PCI is indicated, the goal of PCI should be to restore vessel
666 patency with adequate TIMI flow. Various techniques have been described [103], (the use of
667 compliant balloons; the use of cutting balloons; stenting proximal and distal to the dissection
668 followed by stenting of the dissected segment; or the use of a long stent with 5 mm margins
669 proximal and distal to the dissected segment) but there is limited data to support a single
670 technique and care should be individualized. When PCI is used in STEMI, contemporary data
671 suggests reasonable success with rates exceeding 90% [104].
672
673 **Coronary Embolism**
674

Coronary embolism as a cause of infarction occurs in about 3% of cases of STEMI [105]. It should be suspected when there is angiographic presence of thrombus in the absence of features suggesting a plaque rupture (such as luminal stenosis > 50%, plaque ulceration, plaque irregularity, and/or dissection), or when there is thrombosis in the setting of otherwise angiographically normal coronary arteries [105, 106]. Corroborative evidence that would support a diagnosis of embolism includes the involvement of more than one coronary artery, concomitant systemic embolization, and demonstration of a potential source (e.g., intra-cardiac tumor, prosthetic valves, infective endocarditis, atrial fibrillation, hypercoagulable state, presence of intra-cardiac communication) [107]. A scoring system has been proposed and maybe useful in ambiguous cases, although it has not been externally validated [107]. The management of STEMI in the setting of coronary embolism depends on the size of the embolism, the flow in the involved vessel and the amount of myocardium at risk. A distal coronary embolism or embolism of a small branch vessels may be conservatively managed with IV antithrombotic therapies. In these cases, the focus of management should be on identifying and treating the underlying source of thrombus to prevent future events. On the other hand, a large thrombus burden in a proximal vessel will often require PCI. When PCI is indicated, wire manipulation alone may open coronary occlusions and the resultant improvement in coronary flow will allow for intrinsic fibrinolysis. For larger thrombus burden in a more proximal vessel, clot extraction devices should be employed (See section *Managing Thrombus*). Stent placement is usually not indicated unless intracoronary imaging suggests underlying plaque rupture.

Myocardial Infarction without Obstructive Coronary Artery Disease

Approximately 5% of patients presenting with acute MI do not have obstructive CAD on angiography defined as <50% stenosis of all coronary arteries [108] and are diagnosed with MINOCA. MINOCA is more commonly seen with NSTE-ACS, with ~20% of patients with MINOCA presenting with STEMI. It can be caused by atherothrombotic plaque disruption (rupture, erosion) or non-atherothrombotic conditions such as SCAD, vasospasm, coronary microvascular dysfunction, or coronary embolism. Mortality and re-infarction rates have been reported at 2.6% and 3.9% respectively [109]. While pooled data suggests better outcome with MINOCA than AMI due to obstructive CAD [109], in one report, the adjusted long-term mortality was higher in MINOCA [110]. In STEMI cases and suspected MINOCA, evaluation including serial cardiac enzymes (to confirm myocardial injury), and multimodality imaging with IVUS or OCT followed by cardiac MRI can be helpful to determine the etiology and help excluded MINOCA mimics such as myocarditis, Takotsubo cardiomyopathy or non-ischemic causes of myocardial injury [111]. When possible, cardiac MRI should be performed early after the infarct to improve the diagnostic yield [112]. After the initial evaluation, if the etiologic cause of MINOCA remains unclear then evaluation for coronary microvascular dysfunction including provocative spasm testing, is strongly encouraged.

Consensus Key Points for Managing Non-atherosclerotic Causes of STEMI

- **In the absence of contraindications IC NTG should be administered during the diagnostic angiogram to help diagnose cases of epicardial spasm.**
- **In patients with SCAD and a patent infarct artery with TIMI III flow, conservative management is advised.**
- **Thrombectomy may be used in patients with coronary embolism**
- **When MINOCA is suspected additional investigations such as left ventriculogram, intracoronary imaging, cardiac MRI and/or coronary microvascular dysfunction testing, may be necessary to identify the etiology and exclude MINOCA mimics.**

QUALITY OF CARE AND OUTCOMES

Hospital or health system-level STEMI committees should ideally track each STEMI to review activation times, times to treatment, and outcomes with the intent of identifying areas for improvement, crafting focused interventions, measuring the results of said interventions and considering different strategies until the goal is obtained. Once goals are achieved, maintenance and monitoring of quality should be the objective [6]. Quality of care includes care across the entire system, encompassing prehospital, intraprocedural and postprocedural management. Measures that should be tracked include: 1) Door to ECG acquisition; 2) Door-in- door-out for transferred patients; 3) First door-to-device times for transferred patients; 4) First medical contact to device times for EMS presenting patients; 5) Door to device times for walk-ins; 6) Complaints of chest pain to device times for in-hospital STEMI cases; 7) Periprocedural complications, and 8) Mortality. Quality measures should also consider the balance of false activation rates (which can unduly burden and cost health care systems) and the outcomes for medically treated patients with STEMI. Additionally, the inclusion of every STEMI patient getting primary PCI (ie without exclusions), may be more informative about real world conditions. Individual operators and programs can use national and state registry data for feedback to improve quality as compared to geographic benchmarks.

Consensus Key Points on Quality of Care and Outcomes

- **All hospitals/health care systems should track every STEMI case to assess time to treatment, and outcomes with an aim for continued quality improvement**

FUTURE DIRECTIONS

The percutaneous techniques for managing STEMI have dramatically evolved over the years. Despite these advances, there remain unanswered questions related to the management of certain patient subsets. First, the ECG is not always accurate in identifying patients with acute coronary occlusions, particularly in the left circumflex territory. The use of artificial intelligence to create a detailed scoring system (incorporating clinical and ECG variables) to predict acute arterial occlusion can be extremely useful to improve the number of patients getting timely reperfusion in the setting of an acute occlusion. Additionally, future research is needed to better identify the optimal management of patients who present late after symptom onset, or in those with large thrombus burden or no reflow. Finally, given the inability to adequately estimate the extent of MVO after PCI, the identification of those patients at risk for MVO, and the incorporation of tools to assess microvascular function post PCI could provide important information regarding the need to employ additional therapies for treatment of this subset of patients. These predictive and assessment tools will be particularly useful if we have a more evidence-based approach to treating MVO. For this reason, phase III trials are needed to further explore the options for limiting infarct size in the at-risk population of patients with STEMI.

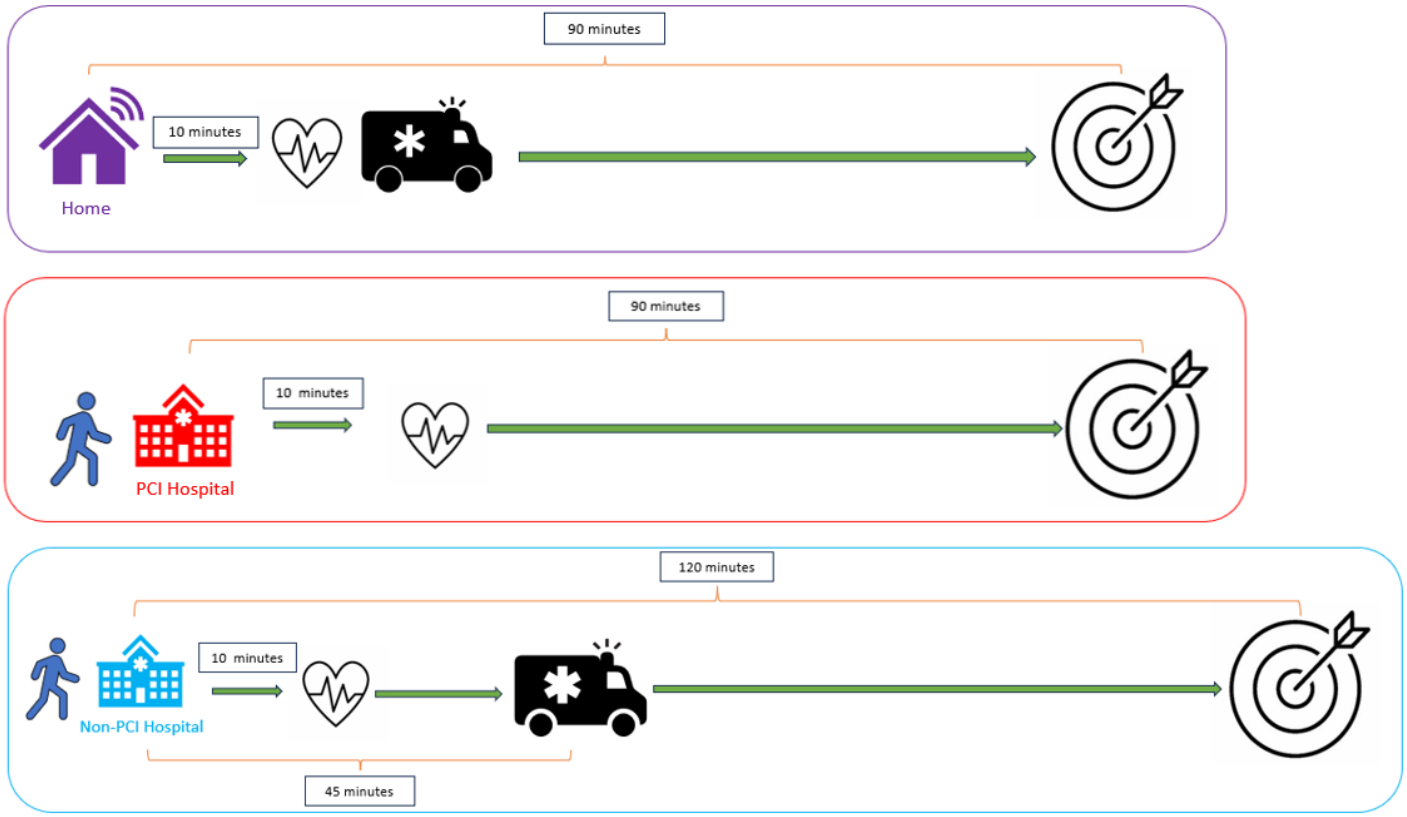
CONCLUSIONS

STEMI remains a leading cause of morbidity and mortality in the United States. Timely reperfusion with primary PCI is associated with improved outcomes. Optimal techniques for performing coronary angiography and PCI should always be employed to minimize procedural complications and optimize timely tissue level reperfusion with the goal of improving short- and long-term outcomes. While there remains variability in practice across hospitals and CCL teams,

773 standard protocols for rapid assessment, mobilization of the CCL team as well as protocols for
774 angiography and PCI can provide consistency in care.
775
776

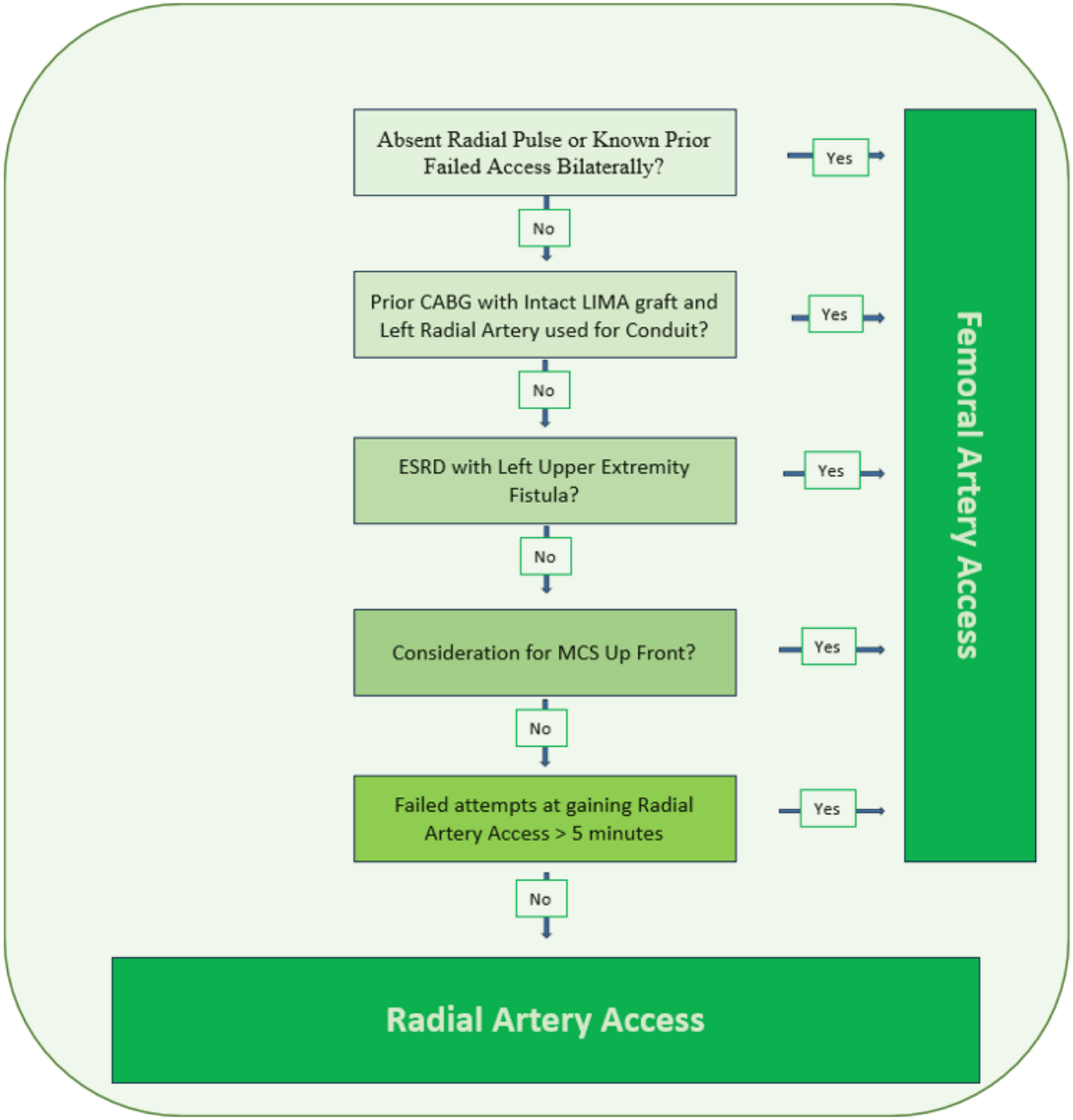
FIGURE LEGENDS

Figure 1: Timeline of objectives in primary PCI for STEMI: The figure depicts the various modes of presentation and the time to treatment goals



782 **Figure 2:** An algorithm for determining the proper arterial access in STEMI: The figure depicts the considerations to be taken when

783 determining arterial access in STEMI


Absent Radial Pulse or Known Prior Failed Access Bilaterally?
Yes
No
Prior CABG with Intact LIMA graft and Left Radial Artery used for Conduit?
Yes
No
ESRD with Left Upper Extremity Fistula?
Yes
No
Consideration for MCS Up Front?
Yes
No
Failed attempts at gaining Radial Artery Access > 5 minutes
Yes
No
Femoral Artery Access
Radial Artery Access

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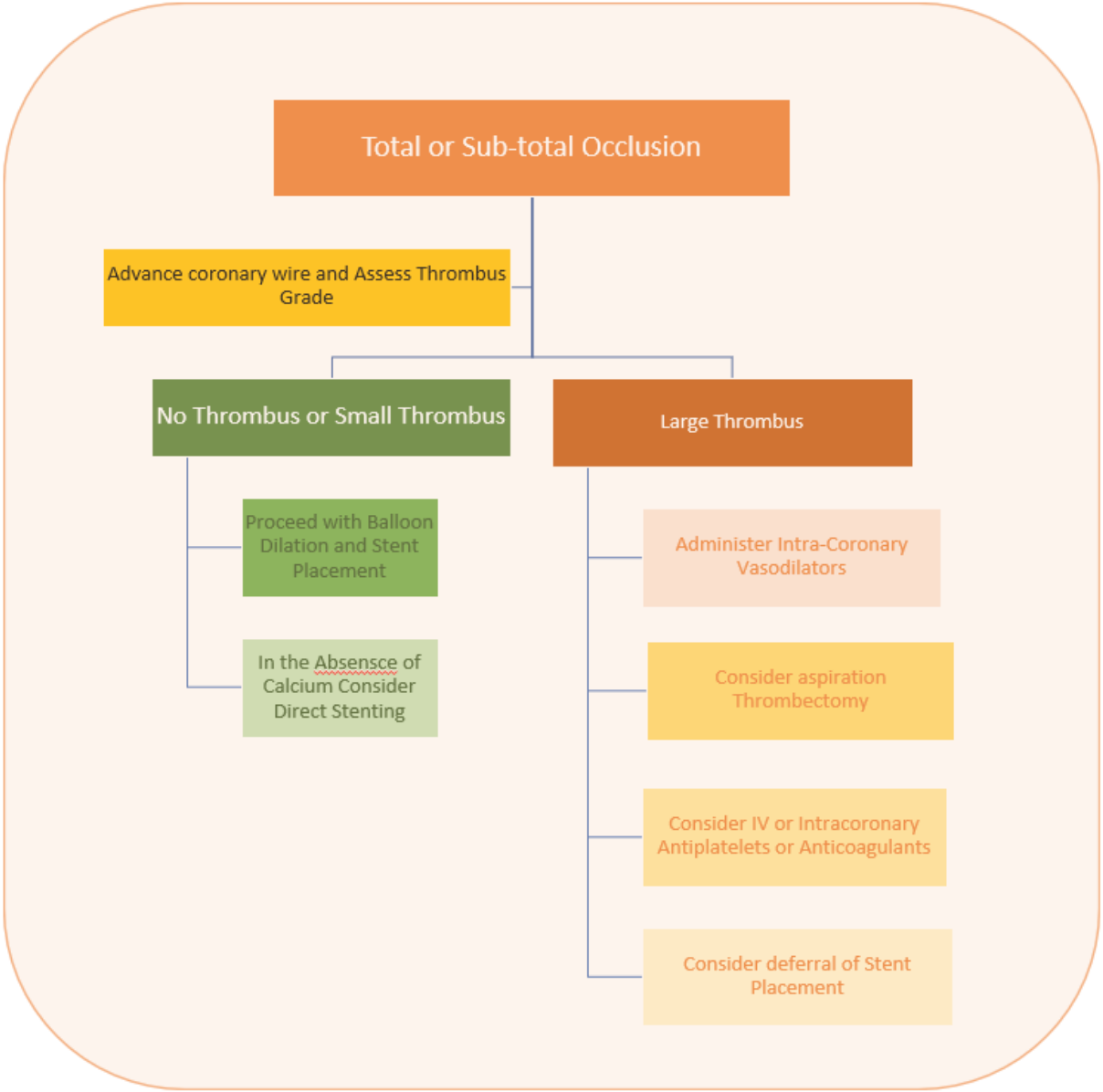
785

786 **Figure 3:** Managing intra-coronary thrombus: The figure depicts the step-by-step approach to managing intra-coronary thrombus

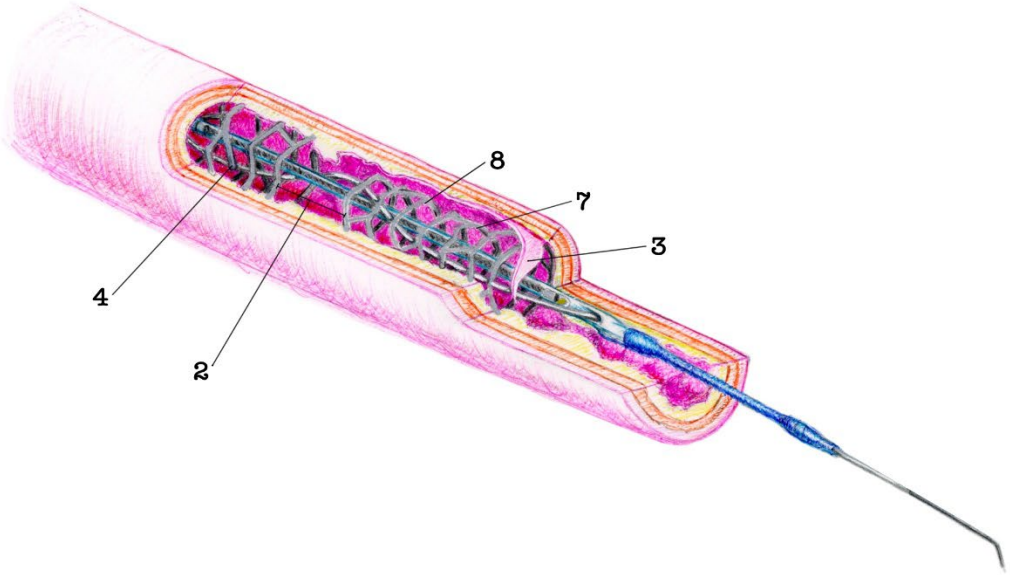

Total or Sub-total Occlusion
Advance coronary wire and Assess Thrombus Grade
No Thrombus or Small Thrombus
Large Thrombus
Proceed with Balloon Dilation and Stent Placement
In the Absensce of Calcium Consider Direct Stenting
Administer Intra-Coronary Vasodilators
Consider aspiration Thrombectomy
Consider IV or Intracoronary Antiplatelets or Anticoagulants
Consider deferral of Stent Placement

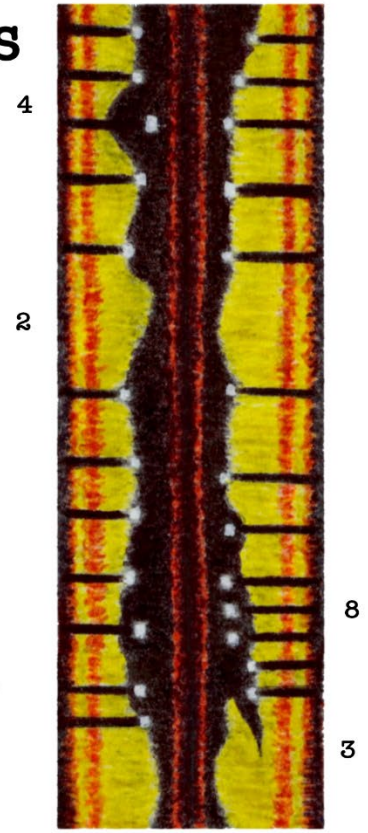
787

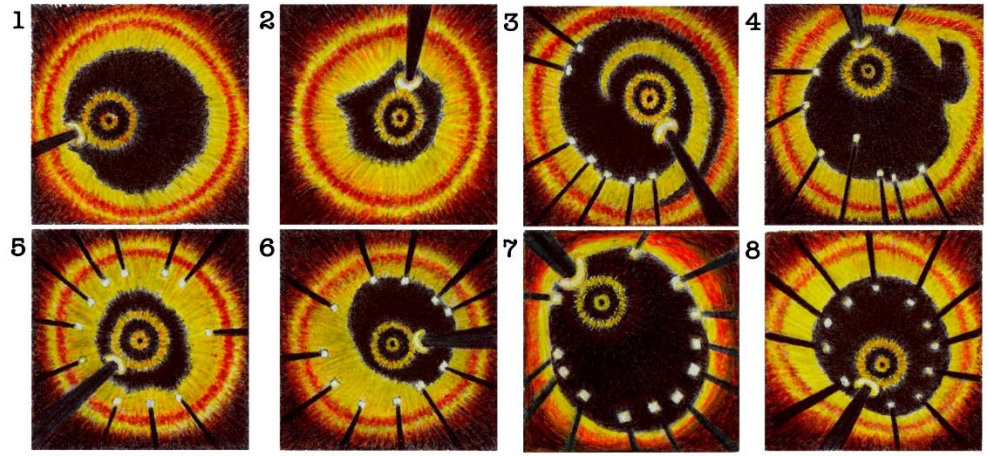
788 **Figure 4:** Mechanisms of Stent Thrombosis: The figure depicts the potential mechanisms of stent thrombosis. These are often

789 assessed using intracoronary imaging .

Mechanisms of Stent Thrombosis







1. Normal Artery
2. Residual Uncovered Plaque
3. Edge Dissection
4. Stent Deformity
5. Neointimal Hyperplasia
6. Neoatherosclerosis
7. Malapposition
8. Underexpansion

790

Figure 5: Important parameters assessed by intracoronary 40maging The figure depicts the important variables that should be determined with intracoronary imaging including proximal and distal vessel diameter, plaque morphology, lesion length, stent expansion, stent dissection and stent apposition.

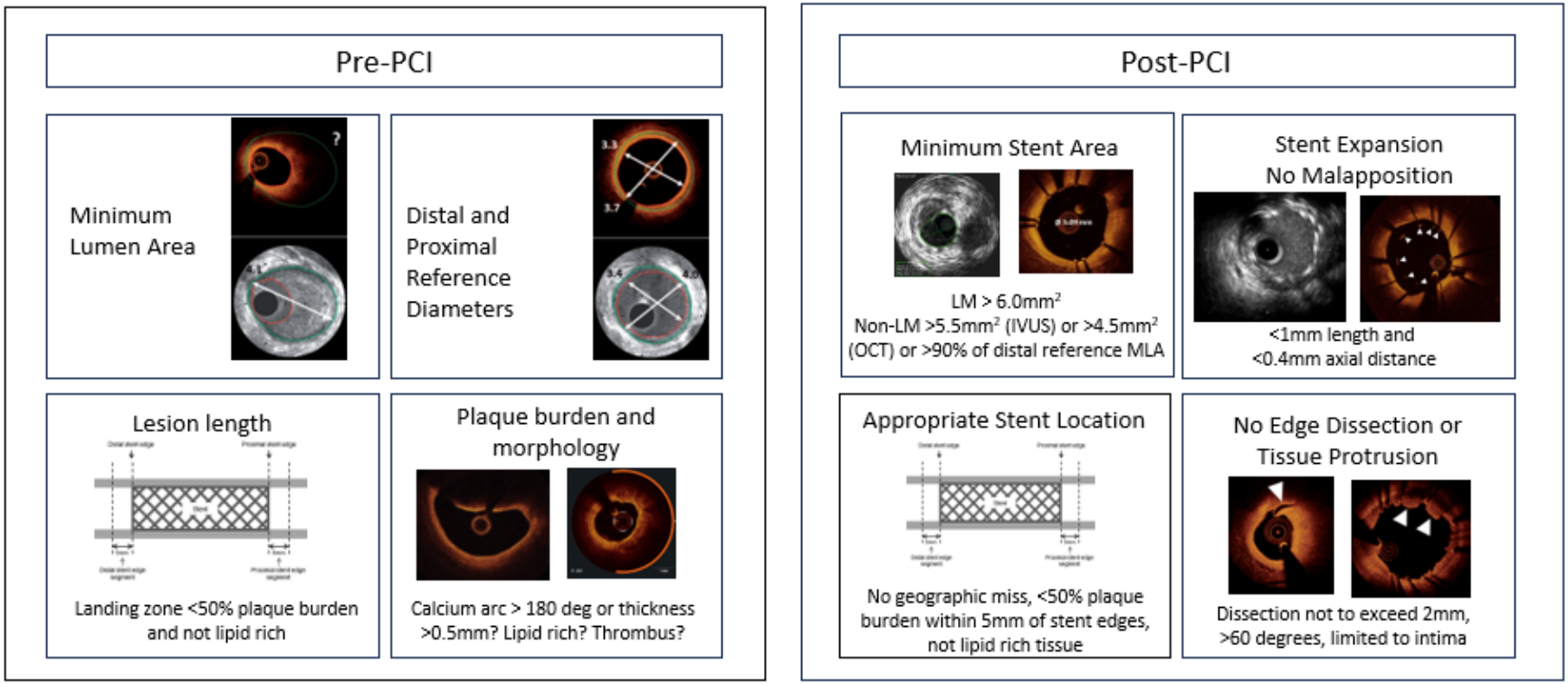


Table 1: Key Components to Successful Catheterization Laboratory Team Readiness

Pre-hospital activation of CCL for patients presenting via EMS
Single activation of all CCL team members
ECG transmission to the CCL team
ED bypass for stable patients presenting via EMS directly to CCL team
Expectations for CCL team members arrival 20-30 minutes from time of page

CCL=cardiac catheterization laboratory; ECG=electrocardiogram; ED=emergency department; EMS=emergency medical servies

Table 2: Essential Equipment for CCL to Perform Primary PCI

Standard Equipment	Comments
Plaque modification tools: At least one of the Following: • Cutting or scoring balloon • Intracoronary lithotripsy • Rotational Atherectomy • Orbital atherectomy	To facilitate stent delivery and expansion in severely calcified lesions
Microcatheters	For delivery of distal meds and exchange of wires in tortuous arteries
Guide extension device	For delivery of balloons and stents in tortuous arteries
Aspiration catheters: At least one of the following: • Manual aspiration catheter • Mechanical aspiration catheter	For cases of large thrombus burden or emboli
Intracoronary Imaging: At least one of the following • IVUS • OCT	To assess lesion morphology and guide PCI
MCS: At least one of the following: Intra-aortic balloon pump Impella CP Extracorporeal membrane oxygenation	For cases of refractory shock
Transvenous pacer	For unstable patients with complete heart block

CCL=cardiac catheterization laboratory; IVUS=intravascular ultrasound; MCS=mechanical circulatory support; OCT=optical coherences tomography; PCI=percutaneous coronary intervention

Table 3: Best Practices for Arterial Access in STEMI

Radial Artery Access	Femoral Artery Access
Pre-procedure assessment to consider right vs left radial access	Fluoroscopic guidance to identify lower half of femoral head
Ultrasound Guidance	Ultrasound Guidance
Radial artery puncture 1-2 cm proximal to styloid process	Micro-puncture needle
Use of hydrophilic sheath	Fluoroscopic guidance when advancing wire
Limited angiography of artery when there is resistance with wire advancement after sheath placement	Femoral Angiography to confirm proper location
Patent hemostasis for sheath removal	Vascular Closure devices (Especially with large bore access)

cm=centimeter; STEMI=ST elevation myocardial infarction

Table 4: Suggested Dosing for Intra-coronary Administration of Drugs used for No Reflow

Agent	Dose	Notable Mentions
Adenosine	50 µgm to 200 µgm	Avoid in heart block
Nitroprusside	50 µgm-200 µgm	Avoid in Severe AS or HCM
Diltiazem	400 µgm	Avoid in CS or heart block
Verapamil	100 µgm -250 µgm	Avoid in CS or heart block
Nicardipine	50 µgm-200 µgm	Avoid in severe AS
Epinephrine	50 µgm-200 µgm	Avoid in ventricular arrhythmias

AS=aortic stenosis; CS=cardiogenic shock; HCM=hypertrophic cardiomyopathy; µgm=microgram

Supplemental Table: Definitions of TIMI Thrombus Grade

Grade	Definition
Grade 0	No thrombus
Grade 1	Possible thrombus
Grade 2	Small thrombus (Greatest dimension ≤ ½ of the vessel diameter)
Grade 3	Moderate thrombus (Greatest dimension > ½ but ≤ 2 times the vessel diameter)
Grade 4	Large thrombus- (Greatest dimension > 2 times the vessel diameter)
Grade 5	Total occlusion

TIMI=thrombolysis in myocardial infarction

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