During the last few decades, acute ST-elevation on an electrocardiogram (ECG) in the proper clinical context has been a reliable surrogate marker of acute coronary occlusion requiring primary percutaneous coronary intervention (PPCI). In 2004, the American College of Cardiology/American Heart Association ST-elevation myocardial infarction (STEMI) guidelines specified ECG criteria that warrant immediate angiography in patients who are candidates for primary PPCI, but new findings have emerged that suggest a reappraisal is warranted. Furthermore, as part of integrated and efficient STEMI systems, emergency department and emergency medical services providers are now encouraged to routinely make the time-sensitive diagnosis of STEMI and promptly activate the cardiac catheterization laboratory (Cath Lab) team. Our primary objective is to provide a practical summary of updated ECG criteria for emergency coronary angiography with planned PPCI, thus allowing clinicians to maximize the rate of appropriate Cath Lab activation and minimize the rate of inappropriate Cath Lab activation. We review the evidence for ECG interpretation strategies that either increase diagnostic specificity for "classic" STEMI and left bundle-branch block or improve diagnostic sensitivity in identifying 4 STEMI-equivalents: posterior MI, acute left main occlusion, de Winter ST/T-wave complex, and certain scenarios of resuscitated cardiac arrest. (Am Heart J 2010;160:995-1003.e8.)

The key trigger point for emergency cardiac catheterization laboratory (Cath Lab) activation is usually a single electrocardiogram (ECG) diagnostic of an acute ST-elevation myocardial infarction (STEMI), which instantly reclassifies a patient with chest pain or other acute cardiac symptoms from “routine evaluation” status to “high-priority” STEMI procedure (Figure 1). The 2004 American College of Cardiology/American Heart Association (ACC/AHA) guidelines1 specify ECG criteria that warrant immediate angiography in patients who are candidates for primary percutaneous coronary intervention (PPCI). Although new findings related to the ECG diagnosis of STEMI have emerged since 2004, they have not been reviewed by any of the more recent guidelines.2-6 Hence, our primary objective is to provide a practical summary of updated criteria for emergency coronary angiography with planned PPCI, thus allowing clinicians to maximize the rate of appropriate Cath Lab activation and minimize the rate of inappropriate Cath Lab activation.

Three background concepts are important. First, ECG analysis is a fundamental clinical skill that is used routinely by a broad range of clinicians, but high-risk ECG findings consistent with acute ischemia continue to be overlooked.7 Second, a common “efficiency challenge” involves the need to quickly differentiate and treat the small cohort of acute STEMI patients from the much larger group of undifferentiated “chest pain” patients (Figure 1). This must be balanced against the potential for poor resource utilization, especially with the current emphasis on early Cath Lab activation by either emergency department (ED) or emergency medical services providers.8,9 Third, a coordinated systems-based approach is currently emphasized by the ACC/AHA STEMI guidelines,2-3 the ACC Door-2-Balloon (D2B) Alliance,10 and the AHA Mission: Lifeline initiative.11 Proposed efforts within Mission:Lifeline12 to comprehensively track all Cath Lab activations and improve overall STEMI system efficiency depend on the existence of clearly

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defined criteria that distinguish appropriate versus inappropriate Cath Lab activation.

To maximize appropriate Cath Lab activations, we critically review and summarize (Tables I and II) real-time ECG interpretation strategies that accurately identify both STEMI and STEMI-equivalents. Criteria defining the term STEMI-equivalent have been gradually standardized by both the National Cardiovascular Data Registry (NCDR) data dictionaries and international consensus, and broadly include any ECG pattern potentially associated with an acute coronary occlusion but lacking “classic” ST-elevation. Efficient STEMI systems of care need to maximize diagnostic specificity for both classic STEMI ECG patterns and new (or presumed new) left bundle-branch block (ie, a STEMI-equivalent), whereas diagnostic sensitivity should be improved in identifying 4 additional STEMI-equivalents: posterior MI, acute left main occlusion, de Winter ST/T-wave complex, and certain scenarios of resuscitated cardiac arrest. Moreover, we propose that clinicians conceptually prioritize 3 questions when analyzing any ECG: (1) Are the ST-segments consistent with acute STEMI or STEMI-equivalent? (2) Has chronic ST-segment elevation that does not represent acute myocardial infarction been excluded? (3) Is the patient a reasonable candidate for reperfusion therapy (PPCI or fibrinolytics)?

Definitions and background concepts

Acute myocardial infarction can be defined from various clinical perspectives: electrocardiography, biomarkers, angiography, imaging, and pathology. However, the 2004 guidelines emphasize the central role of the ECG in decision-making during the acute phase and define “classic STEMI” as ≥1 mm ST-elevation in 2 adjacent leads (class I-A recommendation). Acute and persistent ST-elevation, meeting these criteria in either leads V1 through V4 or leads II/III/aVF, represents the most common (≈80%) ECG finding that is customarily reported, respectively, as an infarction of the anterior or inferior region of the heart in registries, clinical trials, and the International Classification of Diseases, Ninth Revision, billing codes.

As specified in a recent expert consensus document, actual ECG leads should not be simply referred to as “anterior” or “inferior” because this de-emphasizes the important bioelectric principle that all ECG leads are bipolar. Thus, an acute coronary occlusion can manifest on any of the 12 individual leads of a standard ECG with either ST-elevation at the positive pole or ST-depression at the negative pole. For example, leads V1 through V4 might be designated most accurately as “anteroseptal-posterolateral” bipolar leads. This provides the rationale for designating certain types of ST-depression ECG patterns as true STEMI-equivalents (eg, posterior MI, acute left main occlusion, de Winter ST/T-wave complex).

Because current guidelines state that timely PPCI is the preferred reperfusion modality, our review has principally focused upon appropriate Cath Lab activation criteria. However, in many STEMI-care settings, prompt administration of fibrinolytics represents the only realistic reperfusion option for an acute coronary occlusion per the guidelines. Fortunately, initial ECG interpretation strategies for detecting acute STEMI or STEMI-equivalent are similar for treatment pathways involving either PPCI or fibrinolysis, but safe fibrinolytic administration also requires review of a standardized Contraindications Checklist.

Maximizing diagnostic specificity for acute coronary occlusion

Classic STEMI

In the proper clinical context, acute ST-elevation ≥1 mm in 2 contiguous ECG leads represents a reliable surrogate marker for an acute coronary occlusion.
### Table I. Comparison of 2004 ACC/AHA guidelines and authors' proposed update for ECG criteria that enhance the rate of appropriate Cath Lab activation for acute MI

<table>
<thead>
<tr>
<th>Indications for appropriate Cath Lab activation</th>
<th>Diagnostic criteria for patients with symptoms &lt;12 h</th>
<th>2004 ACC/AHA guideline recommendation</th>
<th>Proposed update vs. ACC/AHA guidelines</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classic STEMI Anterior</td>
<td>ST-elevation ≥1 mm in 2 contiguous leads V1-V4</td>
<td>Class I-A</td>
<td>Agree</td>
<td>ST-elevation ≥2 mm (men) and ≥1.5 mm (women) improves diagnostic specificity. Presence of reciprocal changes (ST-depression in opposite leads) improves diagnostic specificity.</td>
</tr>
<tr>
<td>Inferior</td>
<td>ST-elevation ≥1 mm in 2 contiguous leads (II, III, or AVF)</td>
<td>Class I-A</td>
<td>Agree</td>
<td>Presence of reciprocal changes improves diagnostic specificity.</td>
</tr>
<tr>
<td>Lateral</td>
<td>ST-elevation ≥1 mm in 2 contiguous leads (I, AVL, V5, or V6)</td>
<td>Class I-A</td>
<td>Agree</td>
<td>As above.</td>
</tr>
</tbody>
</table>

#### STEMI-equivalents

| New or presumed new-onset LBBB                  | Class I-A | Proposed demotion in future ACC/AHA guidelines | Unless clinically unstable, most LBBB should be evaluated with biomarkers and non-emergent angiography if indicated. An “old” ECG without LBBB does not necessarily confirm that the “new LBBB” is acute. Use of these decision criteria provides >95% specificity and avoids the need to find a prior ECG for comparison. Discordant ST-elevation ≥ 5 mm is also a Sgarbossa criteria, but some studies found it a weak predictor. Recent data demonstrated that most posterior MIs are currently evaluated with urgent (rather than emergent) angiography, but this delay is associated with worse clinical outcomes. Most relevant in any ECG with diffuse ST-depression ≥ 1 mm that does not meet classic STEMI criteria, thus providing a subtle clue that emergency angiography may be warranted. Tall T waves and up-sloping ST depression are persistent, not transient. Associated with proximal LAD occlusion. Generally prudent to perform serial ECGs, because true HATW generally morph quickly into a classic STEMI pattern. Hyperkalemia is another common cause of tall T waves.
| Preexisting LBBB with Sgarbossa concordance     | None      | Proposed addition to future ACC/AHA guidelines | Normal QRS duration |
| Posterior MI (isolated)                         | ST-depression ≥0.5 mm in leads V1-V3 | Fibrinolytics: class IIa-C | Proposed clarification in future ACC/AHA guidelines |
| Left Main coronary occlusion                    | ST-depression ≥ 1 mm in 6 or more leads | Primary PCI: class I-A implied | Proposed addition to future ACC/AHA guidelines |
| de Winter ST/T-wave complex                     | ST depression ≥1 mm up-sloping at the J-point in leads V1-V6 | Proposed addition to future ACC/AHA guidelines |
| Hyper-acute T-waves                             | Tall peaked T waves immediately following symptom onset may represent acute ischemia, but clinical studies are lacking. | Potential addition to future ACC/AHA guidelines | Generally prudent to perform serial ECGs, because true HATW generally morph quickly into a classic STEMI pattern. Hyperkalemia is another common cause of tall T waves. |
Inappropriate Cath Lab activations have a number of negative implications, including poor resource utilization, unnecessary costs, patient safety risk, increased patient anxiety, work-life-sleep issues that may adversely affect career longevity of Cath Lab staff (ie, “burn-out”), and potential community risk from entire Cath Lab teams rushing (ie, driving fast) back to the hospital to treat an after-hours STEMI. Initial reports of ED physician performance in diagnosing acute STEMI identified a 9% rate of false-positive Cath Lab activation (no culprit artery and negative biomarkers). More recent surveillance of “unnecessary” Cath Lab activations (ie, cardiologist did not perform emergency coronary angiography) by ED physicians demonstrated a 5% rate from a single-center experience (n = 249 activations) in Virginia and a 6% rate across 14 hospitals (n = 2,213 activations) in a North Carolina STEMI system. In contrast, evaluation of prehospital-ECG interpretation by paramedics (n = 646) from Los Angeles County, California, found a 20% rate of inappropriate Cath Lab activation (ie, cardiologist did not perform emergency coronary angiography), with an additional 5% of suspected STEMI on PH-ECG that never resulted in Cath Lab activation (although PH-ECG transmission was rarely used). Similar analysis (n = 529) from Orange County, California, found a 23% rate of inappropriate Cath Lab activation.

As proposed previously, we believe a <5% rate of inappropriate Cath Lab activation represents a reasonable STEMI systems goal. Table III provides our conceptual framework summarizing appropriate versus inappropriate Cath Lab activation scenarios, recognizing that not all Cath Lab activations classified as appropriate actually result in PPCI. To help clinicians optimize resource utilization in STEMI systems, 6 ECG-interpretation strategies are reviewed below.

First, the degree of ST-elevation is a continuous variable that has been stratified (<1 or ≥1 mm) in the guidelines to yield a convenient binary diagnosis (STEMI absent vs. present, respectively). However, evidence exists for increased specificity when diagnostic criteria involve ≥2 mm ST-elevation in certain precordial leads. In recognition of benign ST-elevation that is frequently observed in healthy adults, the NCDR data dictionaries (Cath-PCI v4.3 and ACTION-GWTG v2.1) currently set the STEMI threshold in leads V2-V3 at 2 mm for men and 1.5 mm for women. Similarly, a recent AHA/ACC statement in conjunction with the Heart Rhythm Society listed the same criteria (except for men age <40, the threshold is >2.5 mm ST-elevation in leads V2-V3).
The standard 12-lead ECG. By definition, reciprocal magnitude or undetectable due to technical limitations of ples, but this reciprocal ST-depression might be of lesser ECGs with acute STEMI based upon bioelectric princi-

those meeting ST-elevation criteria) should exist for all reperfusion, the cohort with Q waves at baseline had the baseline ECG. Even with contemporary PPCI-based

[1 mm] duration, excluding lead III) already present on

an AHA scientific statement recommended that com-

and positive predictive value for acute MI. More recently,

≥ (ie, ST-depression never occur in ECGs considered normal or those with narrow-complex STE-mimics (except lead aVR with pericarditis). In a fibrinolytic era study, the presence of any reciprocal ST-depression in ≥2 leads was associated with >95% diagnostic specificity and positive predictive value for acute MI. More recently, an AHA scientific statement recommended that computerized interpretation algorithms incorporate the location of ST-depression to predict the location of a coronary occlusion. Collectively, these data suggest that clinicians should evaluate every ECG with suspected STEMI for reciprocal changes: if present, then appropriate Cath Lab activation is almost certain; if absent, the presence of a STE-mimic versus a true STEMI without reciprocal changes should be carefully but expeditiously considered. Observation of reciprocal changes may be also be helpful in resolving challenging “semi-STEMI” cases near the 1-mm threshold, where one clinician may see 1.1 mm of ST-elevation but another sees only 0.9 mm.

Fourth, pathologic Q-waves may develop in leads initially demonstrating ST-elevation, and hence their presence supports the diagnosis of true STEMI (but their absence does not exclude acute STEMI). Furthermore, a recent APEX-AMI substudy analysis of 4,530 patients with ≥6 hours of symptoms identified 2,514 (56%) with a Q wave (defined as ≥40 milliseconds [1 mm] duration, excluding lead III) already present on the baseline ECG. Even with contemporary PPCI-based reperfusion, the cohort with Q waves at baseline had worse outcomes (composite of death, heart failure, or shock at 90 days) after multivariate adjustment.

Fifth, a commonly used ECG computer algorithm demonstrated 90% specificity for identifying acute STEMI when evaluated in a clinical trial, thus providing a reasonable real-time diagnostic aide for clinicians involved in time-pressured ECG interpretation. However, experience with the computer algorithm for prehospital ECGs as part of regional STEMI networks has demonstrated lower specificity and increased rates of inappropriate Cath Lab activation.

Lastly, immediate Wireless transmission and Physician Interpretation (WiPI) of all prehospital-ECGs with suspected STEMI improves the rate of appropriate Cath Lab activation. By increasing the number of “critical eyes” that review an ECG in real-time (simultaneously with patient transport), WiPI can provide an important “appropriateness filter” between a prehospital STEMI Alert and an in-hospital Code STEMI.

### Left bundle-branch block

Left bundle-branch block (LBBB) represents a unique challenge in the acute setting, because it can be categorized as either a STE-mimic or a STEMI-equivalent. As a STE-mimic, chronic LBBB patterns in asymptomatic patients almost always have persistent ST-elevation ≥1 mm in the anteroseptal and/or inferior leads. However, for decades, new (or presumed new) LBBB has also been considered a STEMI-equivalent that requires prompt therapy, with inclusion in guidelines, clinical trials, NCDR registries, and Joint Commission acute MI core measures. Furthermore, both the ACC/AHA and European guidelines strongly recommend (class I-A) immediate reperfusion, based predominately on old meta-analysis data showing reduced mortality in fibrinolytic-treated LBBB patients.

Data have gradually accumulated suggesting that traditional criteria using new (or presumed new) LBBB as a STEMI-equivalent results in low diagnostic specificity and high rates of inappropriate Cath Lab activation. For example, a fibrinolytic era study of 7,725 consecutive patients with suspected cardiac ischemia presenting to a single ED found only 182 (2.4%) patients with LBBB on ECG, and only 24 (13%) of the 182 with LBBB had an acute myocardial infarction using positive biomarkers as the gold standard.

In the modern era, angiographic assessment of “presumed new LBBB” also raised concerns about treating these patients emergently as a true STEMI-equivalent. A recent single-center PPCI-focused study of 7,937 “chest pain” patients in the ED identified 55 (0.7%) presumed new LBBB, 136 (1.7%) confirmed old LBBB, and 7,746 (97.6%) without LBBB. In the primary analysis comparing the “new vs old vs no” LBBB cohorts, the rate of biomarker-confirmed acute MI was similarly low (7.3%,
5.2%, 6.1%, respectively, \( P = .75 \)). Thus, \( >90\% \) of LBBB patients evaluated in the ED do not have an acute coronary occlusion nor require PPCI. Similarly, a recent analysis from the PPCI-focused Minneapolis interhospital transfer network\(^{17}\) identified only 36 (2.6%) of 1335 patients with “presumed new LBBB.” Moreover, 16 (44%) of 36 from this cohort did not have a culprit artery visualized during emergency coronary angiography, and 13 (36%) of 36 had no elevation of serial biomarkers.

The latest ESC guidelines\(^{1}\) acknowledge these more recent findings by stating that biomarker results “may sometimes be helpful” in evaluating LBBB patients, thus implying treatment of stable patients with an urgent rather than emergent PCI strategy. Importantly, clinically unstable LBBB patients or those with acutely abnormal anterior wall motion by echocardiography should still receive emergency angiography.

Published ECG criteria (Table I and online Appendix item II.e) exist that maximize diagnostic specificity for detecting a true STEMI-equivalent in the setting of chronic LBBB. A chronic LBBB normally has discordance (the direction of the major component of the QRS complex is opposite that of ST segment/T wave in any lead), whereas QRS/ST concordance (with ST elevation \( \geq 1 \) mm in \( \geq 1 \) lead) appears to be the most robust predictor of acute ischemia.\(^{6,15,31}\) A recent meta-analysis\(^{32}\) found only 20% sensitivity but 98% specificity for the “concordance criteria,” but much of the data is from the fibrinolytic era when cardiac markers instead of angiography were the gold standard.

Maximizing diagnostic sensitivity for acute coronary occlusion

Posterior myocardial infarction

True posterior MI should be treated as a STEMI-equivalent, in which case isolated ST-depression \( \geq 0.5 \) mm in leads V\(_1\) through V\(_3\) represents the dominant finding (Table I and online Appendix item II.a) on a standard 12-lead ECG.\(^{6,15}\) The use of additional posterior chest wall leads (V\(_7\)–V\(_9\) \( \geq 0.5 \) mm) to detect ST elevation consistent with posterior MI is often recommended,\(^{6,15}\) but their use remains uncommon in daily practice. Clinical trials commonly exclude posterior MI and the guidelines\(^1\) reflect this paucity of evidence with a class IIa-C recommendation for administering fibrinolytics in this subset of patients. Paradoxically, the guidelines\(^1\) indirectly imply a class I-A recommendation for PPCI of posterior MI.

A recent AHA scientific statement\(^{15}\) critiqued the term posterior MI (used in the 2004 ACC/AHA guidelines, clinical trials, NCDR registries, and International Classification of Diseases, Ninth Revision, codes) because the actual infarct occurs in the lateral wall of the left ventricle when assessed by advanced imaging techniques. Although replacement terms like posterolateral MI were considered, the expert statement ultimately recommended retaining the term posterior MI for ST-depression \( \geq 0.5 \) mm in leads V\(_2\) through V\(_3\) (or ST-elevation \( \geq 0.5 \) mm in leads V\(_1\)–V\(_3\)) until additional data become available.

The TRITON-TIMI-38 study\(^{33}\) enrolled patients with both STEMI and non-STEMI, and thus it provided a unique opportunity to evaluate contemporary posterior MI therapy. According to a recent substudy,\(^{34}\) isolated ST-depression in leads V\(_1\) through V\(_9\) on index ECG was present in 1,198 (8.8%) of the 13,608 enrolled patients and retrospectively classified by the core lab as “posterior MI” (26.2%), non-STEMI (53.5%), and unstable angina (20.3%) based upon cardiac markers and angiography. Despite guideline recommendations,\(^1\) the vast majority (1193 [99.6%]) of study patients with isolated ST-depression in leads V\(_1\) through V\(_3\) were treated with non-emergent PCI (>6 hours after index ECG). However, the cohort of “slowly treated” posterior MIs had an acutely occluded coronary (most commonly the left circumflex) and had a significantly higher rate of 30-day death or MI as compared with those classified as non-STEMI.

Left main coronary occlusion

Acute left main coronary occlusion (LMCO) generally causes massive ischemia and is rapidly lethal, but a small proportion of patients have enough perfusion from right-sided collaterals to arrive alive at the hospital. A recent Cath-PCI Registry analysis\(^{35}\) spanning 2004 to 2008 identified 434 (0.35% of all attempted PCIs in Cath-PCI) with acute STEMI from a left main culprit that was “unprotected” (ie, no prior coronary artery bypass grafting to distal vessels). Interestingly, about one third of patients did not present in cardiogenic shock, even though angiography demonstrated that 85% of these patients had \( >90\% \) stenosis and 55% had complete LMCO. Importantly, survival to hospital discharge was 42% in this series.

STEMI-equivalent ECG criteria (Table I and online Appendix item II.b) exist for diagnosing the critical condition of acute LMCO, but these are not mentioned in any of the guidelines.\(^{1,3,5}\) In 2001, a small series reported that \( \geq 0.5 \)-mm ST-elevation in lead aVR is consistent with acute LM occlusion, especially when the degree of ST-elevation in aVR is greater than lead V\(_1\) and inferior ST-depression is present.\(^{36}\) A combined analysis of 3 studies with a total of 75 acute LM occlusions confirmed by angiography demonstrated \( >75\% \) sensitivity and specificity for these ECG findings.\(^{37}\)

A 2009 AHA scientific statement\(^{15}\) recommended “when the resting ECG reveals ST-depression \( >1 \) mm in 8 or more surface leads coupled with ST-elevation in aVR and/or VI but is otherwise unremarkable, the automated [computerized] interpretation should suggest ischemia due to multi-vessel or left main coronary artery
obstruction.” A 2010 international consensus document13 provided similar STEMI-equivalent criteria for acute LMCO (or severe angiographic disease): a myocardial ischemia vector causing ST-depression in 6 or more leads (maximal in V2-V6), and associated ST-elevation limited to lead aVR and V1.

de Winter ST/T-wave complex

In 2008, de Winter et al38 described a novel STEMI-equivalent ECG pattern that signifies acute occlusion of the proximal left anterior descending (LAD). Following examination of their Dutch registry, the “de Winter ST/T-wave” pattern was first recognized in 30 unique patients (all with normal baseline serum potassium levels) and represented 2% of all angiographically confirmed anterior MIs. Diagnostic criteria on ECG include 1 to 3 mm of ST-depression that is up-sloping at the J-point in leads V1 through V6, and associated with persistently tall, upright, and symmetric precordial T-waves (Table I and online Appendix item II.c).

The de Winter ST/T-wave complex is distinct from transient hyper-acute T-waves (HATW) that may occur within minutes of an acute coronary occlusion (representing a chronic lesion), and demonstrated classic STEMI pattern.38,39 Transient HATW can be induced with experimental occlusion of a coronary for a few minutes, but they have not been used as inclusion criteria for STEMI clinical trials.

In addition, de Winter ST/T-wave complex should not be confused with Wellens syndrome,39 in which the ECG demonstrates either biphasic or inverted T waves in leads V2 through V3 (online Appendix item II.d). Wellens syndrome typically involves a chronic high-grade LAD stenosis that can usually be evaluated by non-emergent angiography.

Resuscitated cardiac arrest

For more than a decade, it has been known that approximately 50% of patients with out-of-hospital cardiac arrest (OHCA) and return of spontaneous circulation (ROSC) have an acute coronary artery occlusion when evaluated by immediate diagnostic angiography.40 Hence, in addition to the acute STEMI and STEMI-equivalent ECG criteria (Table I) reviewed thus far, certain scenarios of OHCA with ROSC appear to be gaining acceptance as a trigger for appropriate Cath Lab activation (Table II). Many OHCA patients simply represent a dramatic presentation of an acute coronary occlusion, with sudden death as both the chief complaint and the clinical STEMI-equivalent. Neither the ACC/AHA guidelines nor the European guidelines1-3,5 comment on the role of immediate diagnostic coronary angiography for OHCA patients, but both acknowledge that most of the deaths in STEMI patients occur from lethal arrhythmias within 1 to 2 hours of symptom onset.

Historically, the practice of taking resuscitated OHCA patients for emergency coronary angiography and planned PPCI was rare and usually restricted to a small cohort of “ideal” patients: witnessed arrest, early chest compressions, early defibrillation of a shockable rhythm (ventricular tachycardia or fibrillation), early ROSC, hemodynamically and electrically stable on arrival to the hospital, and clinically perceived on initial assessment to have experienced only minimal neurologic injury from ischemia. Although randomized controlled trials of STEMI almost uniformly exclude OHCA, observational registry data has gradually accumulated and demonstrated good outcomes in resuscitated OHCA patients who traditionally have been considered “less than ideal” candidates for Cath Lab intervention. Thus, both the 2008 International Liaison Committee on Resuscitation41 consensus statement and the 2009 state-of-the-art paper by Ewy and Kern12 recommended aggressive postresuscitation care involving both PCI and invasive hemodynamic support for a greater proportion of patients resuscitated from OHCA.

Despite the International Liaison Committee on Resuscitation consensus statement, controversy still exists regarding the ability of a postresuscitation ECG to identify patients with an acute coronary occlusion.43,44 However, the largest experience to date is the PROCAT45 registry, which provided compelling new evidence supporting a routine Cath Lab based treatment pathway for OHCA regardless of ECG pattern after resuscitation. In the PROCAT registry spanning 2003 to 2008, all 435 resuscitated OHCA patients with no obvious extracardiac cause of arrest received emergency coronary angiography. Of the 134 patients with a post-ROSC ECG demonstrating classic STEMI, 128 (96%) had at least one significant lesion (>50% reduction in luminal diameter) and 99 (74%) underwent successful PCI. In the remaining 301 patients without classic STEMI on post-ROSC ECG, 176 (58%) had a significant lesion and 78 (26%) received PCI. Multivariable analysis demonstrated successful PCI to be an independent predictor of survival (OR 2.1, 95% CI 1.2-3.7), irrespective of postresuscitation ECG pattern. As compared to a historically low rate of survival (<10%),45 overall survival to hospital discharge was 40% in the PROCAT registry. Moreover, there was a 94% rate of favorable neurologic outcomes in a cohort with a 68% rate of shockable first rhythm and 85% rate of in-hospital therapeutic hypothermia.

A recent AHA policy statement proposed the development of cardiac resuscitation centers46 in association with an existing network of STEMI receiving centers.11,22 The statement emphasized that optimal postresuscitation care is complex, multi-disciplinary, and is probably most efficiently provided in designated hospitals via a coordinated systems-based approach that is supported by a robust quality improvement infrastructure. Three different
prehospital scenarios warrant consideration with regard to appropriate Cath Lab activation (Table II). Furthermore, within each general scenario, real-world clinical decision-making likely involves assessment of the “down time” before initiation of certain critical prehospital interventions (ie, time delay from patient collapse to 9-1-1 call, to chest compressions, and to defibrillation) and each patient’s clinical status upon hospital arrival.

Appropriate patient selection

The final decision point before a Cath Lab activation involves clinical correlation, and hence our deliberate use of the term appropriate Cath Lab activation. This broader term includes both traditional “false-positive” Cath Lab activation (ie, technical competency in ECG interpretation for STEMI and STEMI-equivalents) as well as physician judgment regarding whether or not an individual patient is a “candidate” (class I-A recommendation) for PPCI. Identification of STEMI and STEMI-equivalent patients for whom Cath Lab activation is “inappropriate” requires frontline physicians to document a brief yet careful assessment of the overall clinical scenario, age, comorbidities, functional status, do-not-resuscitate status, family perspective, and patient preferences.

New findings are also informing appropriate patient selection strategies in patients resuscitated from sudden cardiac arrest. Interest in early Cath Lab activation and PPCI-based myocardial reperfusion has traditionally been overshadowed by significant concerns regarding meaningful neurologic recovery from the initial anoxic insult. However, data have gradually accumulated from both clinical trials and observational registries supporting the ability of therapeutic hypothermia to significantly improve clinical trials and observational registries supporting the ability of therapeutic hypothermia to significantly improve meaningful neurologic recovery from the initial anoxic insult. However, data have gradually accumulated from both clinical trials and observational registries supporting the ability of therapeutic hypothermia to significantly improve functional neurologic status immediately after ROSC should not preclude early Cath Lab activation because current AHA expert statements11,46 emphasize deferral of cerebral recovery prognostication for at least 72 hours after collapse in patients treated with therapeutic hypothermia. Furthermore, although the “best” hypothermia protocol has yet to be definitively proven, current consensus41,46 suggests initiating cooling as early as possible (ie, pre-arrival to the Cath Lab) using some approximation of a 4/24/8@33 protocol (4-hour induction [1°/h] to a target temperature of 33°C, 24-hour maintenance at 33°C with minimal temperature variation, and 8 hours of rewarming (0.5°C/h) back to normothermia.

Conclusion

In summary, broad awareness should exist regarding evidence-based triggers for appropriate Cath Lab activation. A diverse group of frontline clinicians making these time pressured decisions need a comprehensive list of precise criteria, because not all “acute MIs” have classic ST-elevation on ECG (eg, STEMI-equivalents and certain OCHA scenarios), not all ST-elevation patterns represent “true STEMI” (ie, STE-mimics), and some “true STEMI” patients are not reasonable candidates for an aggressive treatment strategy involving PPCI. Optimal ECG interpretation proficiency by all clinicians in identifying both classic STEMI and STEMI-equivalents constitutes a major cornerstone of ongoing efforts to maximize STEMI system efficiency.

Disclosures

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References

Appendix. Online Data Supplement

Title: Appropriate cardiac catheterization lab activation: optimizing electrocardiogram interpretation and clinical decision-making for acute ST-elevation myocardial infarction
Authors: Rokos, French, Mattu, Nichol, Farkouh, Reiffel, and Stone.

14 Electrocardiogram (ECG) Examples

I. Classic STEMI
   a. Anterior-STEMI with reciprocal changes ⇒ proximal LAD occlusion
   b. Anterior-STEMI without reciprocal changes ⇒ middle LAD occlusion
   c. High-lateral STEMI ⇒ diagonal branch occlusion
   d. Lateral STEMI ⇒ left circumflex occlusion

II. STEMI-equivalents
   a. Posterior MI (isolated)
   b. Left main coronary occlusion: lead aVR STE and infero-lateral STD
   c. de Winter ST/T-wave complex
   d. Wellens syndrome (distinguish from de Winter ST/T-wave complex
   e. LBBB with Sgarbossa criteria

III. ST-elevation mimics (STE-mimics)
   a. Benign early repolarization (BER)
   b. Acute pericarditis
   c. Left ventricular aneurysm
   d. Normal-variant chronic ST-elevation
   e. Brugada syndrome
I.a Anterior STEMI with reciprocal changes  
(IRA = proximal LAD)

I.b Anterior STEMI without reciprocal changes  
(IRA = middle LAD)
I.c “High” Lateral STEMI
(JRA = Diagonal branch)

I.d Lateral STEMI
(JRA = Left Circumflex)
II. STEMI-equivalents

II.a Posterior MI (Isolated)

II.b Left Main coronary occlusion (LMCO): Lead aVR with ST-elevation and associated infero-lateral ST-depression
II.c de Winter ST/T-wave complex

II.d Wellens syndrome
(This is not a STEMI-equivalent. Example provided to compare with leads V2-V3 of de Winter ST/T-wave complex)
II.e LBBB MI with Sgarbossa criteria concordance in V₂ through V₅

III. ST-elevation mimics (STE-mimics)

III.a Benign early repolarization (BER)
III.b Acute pericarditis

III.c Left Ventricular aneurysm
III.d Normal-variant chronic ST-elevation

III.e Brugada syndrome