# ST-Segment Elevation Myocardial Infarction: Part II Management

L C Ikpeama, S D Ruppert

## Citation

## Abstract

ST-segment elevation myocardial infarction (STEMI) is a medical emergency where the coronary artery previously affected by atherosclerosis is abruptly occluded by a thrombus at the site of vascular injury. Oxygen and blood supply are blocked off to the heart muscle being supplied by the affected artery, causing death to the muscle. Outcomes following a STEMI may range from mild to severe heart failure to multi-system organ-failure and death. Immediate activation of the emergency cardiology STEMI team for reperfusion therapy followed by necessary medical treatments give the patient with STEMI possible improved outcomes. An interprofessional team approach is needed to maximize care and make critical treatment decisions aimed at providing timely treatment and mitigating complications. This second of a 2-part series of articles highlights specific effective treatments that support early goal achievement to improve outcomes for the patient, as well as common complications that might arise even when treatments are appropriately given. The significance of interpreting the physical and diagnostic data in STEMI cases was previously discussed along with the related pathophysiology in Part I.1

## INTRODUCTION

ST elevation myocardial infarction (STEMI) is a medical emergency, and continues to cause significant problems in both developing and developed countries. STEMI occurs when a coronary artery, previously affected by atherosclerosis, is abruptly occluded by a thrombus at the site of a vascular injury. This occlusion completely blocks off oxygen and blood supply to the heart muscle being supplied by the affected artery resulting in ischemia and ultimate death of heart muscle. Of the 683,000 patients hospitalized in the United States (US) in 2009 with acute coronary syndrome, 25-40% were STEMI.2 Availability of effective, time-sensitive treatment for STEMI and the emphasis on affordable health care is the impetus for clinicians to ask the right questions, obtain the right answers, institute the appropriate interventions to maximize patient outcomes, and reduce length of hospital stay.

In the past decade, improved outcomes in STEMI cases have been due to factors which include increased awareness of the disease, early recognition and diagnosis, and timely reperfusion therapy combined with adjuvant medical treatment.3 The importance of prompt recognition and triage of patients with chest pain or signs/symptoms suggestive of acute coronary syndrome (ACS) must be followed by quick assessment that includes obtaining an electrocardiography (ECG), preferably pre-hospital by emergency medical services, to confirm diagnosis of STEMI or non-STEMI, and aid in determining appropriate transport to percutaneous coronary intervention (PCI)-capable or non-PCI-capable hospital.2

Treating the patient with a STEMI is complex and an interprofessional team effort that includes emergency department providers, the rapid response team, cardiologists, nurses, and support staff working to achieve multiple goals that include relieving ischemic pain, timely and appropriate reperfusion therapy, mitigating and treating complications, and providing follow-up for the surviving patient. This second of 2 articles highlights specific effective treatments based on the American College of Cardiology (ACC)/American Heart Association (AHA) guideline published in 2004 with a focused update in 2009 and revision by the American College of Cardiology Foundation (ACCF)/AHA in 2013 that supports early goal achievement for improved outcomes for the patient with a STEMI.2,4,5 Complex treatment plans that include primary PCI and adjuvant medical therapies are reviewed.

## INITIAL PLAN

Activate facility's emergency STEMI team pager stat

STEMI confirmed in ECG by qualified medical personnel identifies patients who will benefit from immediate reperfusion therapy.4 Patient survival with good outcomes depends on immediate restoration of retrograde coronary blood flow, the time taken to achieve this, and continued patency of the affected vessel.6,7 Facilities are charged to engage a interprofessional team that emergently responds at identification of STEMI, and uses structured protocols, checklists, or clinical pathways designed for triaging and managing patients with symptoms suggestive of STEMI in the field, emergency department, and in-hospital.2 An organized, well defined process is needed to minimize the time to therapeutic treatment.

Brodie et al.8 tested the impact of timing of reperfusion therapy on patient mortality, and evaluated the impact of door-to-balloon (DBT) time on mortality depending on clinical risk and time of presentation. These researchers found that a DBT that is less than or equal to 90 minutes is associated with a lower mortality rate in patients with early presentation, but has less impact on the mortality rate in patients presenting later. The absolute mortality rate reduction with short DTB is greatest in high-risk patients presenting early.8 ACCF/AHA recommend that the delay from patient contact with a health care provider to initiation of fibrinolytic therapy, which is referred to as door-to-needle time, should be less than or equal to 30 minutes.2If percutaneous coronary intervention (PCI) is chosen, DTB time less than or equal to 90 minutes is recommended.2

Give oxygen supplementation via nasal cannula

Supplemental oxygen (2-4 liters/minute) should be administered to patients with arterial oxygen less than 90%, dyspnea, or heart failure since ventilation-perfusion mismatch may occur. Supplemental oxygen can increase coronary vascular resistance, and should also be used cautiously in patients with chronic obstructive pulmonary disease.2,9

Administer nitroglycerin

The current ACCF/AHA practice guideline recommends that patients with ischemic-type chest pain receive nitroglycerin (NTG), a nitrate, 0.4 mg sublingually up to 3 consecutive doses administered 5 minutes apart for ongoing chest pain.2 NTG reduces chest pain by vasodilatation of both peripheral arteries and veins, relaxing coronary arteries, and improving coronary blood flow. Patients with coronary spasms also benefit from the vasodilatation of coronary arteries seen with

NTG therapy. Intravenous NTG can be administered for patients who have ischemic pain, hypertension, and pulmonary congestion at a rate of 10 micrograms per minute (mcg/min) with titration to desired blood pressure effect.2 Nitrates should not be administered to a patient with STEMI whose systolic blood pressure is either less than 90 or 30 mm Hg below baseline, who has marked bradycardia or tachycardia, or who has used 5´-phosphodiesterase inhibitor within the previous 24-48 hours.2

Administer morphine sulfate intravenously

Acute relief of myocardial ischemic pain should be one of the primary goals of management in STEMI. Severe pain contributes to increased adrenergic activity with subsequent increase in myocardial oxygen demand. Morphine also alleviates the work of breathing, aids in reducing anxiety, and positively affects ventricular loading.2 Intravenous administration of morphine sulfate in 4-8 milligrams (mg) initially is the analgesic of choice for myocardial ischemic pain for the patient with STEMI. Doses of 2-8 mg should be given every 5-15 minutes as needed for cardiac pain.2 Elderly patients should receive lower doses.

Administer antithrombotic therapy before reperfusion therapy

Administer aspirin 325 mg chew and swallow
Aspirin is an effective antiplatelet therapy for patients with ACS and is essential in the management of those with suspected STEMI. Aspirin in the dose of 162-325 mg tablet should be administered and chewed by all patients suspected of having STEMI who has no contraindication for aspirin therapy.2 Aspirin inhibits thromboxane A2 production by producing a rapid clinical antithrombotic effect.4 Because this medication decreases mortality in MI, aspirin should be administered as early as possible and continued indefinitely in patients with ACS.

In the International Study of Infarct Survival 2 (ISIS-2) study, 77,187 patients with STEMI were randomized to treatment with aspirin, streptokinase, or placebo. The result of this 5-week study showed that the effect of aspirin was as great as that of streptokinase alone, and the benefits of each were partially additive. Results showed a decrease in the incidence of vascular death by 23% with aspirin and heparin, by 25% with streptokinase and heparin, and by 41% with the combination of all 3 agents.10

P2Y12 receptor antagonists
Oral thienopyridines are adjuvant antiplatelet agents whose

active metabolites inhibit the activation and aggregation of platelets by the binding of adenosine diphosphate (ADP) to the P2Y12 receptor.[4,11] ACCF/AHA recommends a loading dose of a P2Y12 receptor inhibitor (clopidogrel 600 mg, prasugrel 60 mg, or ticagrelor 180 mg) be given as early as possible before or at the time of primary or non-primary PCI.[2] In patients that received a bare metal stent (BMS) or drug-eluting stent (DES) during PCI, the P2Y12 inhibitor therapy duration guideline recommends a daily maintenance dose (clopidogrel 75 mg, prasugrel 10 mg, or ticagrelor 90 mg) for at least 12 months in the absence of bleeding problems, but can be discontinued earlier if the risk of bleeding outweighs the benefit of the medication.[2] P2Y12 agents are used in conjunction with aspirin as dual anti-platelet therapy (DAPT).

Clopidogrel provides greater anti-platelet effects than aspirin, and reduces the risk of death and cardiovascular complications in patients receiving PCI.[12,13] An increased loading dose of clopidogrel (600 mg) has shown significant improvement on outcomes for STEMI patients undergoing PCI ischemic adverse event rates compared with a 300 mg loading dose. Mangiacapra et al.[14] enrolled 384 consecutive patients who presented with STEMI and infarct-related artery of thrombolysis in myocardial infarction (TIMI) grade of 0 or 1 who had PCI from January 2006 to December 2007, and analyzed whether a 600 mg loading dose of clopidogrel was associated with better procedural and 1-year clinical outcomes compared to a 300 mg loading dose. Prospectively, 157 patients were included in the 600 mg group, and 98 in the 300 mg group. Results showed a significant lower incidence of post-PCI myocardial blush grade 0 or 1, and significantly less common no-reflow phenomenon in the 600 mg group compared to the 300 mg group. The researchers concluded that a 600 mg loading dose of clopidogrel was associated with improvements in procedural angiographic end points as well as 1-year clinical outcomes in patients with STEMI who undergo primary PCI compared with a 300 mg loading dose. However, response to clopidogrel can be reduced in the 20-30% of patients with the CYP2C19 allele.[2]For patients with known variants, treatment with an alternate agent such as prasugrel or ticagrelor would be advisable.

Prasugrel has been shown to produce greater platelet aggregation inhibition than clopidogrel.[15]Thirty day stent thrombosis was also significantly lower with use of this agent. However, prasugrel does have a higher risk of bleeding and should not be used in patients with stroke or transient ischemic attack. This agent shows no benefit in

patients older than 75 years of age or who weigh less than 60 kilograms.[15]

Ticagrelor, a non-thienopyridine P2Y12 receptor agonist, has been shown to have lower rates of stent thrombosis and death than clopidogrel.[16] However, numbers of intracranial hemorrhage and strokes are higher. Thus, in patient who will be on long term therapy with this medication, daily aspirin dose should not exceed 100 mg.[2]

For patients receiving fibrinolytic therapy as a primary modality, a loading dose of clopidogrel 300 m should be administered (75 mg dose if the patient is over 75 years of age).[2] As part of DAPT with aspirin, the clopidogrel should be continued for at least 14 days and up to 1 year after fibrinolytic therapy is received.[2]

Glycoprotein IIB/IIIA inhibitor

A platelet molecule contains approximately 90,000 glycoprotein (GP) IIb/IIIa receptors, and platelet agonists such as epinephrine, collagen, serotonin, and thrombin converge on receptor sites to promote platelet aggregation.[17] Intravenous GP IIb/IIIa receptor inhibitors are potent agents that inhibit the final common pathway for platelet aggregation. These agents are recognized as supportive anti-platelet therapy in patients undergoing PCI. An intervention review assessed the effects and safety of IIb/IIIa blockers when administered during PCI. Forty-eight RCTs of 62,417 patients were included in the review, and showed that IIb/IIIa blockers decreased mortality at 30-days and at 6-months when administered during PCI. Myocardial infarction and death were also slightly decreased at 30-days and 6-months, although risk of severe bleeding was increased. However, their efficacy was reduced in patients who were being treated with clopidogrel.[18]

Use of these agents was established before the use of DAPT with a P2Y12 agent and aspirin. Use of GP IIb/IIIa agents is now limited mainly to cases of a large thrombus burden or when inadequate P2Y12 receptor antagonist loading occurs.[2]

Administer beta-blocker

Oral beta blocker therapy is recommended for patients with acute STEMI and should be initiated promptly or within 24 hours of the cardiac event for patients without signs of heart failure, hypotension, risk for cardiogenic shock, or other contraindications to beta blockade such as heart block, active asthma or reactive airway disease.[2] Beta blockers have been shown to prevent recurrent ischemia and life-threatening

ventricular arrhythmias and should be offered to patients receiving primary PCI or fibrinolysis. Beta blockers diminish myocardial oxygen demand by decreasing heart rate, systemic arterial pressure, and myocardial contractility.4 If a patient has an initial contraindication to administration of a beta blocker, use should be reevaluated after 24 hours.2 Beta blocker therapy should be continued throughout hospitalization and after discharge if no contraindications exist. The ACCF/AHA guideline recommends titrated use of metoprolol or carvedilol as beta blockers of choice.2

Administer renin-angiotensin-aldosterone system inhibitor Numerous studies have demonstrated the reduction of fatal and nonfatal cardiovascular events in patients with STEMI with the use of renin-angiotensin-aldosterone system inhibitors. An angiotensin-converting enzyme (ACE) inhibitor is recommended to be initiated within the first 24 hours in patients with anterior location STEMI whose ejection fraction (EF) is less than or equal to 40%, without contraindications such as hypotension, shock, renal failure [with or without ACE inhibitor therapy], bilateral renal stenosis, or drug allergy/intolerance.2 In ACE inhibitor intolerant patients, an angiotensin receptor blocker (ARB) can be used. An aldosterone antagonist is recommended to be added for patients with STEMI who have an EF less than or equal to 40% and either heart failure symptoms or diabetes. The aldosterone antagonist is considered an additional agent for patients already receiving ACE inhibitors and beta blockers.2

## REPERFUSION THERAPY

The main goal of STEMI management is rapid, effective, and maintenance of reperfusion to establish coronary blood flow to ischemic areas of the myocardium. Reperfusion therapy should be administered within 12 hours of symptom onset.2 All effective medical systems must strive to achieve this goal through improvement of door to needle and door to balloon times, usually 30 minutes and 90 minutes respectively.4 Even with adequate reperfusion and restoration of flow, perfusion of the infarct zone may still be compromised by microvascular damage and reperfusion injury.4 This effect may be due to downstream embolization of platelet particles and microemboli from activated platelets that make conditions favorable for occlusion and spasm in the microvascular bed. Cellular edema, free radical formation, calcium overload, and apoptotic process may occur. Cytokine activation causes neutrophil accumulation and inflammatory mediators that contribute to tissue injury.4

Reperfusion can be achieved either pharmacologically (fibrinolysis) or by PCI or both (fibrinolysis-facilitated primary PCI), or surgical intervention (coronary artery bypass grafting (CABG). Selection of a specific therapy involves assessment of variables such as time from onset of symptoms, risk of bleeding, risk of STEMI, the time for transport, and availability of a skilled PCI lab.4 Any chosen option accelerates the opening of occluded arteries and greatly increases the number of patients in whom restoration of flow in the infarct-related artery is accomplished. The timely restoration of flow and improved perfusion of the downstream zone of infarcted myocardium through PCI or fibrinolysis is the key determinant of short and long term outcomes. Additional interventions such as minimizing tachycardia and treating hypertension, heart failure (HF), and pain help to maintain optimal balance between oxygen supply and demand and add to further protect ischemic myocardium.

Primary percutaneous coronary intervention (PCI)

Primary PCI mechanically disrupts the occluded coronary artery by directly compressing the underlying thrombotic stenosis either with the use of balloon or coronary stent deployment to rapidly restore blood floor to the ischemic area of the myocardium.19 When performed timely and accurately, this procedure is the reperfusion therapy of choice for patients with acute STEMI compared to fibrinolysis. Primary PCI is superior to fibrinolysis in achieving TIMI 3 normal flow 93-96% versus 50-60%, 20,21and has lower rates of recurrent ischemia, reinfarction, and death.22

Coronary stents (BMS and drug-eluting stents [DES]) are metal mesh tubes inserted by interventional cardiologists to keep narrowed or blocked coronary arteries open during angioplasty. BMS are metal stents with no special coating. These stents act simply by forming scaffolding that props open coronary arteries. As time passes, the arteries heal and tissue grows over the stent, holding it in place. DES are coated with slow releasing medication that prevents the growth of scar tissue in the artery wall, and were developed for this special characteristics, to help prevent re-blockage caused by tissue over-growth which was noticed in some patients with BMS.23 However, no significant differences in death, MI, or vessel blockage between DES and BMS usage have been documented.23

Fibrinolysis

The principal goal of fibrinolysis is prompt restoration of full coronary arterial blood flow. This therapy can be administered before arrival to the hospital and has shown mortality reduction by up to 17%.4Fibrinolysis reduces the relative risk of in-hospital death by up to 50% when administered within the first hour of the onset of STEMI symptoms.23 Fibrinolysis can be a  beneficial reperfusion strategy for patients presenting within 12 hours of symptoms, if problems occur with transportation to of availability of a suitable PCI center occurs.2 This therapy should be given if primary PCI cannot be performed within 120 minutes of first medical contact.2 Pre-hospital fibrinolysis has been shown to reduce treatment delays by 1 hour, and mortality by 17%.24

Agents like streptokinase, tenecteplase (TNK), and reteplase (rPA) used in patients with STEMI all act by promoting the conversion of plasminogen to plasmin, which subsequently lyses fibrin. When used appropriately, fibrinolytic agents reduce infarct size, limit left ventricular (LV) dysfunction, and reduce the incidence of serious complications such as septal rupture, cardiogenic shock, and malignant ventricular arrhythmias.23 Patients who are age 75 years or less show a greater relative reduction in mortality with fibrinolytic therapy that those greater than 75 years, as well as absolute mortality rate (15-25%).23 Due to bleeding risks, absolute and relative contraindications exist for the use of thrombolytic agents for patients with STEMI (see Table 1).

**TABLE 1**

Contraindications/Cautions for the Use of Thrombolytic Agents2

| Absolute Contraindications | Relative Contraindications |
|---|---|
| Prior intracranial hemorrhage | History of chronic, severe, poorly controlled hypertension |
| Active bleeding or bleeding diathesis | |
| Suspected aortic dissection Malignant brain neoplasm | Severe hypertension or systolic blood pressure > 180 or diastolic 110 mm Hg History of prior ischemic stroke (> 3 months) |
| Structural cerebrovascular lesion (e.g.; arteriovenous malformation) | Known intracranial pathology (not covered in absolute contraindications) |
| Ischemic stroke within the past 3 months (except ischemic stroke within 4.5 hours) | Active peptic ulcer disease |
| Closed facial of head trauma within the past 3 months | Major surgery < 3 weeks) |
| Intracranial or intraspinal surgery (within 2 months) | Recent internal bleeding (within 2-4 weeks) |
| Severe uncontrolled hypertension (unresponsive to emergency therapy) | Traumatic or prolonged (> 10 minutes) cardio-pulmonary resuscitation (CPR) |
| Prior streptokinase within the previous 6 months (contraindication for streptokinase only) | Non-compressible vascular punctures |
| | Oral anticoagulant therapy |
| | Pregnancy |
| | Dementia |

Adjunctive anticoagulation therapy should be used for at least 48 hours after reperfusion via fibrinolytic therapy. Therapy should preferable be continued up to 8 days or until the patient undergoes revascularization.2 Immediate arrangements should be made for transfer to a PCI-capable hospital if fibrinolysis was performed in a facility without this capability.

PCI versus Fibrinolysis

Multiple studies have validated better outcomes for reperfusion from primary PCI when compared with fibrinolysis therapy.  A large meta-analysis that included all randomized controlled trials (RCTs) and observational studies published up to 2008 compared primary PCI and fibrinolysis therapy in STEMI, and found that primary PCI was associated with a 34% reductions in short term (less than or equal to 6 weeks) mortality in RCTs; and 23% lower mortality in observational studies.25 PCI demonstrated a 63% reduction in stroke in the RCTs, and 61% in observational studies compared with fibrinolysis. Long term (greater or equal to 1- year), RCTs showed that PCI was associated with a 24% reduction in mortality, and a 51% reduction in re-infarction, but no significant benefit in observational studies.25

A meta-analyses of 22 RCTs evaluated the efficacy and safety of primary PCI versus fibrinolysis (n = 6,763), and found that patients randomized to PCI had lower mortality than those randomized to fibrinolysis (5.3% vs. 7.9%).26 The reduction of relative mortality by primary PCI was similar at all levels of estimated risk. The researchers concluded that primary PCI is consistently associated with a strong relative reduction in 30-day mortality, irrespective of patient baseline risk, and should be considered as the first choice reperfusion strategy whenever possible.26

Primary PCI is especially beneficial for patients for whose diagnosis is in doubt, who have a high risk of fibrinolytic-associated hemorrhage, or who are in cardiogenic shock. However, PCI is limited to facilities that have the capability to perform the procedure. Ninety-five percent of patients that receive primary PCI obtain complete reperfusion versus 50-60% of patients treated with fibrinolytics. PCI is also associated with a lower risk of hemorrhagic stroke compared with fibrinolysis.27

Initial therapy with PCI requires that a patient present early after symptom onset or can be rapidly transported to a PCI capable facility.  However, patients may delay in seeking medical care after symptom onset or timely transport to a

PCI capable facility may not be feasible. In a study of 1892 patients with STEMI who presented within 3 hours of symptom onset but who were unable to undergo primary PCI within 1 hour, Armstrong et al.[28] found that fibrinolytic therapy prior to transport to a PCI capable hospital resulted in effective reperfusion. In the fibrinolysis group, emergency coronary angiography was required in 36.3% of patient due to failed therapy while the remaining 64% of patients underwent angiography at a median of 17 hours. Complications of death, shock, heart failure, or reinfarction (within 30 days) occurred in 12.4% of the fibrinolysis group as compared to 14.3% in the primary PCI group. The fibrinolysis group had a slightly increased risk of intracranial bleeding (6.5%), but the rate was not significant (P = 0.11) as compared to the primary PCI group (4.8%).

## POST REPERFUSION THERAPY

Management of Complications and Conditions

Stent Thrombosis

BMS and DES, two different stents used during PCI, induce platelet aggregation and adhesions; antiplatelet agents are needed until the stents are covered with endothelial cells that do not induce thrombus formation. Stent thrombosis is a severe complication following stent implantation, and may occur early after implantation date to very late at more than 12 months after implantation.[29] Stent thrombosis increases risk of major cardiovascular events and is associated with very negative outcomes, with early stent thrombosis resulting in 29.4% rates of death, 76.5% MI, and overall mortality of 41.2%.[30] With antiplatelet therapy of aspirin, clopidogrel and GP IIb-IIIa inhibitors, complications of stent thrombosis have decreased from 10-15% to less than 2%; for patients who develop stent thrombosis, mortality can be up to 20-40%, with 70% developing acute MI.[31]

Predictors of stent thrombosis include the following: male gender, renal failure, diabetes, premature antiplatelet discontinuation, prior brachytherapy, bifurcation lesion, advanced age, unprotected left main artery, thrombus, unstable angina, and a host of other variables.[29] Data have been presented on a population of patients with risk of stent thrombosis due to clopidogrel resistance and non-responsiveness due to its variability in patients.[32] Variability to this antiplatelet therapy may be due to genetic variants, clinical conditions, and comorbidities.[17] Additional or different anti-platelet agents such as the third generation thienopyridines (prasugrel or ticagrelor) that irreversibly antagonizes the platelet adenosine diphosphate

(ADP P2Y) receptor may be the anti-platelet therapy of choice in combination with aspirin for this population of patients.[33]

Cardiogenic Shock

Cardiogenic shock caused by pump failure is the primary cause of in-hospital mortality for patients with STEMI. Most cases occur within 24 hours either at time of presentation or during the hospitalization.[2] The goals for treatment of cardiogenic shock include hemodynamic stabilization, increased tissue perfusion, adequate oxygenation, acid–base balance, plus rapid investigation of any potentially reversible causes for the patient's condition.[23,27]

Arrhythmias

Arrhythmias are common complications following a STEMI, and must be treated to prevent death. Atrial and ventricular tachycardia or fibrillation is caused by ischemia, slow infarcted myocardium conduction, electrolyte imbalances, and autonomic nervous system disturbances. Early treatment of ischemia, beta-blocker administration, electrolyte imbalance treatment, and the success of defibrillation or cardioversion have eliminated the need for prophylactic antiarrhythmic medications like lidocaine, which is no longer recommended in this patient population.[2] Patients with sustained ventricular tachycardia or fibrillation not due to transient or reversible causes may be candidates for an implantable cardioverter-defibrillator.

Pericarditis

Although the incidence of pericarditis has been reduced due to reperfusion therapy, this post-STEMI complication can result in recurrent chest pain. Aspirin is the recommended treatment agent. In aspirin intolerant or allergic patients, acetaminophen, colchicine, or narcotic analgesics can be used.[2]

Dyslipidemia

Lipid management is essential in lowering the risk of coronary health disease death and subsequent MI. In patients without contraindications to use, high intensity statin therapy should be initiated during hospitalization. Dose adjustment can be made post hospitalization to achieve LDL targets and lower non-HDL levels.[2]

## COMPREHENSIVE LONG-TERM

## CARE/CONTINUITY OF CARE

The interprofessional team must be involved in planning immediate and long-term care for the complex patient with a STEMI. Discussions must encompass maintenance treatment of co-morbidities such as diabetes, hyperlipidemia, hypertension, obesity, depression, and tobacco and alcohol use. Medication optimization and teaching on benefits of compliance can prevent worsening of the disease with subsequent frequent hospitalization. Continuing care of complications related to hospital course most often include treatment for deconditioning via a comprehensive cardiac rehabilitation program aimed at improving stamina and exercise tolerance. Patient and family education is crucial to reconcile medications, understand new disease states, recognize symptoms of myocardial infarction, and secure dates for any specialty appointments and diagnostic work follow-ups. Social services can direct patients to support groups and resources within their communities.

## NURSE PRACTITIONER ROLE

The nurse practitioner is involved in all aspects of care for the patient with a STEMI. As a healthcare team member in the forefront of care, the NP is present in primary care clinics, emergency departments, rapid response teams, or as acute care providers and must be competent in immediate triage, assessment, and interpretation of the ECG to recognize and diagnose patients with STEMI and initiate immediate evidence-supported treatment aimed at improving outcomes. Through highly developed skills of communication, team leadership, and care-coordination, the NP is able to interact with various specialties involved with care, and bring cohesiveness in the in-patient and long-term management of care. Additionally, the NP in an acute care setting is in a key position to facilitate transition across care settings and from hospital to home. The NP is an important resource for delivering needed education to the patient and family, and encouraging follow-up with the primary care provider, cardiologist, and any specialty referrals.

## CONCLUSION

STEMI is cardiovascular disease with significant morbidity and mortality, and must be recognized as a medical emergency. Prompt recognition of patient symptoms is crucial to immediate confirmation of STEMI by ECG. Treatment strategies include relieving ischemic pain, timely and appropriate reperfusion therapy, mitigating and treating complications, and providing follow-up for the surviving patient; all with the goal of maximizing outcomes. Treating the patient with a STEMI is complex and requires a coordinated and collaborative interprofessional team effort at all levels of the care process across settings.

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## Author Information

**Louisa Chika Ikpeama, DNP, RN, ACNP-BC Acute Care Nurse Practitioner**
Michael E. DeBakey Veteran Affairs Medical Center
Houston, Texas, USA

**Susan D. Ruppert, PhD, RN, ANP-BC, NP-C, FNAP, FCCM, FAANP, FAAN Professor**
The University of Texas Health Science Center at Houston School of Nursing
Houston, Texas, USA
Susan.D.Ruppert@uth.tmc.edu