JOINT TRAUMA SYSTEM CLINICAL PRACTICE GUIDELINE (JTS CPG)

Acute Coronary Syndrome (ACS) (CPG ID:86)

This CPG provides guidance on best management for ACS patients in deployed and resource constrained environments.

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OVERVIEW

Deployed medical providers at all roles of care must be prepared to recognize and manage Acute Coronary Syndrome (ACS). Under optimal conditions, treatment is initiated with medical therapy and may be followed by prompt coronary angiography and revascularization. Emergent percutaneous coronary intervention (PCI) is not available in most deployed locations however, and the time for such intervention is often dependent on long-range evacuation. This CPG provides guidance on best management for ACS patients in the deployed and resource constrained environment.

BACKGROUND

Coronary heart disease (CHD) is the leading cause of mortality in the U.S. and worldwide.[1,2] Cardiovascular health may in fact be worse for active duty servicemen when compared to the civilian population.[3] Additionally, cardiovascular disease is the second most common chronic disease among active duty Army personnel following arthritis.[4] While clinical CHD typically presents at older ages, significant coronary atherosclerosis and myocardial infarction can manifest in young adults and teenage patients.[5] In a cross-sectional study by Webber et al, coronary atherosclerosis was identified in 8.5% of the autopsies performed on U.S. military personnel in Operation Iraqi Freedom/Operation New Dawn (OIF/OND) and Operation Enduring Freedom (OEF).[6] In this study population, older age, lower educational level, higher body max index at military entrance, and a prior diagnoses of: dyslipidemia, hypertension, and obesity, were associated with a higher prevalence of atherosclerosis. Additionally, myocardial infarction (MI)/ACS was the most common medical/non-surgical diagnosis for Critical Care Air Transport Team (CCATT) evacuations, representing 6.6% of 290 patients evacuated by CCATT during OIF/OEF.[7]

Acute coronary syndrome is typically a consequence of CHD and refers to a spectrum of disease among patients experiencing, or suspected of experiencing, myocardial ischemia.[8] ACS is further divided into three subgroups: 1) ST elevation myocardial infarction (STEMI), 2) non-ST elevation myocardial infarction (NSTEMI), and 3) unstable angina.

When evaluating patients with chest pain concerning ACS, it is important to consider other life-threatening causes of chest pain such as pulmonary emboli, pneumonia, aortic dissection, pneumothorax, esophageal rupture, and myocarditis as they are managed differently. A thorough history and physical exam may be helpful in guiding the deployed provider towards a presumptive diagnosis. When available, imaging, labs, and electrocardiogram (ECG) can further assist in differentiating the etiology of chest pain.[9,10]

Diagnosis and management of ACS can be uniquely challenging in locations where PCI centers or treatment with fibrinolytics are not available. PCI is recommended within 120 minutes of first medical contact and is the preferred and most common treatment for ACS in the U.S..[11] However, if evacuation is required, the delay to reach a PCI center may exceed the recommended door-to-balloon time. Military physicians located Outside Continental United States (OCONUS) may therefore be required to treat ACS with fibrinolytics, which is not common in most U.S. locations. Additionally, many diagnostic tools helpful in evaluating chest pain, such as ECGs and cardiac troponin assays, may not be available in many deployed locations. Refer to Appendix C for ACS management recommendations by role of care.

It is the responsibility of every medical provider to be prepared for initial management of ACS, to include understanding how to use teleconference services. Diagnosis and treatment of ACS should be guided by expert consultation. In addition, the capability to obtain and transmit an ECG and to provide cardiac first aid treatments with oxygen, nitroglycerin and aspirin are key capabilities in initial stabilization and should be available to all deployed medical providers. Expanded capabilities for medical stabilization with anti-platelet agents, systemic anticoagulation and fibrinolytic therapy should be available in the evacuation chain within 24 hours, and must be considered during operational planning. Fibrinolytics are ideally given within 12 hours of symptom onset; however there may be a benefit up to 24 hours after symptom onset.[12,13]

DIAGNOSIS

ACS is a clinical syndrome that includes MI and unstable angina. An MI can present as either a STEMI or NSTEMI. A STEMI typically occurs as a result of plaque rupture and subsequent thrombosis of a coronary artery that leads to infarction and subsequent ECG changes. An NSTEMI does not have the ECG changes seen in STEMI but can also occur secondary to plaque rupture (Type 1 NSTEMI) or to inadequate blood flow to meet the metabolic demands of the myocardium in conditions like sepsis, pulmonary embolism, or coronary vasospasm (Type 2 NSTEMI). In either case, both STEMI and NSTEMI will result in an elevated troponin. Unstable angina is characterized by new onset chest pain, pain lasting greater than 20 minutes or pain at rest or with minimal exertion. While unstable angina can occur with ECG changes not diagnostic of STEMI, it does not have elevated troponins.[10] It is essential to differentiate between STEMI and NSTEMI since those with STEMI require emergent coronary intervention.[9] NSTEMI is more common than STEMI, representing 60-70% of myocardial infarctions.[14] NSTEMI with coronary occlusion is present in 25% of cases.[15]

ACS pain is classically described as exertional, pressure-like chest pain with radiation to the jaw, neck, one or both arms/shoulders, or back. Associated symptoms include nausea/vomiting, shortness of breath, and diaphoresis.[10] Atypical symptoms (dyspnea alone, fatigue, weakness, epigastric pain, palpitations, syncope, nausea alone, and cardiac arrest) are more common in the elderly, women, and diabetics. Atypical symptoms are presented symptoms in one-third of confirmed myocardial infarctions.[16]

Troponin is a protein released from myocardial tissue and is indicative of cardiac cell death. Serum troponin levels begin to rise measurably as early as 2 to 3 hours after onset and can remain elevated for up to 7 days.[10] When available, cardiac troponin assays (cardiac specific I or T) should be used in the diagnosis of MI. A troponin value above the 99th percentile upper reference limit (determined by the assay type) is considered positive. Troponin values may continue to rise, or they may decline depending on the timing of the clinical event, and both situations should raise concern for ACS.[10,17] Due to the time elapsed between cardiac cell death and laboratory detectable rise in troponin levels, the troponin level may be checked serially, usually every 3 to 6 hours.[18]

The following are clinical findings in combination with a rise and/or fall in troponin values that constitute the diagnosis of acute MI:[19]

- Symptoms of ischemia (See above paragraph.)
- New ischemic ECG changes
- Development of pathological Q waves

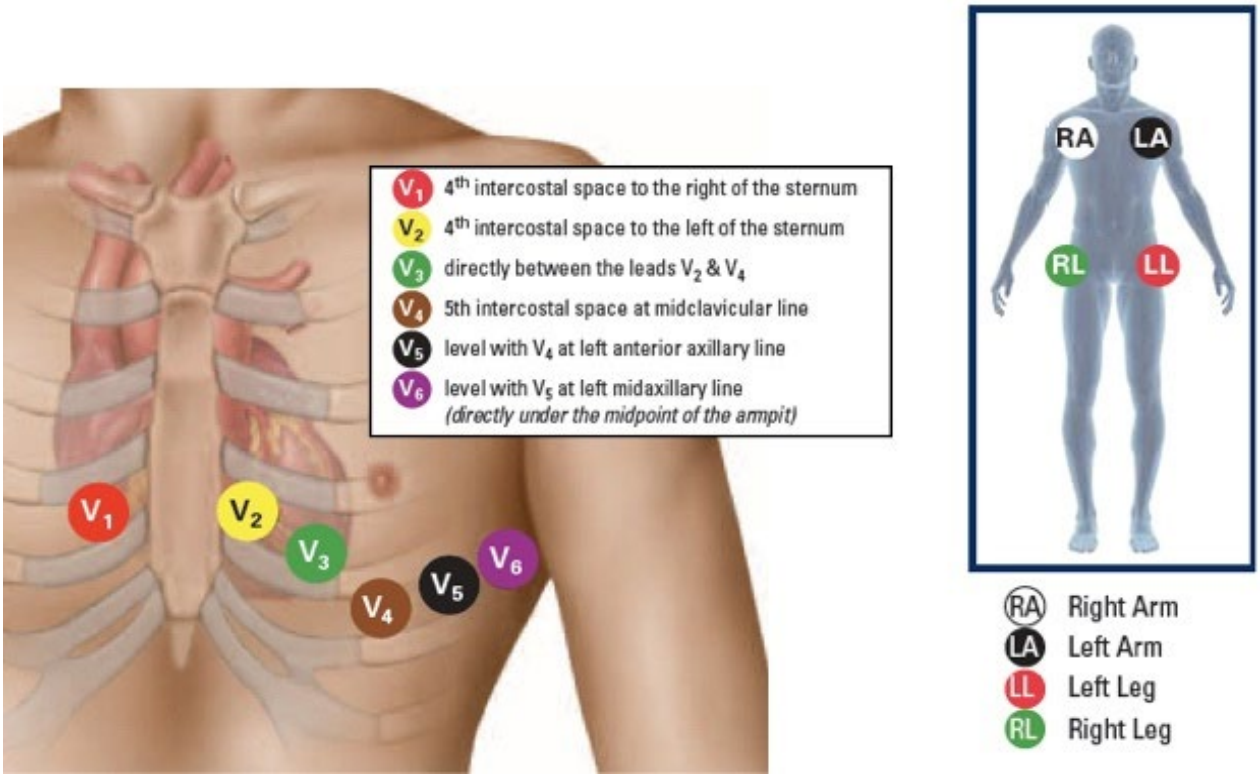
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
- Identification of an intra-coronary thrombus on angiography or autopsy

Other clinical conditions may present with elevated troponin. Some of these conditions may manifest symptoms similar to ACS (myocarditis, pulmonary embolism, and aortic dissection), while other conditions may present without features of ACS (such as rhabdomyolysis, sepsis and renal failure) and their management is distinct from that of ACS.[19] It is therefore important to consider the clinical manifestations and additional studies (ECG, echocardiography) in addition to troponin in the diagnosis of ACS. Keep in mind that in forward deployed locations, laboratory testing for troponin may not be available, and a high index of suspicion must be maintained in the presence of appropriate symptoms.

DIAGNOSIS OF ACS BY ECG CHARACTERISTICS

The 12-lead ECG of STEMI follows an anatomical distribution based on the infarcted artery. ST-elevation in one region should manifest with reciprocal ST-depression in the opposite anatomic region. ST-elevation must be present in two contiguous leads to be diagnostic. Figure 1 shows proper ECG lead placement for a typical 12-lead ECG.

Figure 1. Correct 12-Lead ECG Placement[20]



In conjunction with symptoms consistent with myocardial ischemia, the following are the ECG criteria diagnostics of STEMI:[19]

- New ST-elevations ≥ 0.1 mV in 2 or more contiguous leads except V_2-V_3
- New ST-elevations in V_2-V_3 with the below criteria:
  - ≥ 0.25 mV in men aged < 40 years
  - ≥ 0.2 mV in men aged ≥ 40 years
  - ≥ 0.15 mV in women

Figures 2 - 4 demonstrate typical patterns of anterior, inferior, and lateral wall STEMI, respectively.

Figure 2. 12-Lead ECG Evidence of Anterior STEMI. Notice the ST-elevations in leads I, aVL, and V_1-V_6 with reciprocal ST-depressions in the inferior leads (III and aVF)[21]

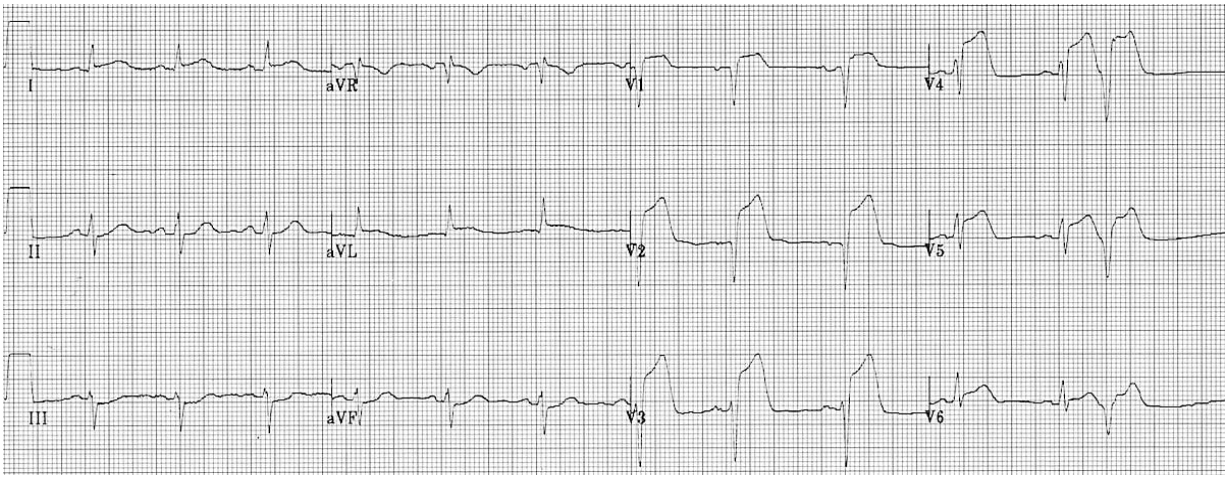


Figure 3. 12-Lead ECG of Inferior STEMI. Notice the ST-elevations in leads II, III, and aVF with reciprocal ST-depressions in antero-lateral leads (I, aVL,V_2, V_3 and V_4)[22]

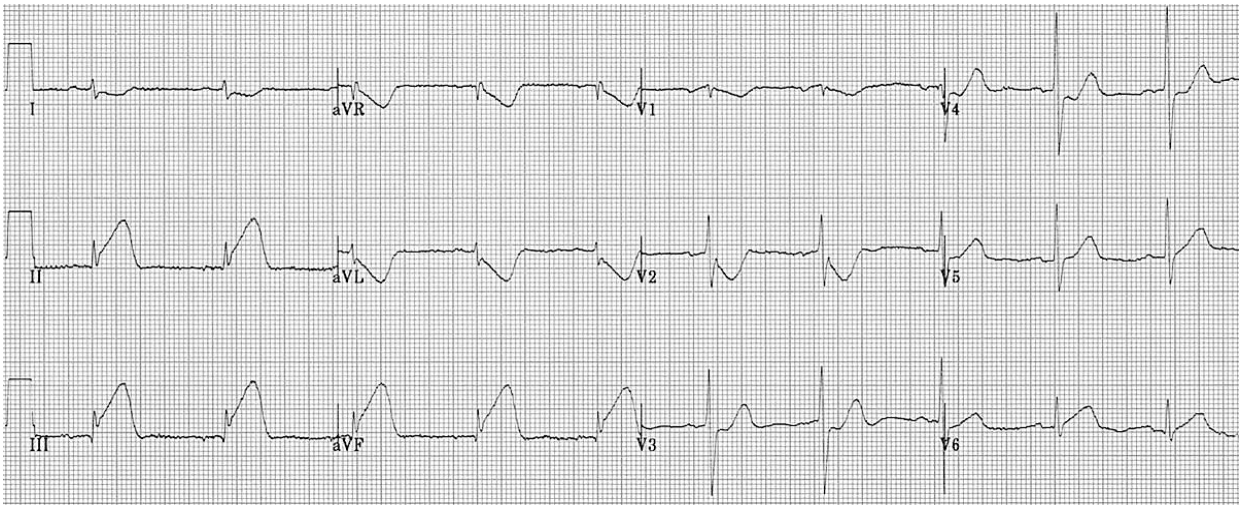
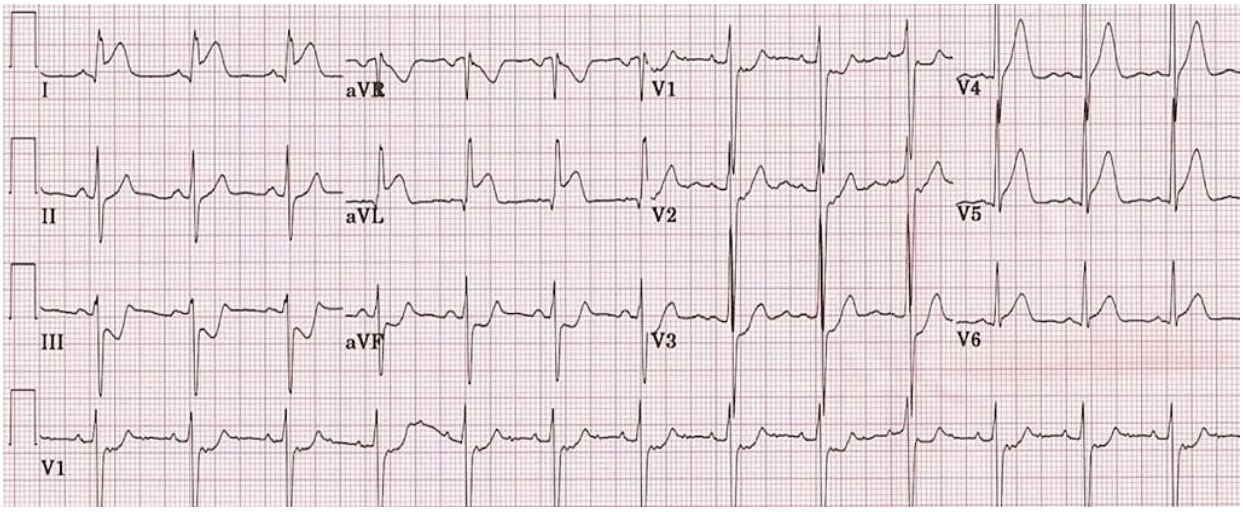


Figure 4. 12-Lead ECG of Lateral STEMI. Notice the ST-elevations in leads I, aVL, V_5, and V_6 with reciprocal ST-depressions in the inferior leads (II, III, and aVF) [23]



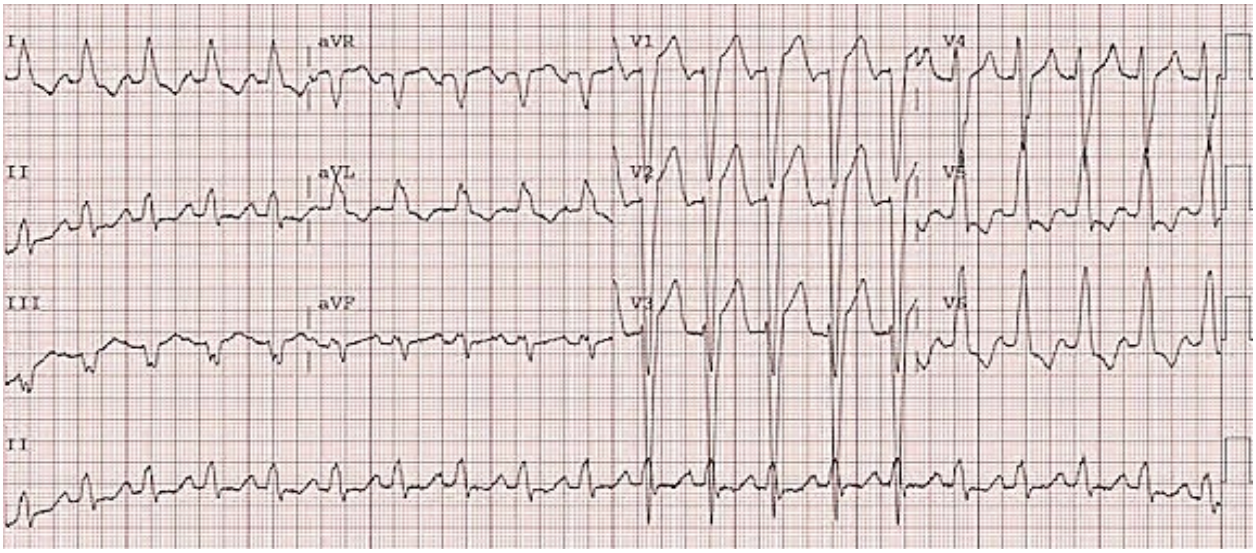
Outside of the standard ECG findings which are diagnostic of a STEMI previously mentioned, there is currently only one universally recognized "STEMI equivalent," which is a left bundle branch block (LBBB) meeting the Sgarbossa's criteria.[19]

There are other highly concerning ECG patterns that include the de Winter pattern, the left main pattern, hyperacute T waves, Wellen's syndrome and posterior MI. These patterns are not considered a STEMI equivalent but emergent/urgent revascularization with PCI that should be considered.[24] ECG interpretation may be complex and urgent teleconsultation with a cardiologist should be considered. ECG changes that should also raise concern for ischemia include ST depressions ≥ 0.5 mm (in ≥ 2 contiguous leads) or T-wave inversion.

1. **LBBB with Sgarbossa's criteria** – Sgarbossa's criteria was developed to identify an acute MI in the setting of a LBBB since this pattern can cause ST- and T-wave changes that make it difficult to identify an MI. The ECG is considered consistent with MI if any of the following three criteria are met and combine to equal ≥ 3 points:[25]
    - ≥ 1 lead with ≥ 1 mm of concordant ST-elevation with the QRS complex (5 points)
    - ≥ 1 lead of V_1-V_3 with ≥ 1 mm of concordant ST-depression with the QRS complex (3 points)
    - ST- elevation of ≥ 5 mm that is discordant with the QRS complex (2 points)
2. **Modified Sgarbossa criteria** (listed below) may outperform the original proposed criteria.[26] Any of the changes listed below are diagnostic of MI in the modified criteria.
    - ≥ 1 lead with ≥ 1 mm of concordant ST elevation with QRS
    - ≥ 1 lead of V1-V3 with ≥ 1 mm of concordant ST depression with QRS
    - ≥ 1 lead anywhere with ≥ 1 mm ST-elevation and proportionally excessive discordant ST-elevation, as defined by ≥ 25% of the depth of the preceding S-wave (an ST:S ratio of ≤ -0.25)

Figure 5. 12-Lead ECG of STEMI using the modified Sgarbossa' s criteria

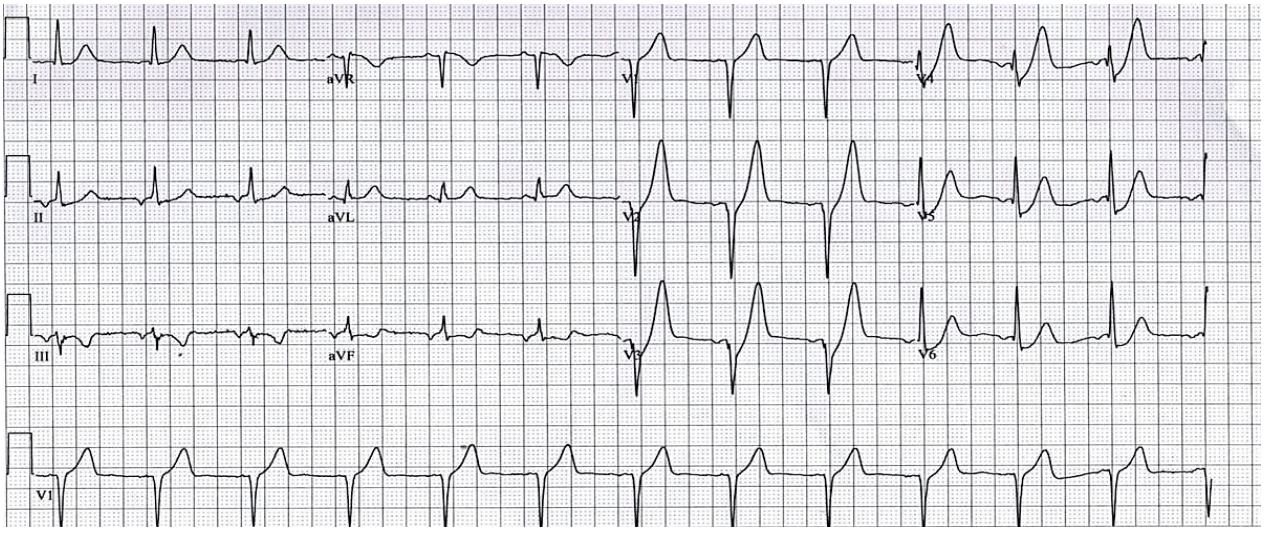
https://epmonthly.com/article/stemi-in-the-presence-of-lbbb/



ECG Patterns Highly Concerning for Myocardial Infarction

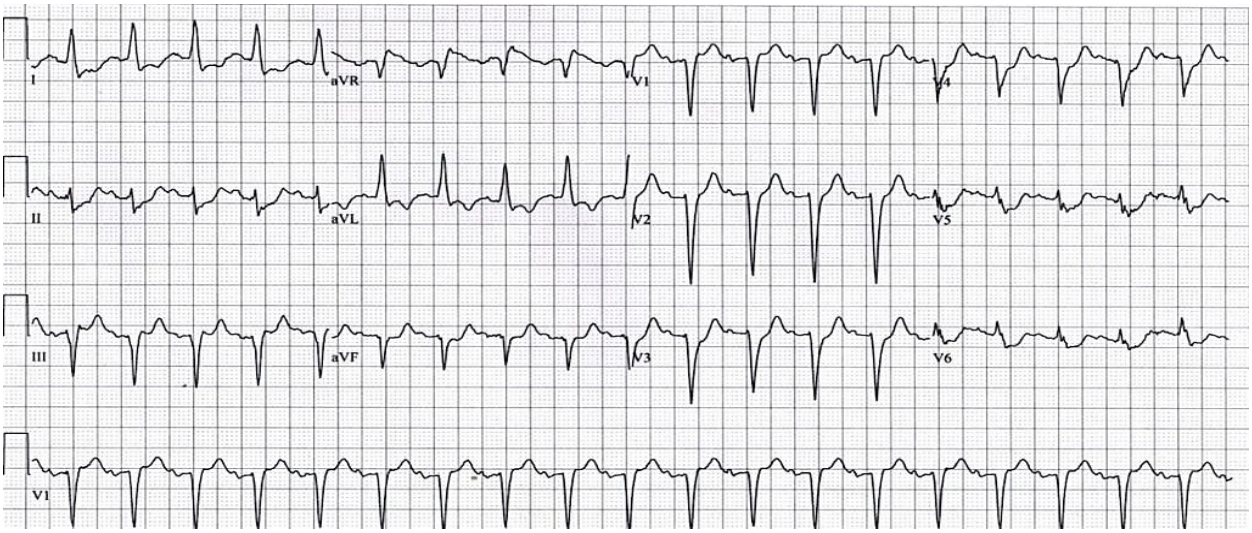
1. **De Winter pattern** – ST-depressions at the J point in V_1-V_6 and tall, peaked, symmetric T-waves are consistent with a de Winter pattern and associated with proximal left anterior descending artery (LAD) occlusion.

Figure 6. 12-Lead ECG of STEMI with De Winter pattern[27]



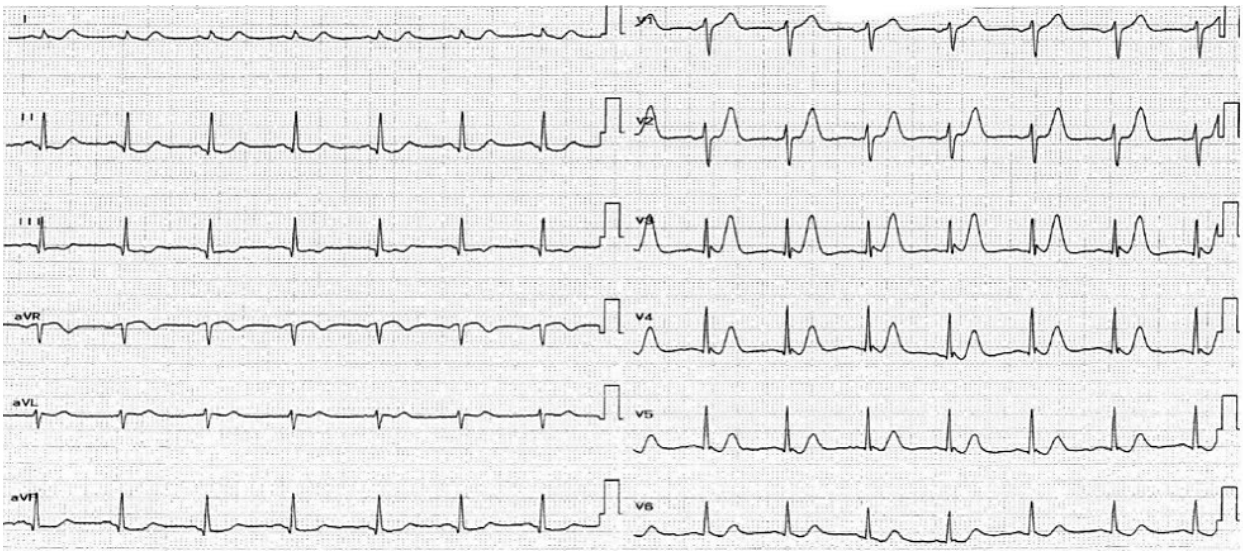
2. **Left main pattern** - Multi-lead ST-depression with coexistent ST-elevation in lead aVR has been described in patients with left main artery or proximal LAD occlusion. This is referred to as a left main pattern.

Figure 7. 12-Lead ECG of STEMI with left main pattern[28]



3. **Hyperacute T waves** – broad, asymmetrically peaked T-waves can be seen in early STEMI.

Figure 8. 12-lead ECG of STEMI with hyperacute T-waves[29]



4. **Wellen's syndrome** – Biphasic T-waves (Type A) or deep, symmetric T-wave inversions (Type B) in leads V_2-V_3 are strongly associated with proximal LAD occlusion. This is referred to as Wellen's syndrome.

Figure 9. 12-Lead ECG of STEMI with biphasic T-waves indicative of Wellen's syndrome[30]

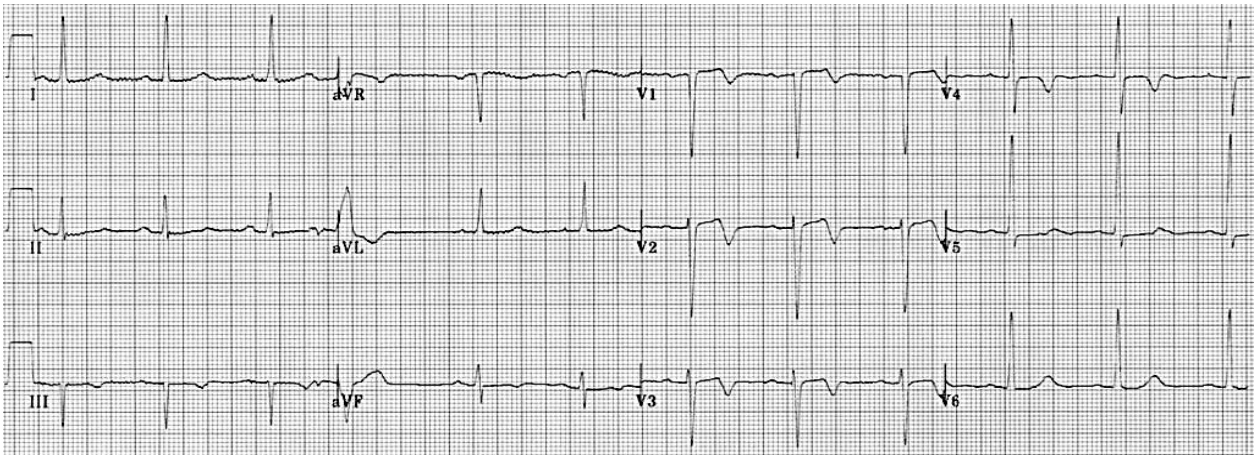
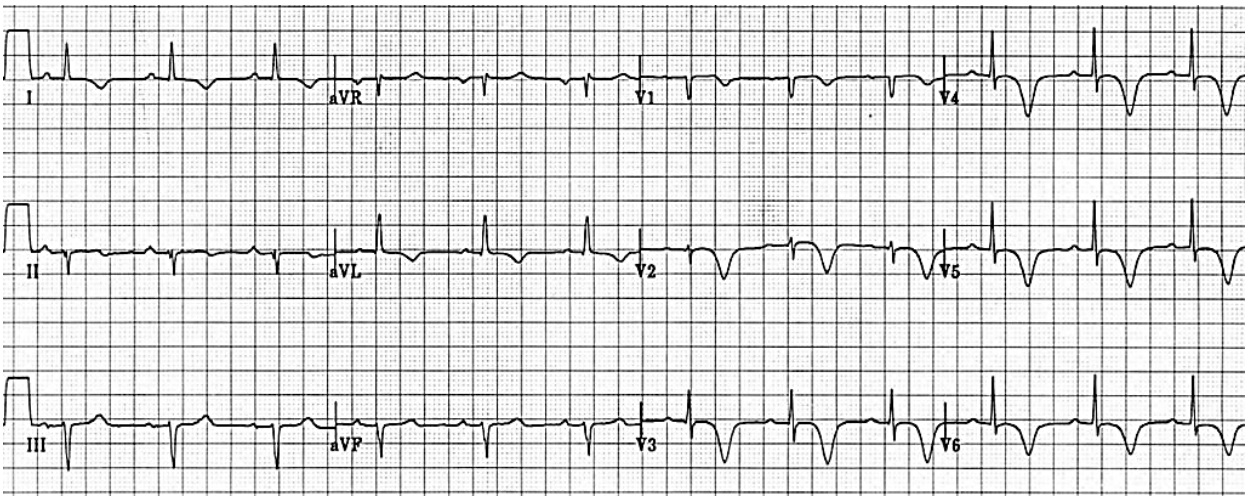


Figure 10. 12-lead ECG of STEMI with deep, symmetrical T-wave inversions indicative of Wellen's syndrome[30]



Another indication of acute cardiac ischemia on the ECG is flat or down-sloping ST-depression ≥ 0.5 mm in 2 contiguous leads (Figure 11). If the ST-depressions are seen in leads V_1-V_3 then the next step should be to obtain a 12-lead ECG with posterior leads. This is accomplished by removing leads V_4-V_6 from the patient and placing them at the inferior margin of the left scapula, as shown in Figure 12. If these leads (now called V_7-V_9) show ST-elevation this will be consistent with a posterior STEMI (Figure 13).

Figure 11. 12-lead ECG showing ST-depressions in V_2-V_3 which is suspicious for posterior STEMI[31]

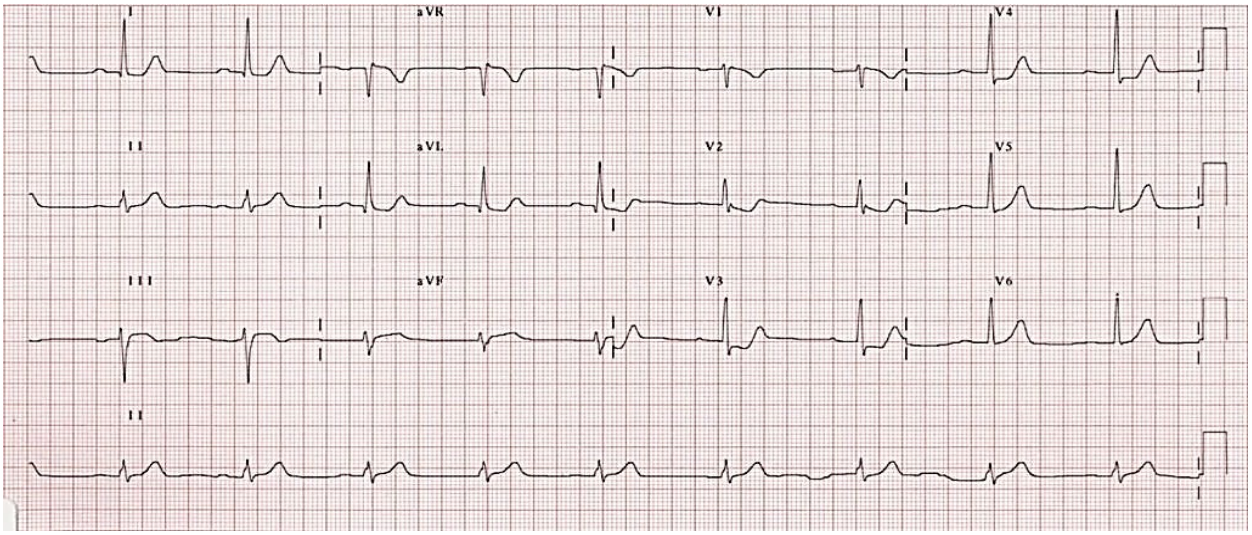


Figure 12. Posterior ECG lead placement[32]

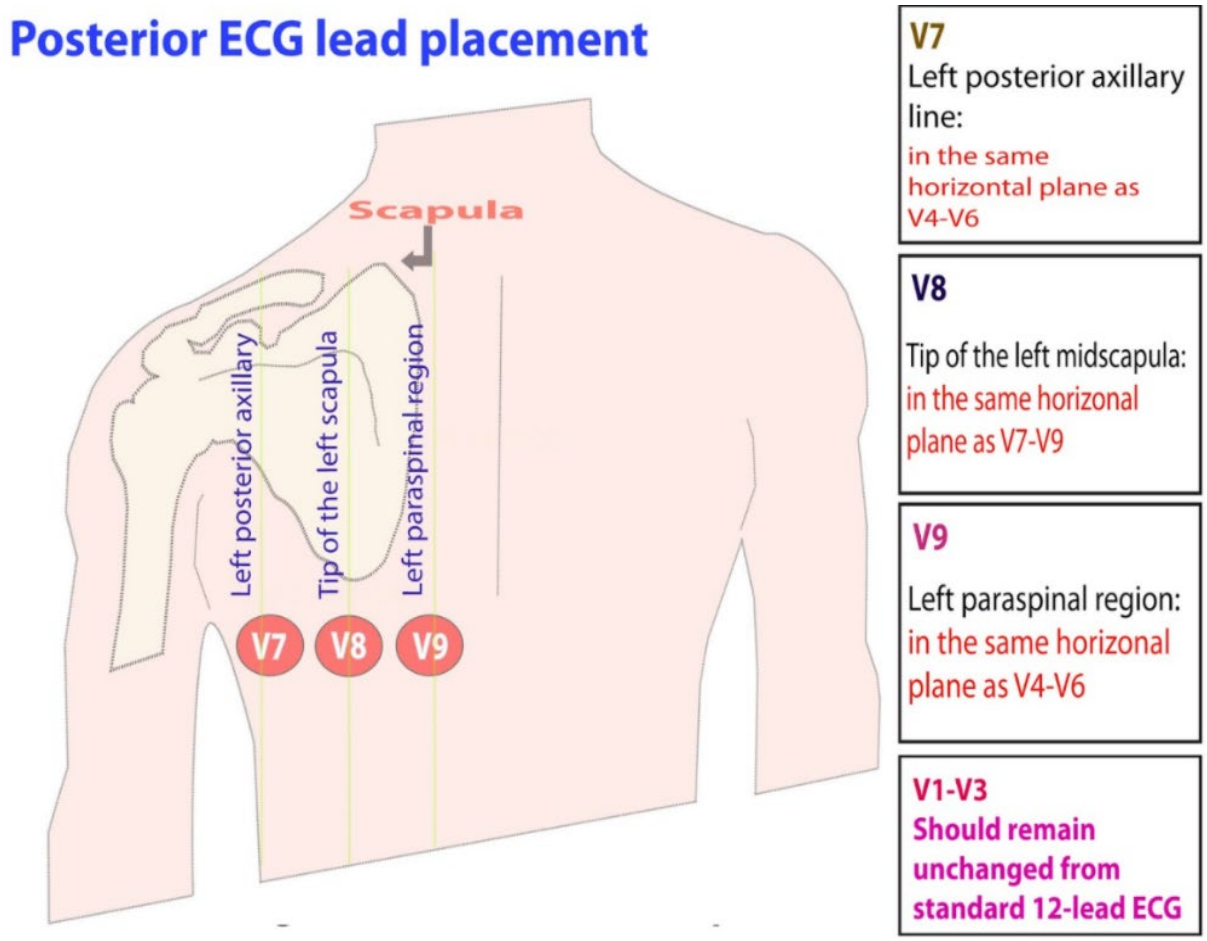
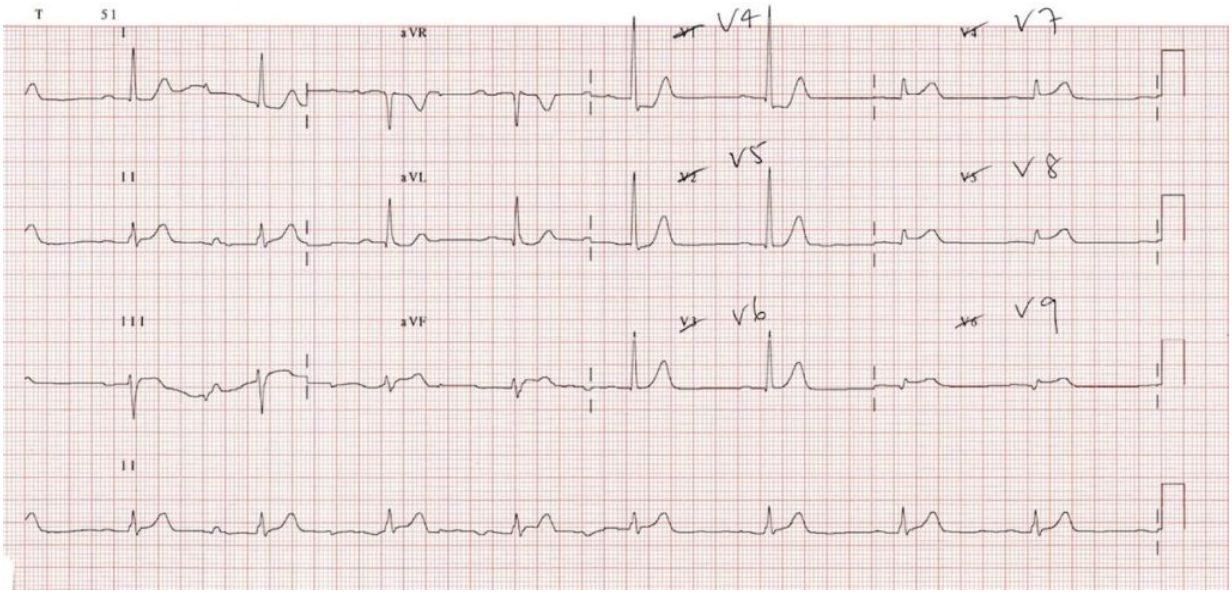
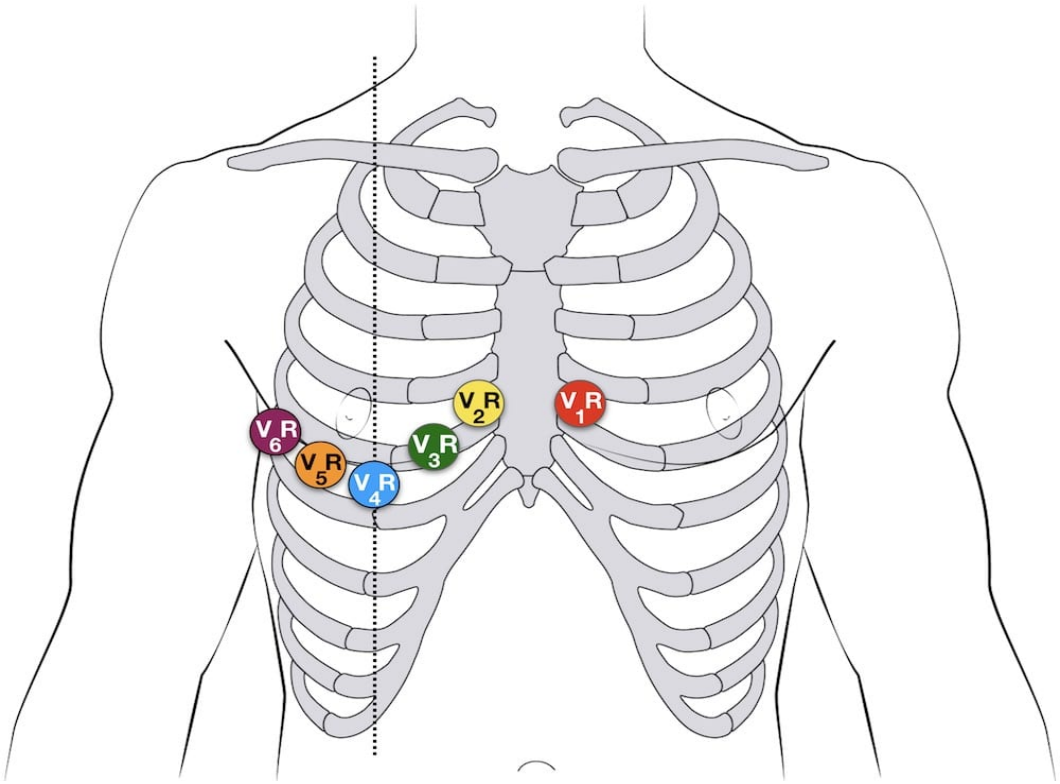


Figure 13. 12-lead ECG STEMI Showing posterior STEMI. Notice ST-elevations in posterior leads V_7-V_9[31]



Similarly, if the initial ECG shows evidence of an inferior STEMI (ST-elevation in lead II, lead III, and aVF with reciprocal changes), then consider placing leads V_1-V_6 on the patient as a complete mirror reflection (as shown in Figure 14). If these leads (now called V_1R-V_6R) show ST-elevation this will be consistent with right ventricular infarction.

Figure 14. Right-sided ECG lead placement[33]



If the initial ECG is non-diagnostic (i.e. only one lead meeting ST-elevation criteria but the second contiguous lead ST-elevation is less than 0.1 mV) but the suspicion for ACS is present, then serial ECGs (every 15 minutes for the first hour) should be performed to ensure STEMI is not missed.[17,19]

In contrast to STEMI, UA/NSTEMI can present with a myriad of ECG findings. These may include normal ECGs, nonspecific T-wave inversions, ST-depression or transient ST-elevation.

TREATMENT

MEDICAL THERAPY

Initial medical therapy for ACS includes rapid delivery of antiplatelet agents, anticoagulants and revascularization therapy to include fibrinolytic therapy (if no contraindications exist and PCI is not immediately available). The below sections include lists of common available medications in each class.

Antiplatelet Therapy

Aspirin should be administered immediately to all patients with suspected ACS if no contraindications exist. Once the diagnosis of ACS has been made, a platelet adenosine diphosphate receptor (P2Y).[12] An inhibitor (e.g., clopidogrel, ticagrelor) should then also be administered if no contraindications exist. Initial dosing for all agents is listed below.

1. **Aspirin 162** – 325mg PO x 1 (chewable is preferred, non-enteric coated preferred if chewable not available) then 81mg PO daily.

AND

2. **Clopidogrel (Plavix)**
    a. Age ≤ 75 years old: initial loading dose of 300mg PO x 1 then 75mg PO daily.
    b. Age > 75 years old: initial dose of 75mg PO x 1 then 75mg PO daily.

OR

3. **Ticagrelor (Brilinta)** 180mg PO x1 then 90mg twice daily.
    It is important to note that ticagrelor can cause dyspnea which is thought to be benign, though sometimes concerning to the patient/clinician.

Anticoagulant Therapy

Systemic anticoagulation should be administered as soon as the diagnosis of ACS is made if no contraindications exist. Options for anticoagulants include unfractionated heparin and low molecular weight heparin (LMWH) (i.e. enoxaparin) which exert their anticoagulant properties by indirectly inhibiting thrombin. Another option is factor Xa inhibitor (i.e. fondaparinux). Heparin is administered as a bolus followed by a drip, and its effects are monitored by the activated partial thromboplastin time (aPTT). Currently, typical CCATT that transports patients out of theater do not have the ability to monitor aPTT for heparin drips. Thus, enoxaparin is preferred over a heparin drip when transport is required, given its ease of use. If enoxaparin or another LMWH is not available, then consideration of fondaparinux should be utilized if available. If heparin is the only agent available, then it should be given. Dosing consideration for transport would be decided based on an aPTT immediately prior to the

flight. For patients that have a known or suspected history of heparin induced thrombocytopenia, fondaparinux should be used. Anticoagulation should continue for at least 48 hours or until revascularization.

The following are the dosing for each agent listed below:

1. **Enoxaparin (Lovenox) – *preferred agent***
    a. **STEMI dosing:**
        - Age < 75 years: single 30mg IV bolus **plus** 1mg/kg SubQ (maximum 100mg for the first 2 doses only). then 1mg/kg SubQ every 12 hours
        - Age ≥ 75 years: 0.75 mg/kg SubQ every 12 hours
    b. **NSTEMI dosing**: 1mg/kg SubQ every 12 hours.
    c. Consult pharmacy to adjust this dose if there is any renal injury (rise in creatinine).

OR

2. **Heparin Sulfate**
    a. **STEMI dosing**: 60 U/kg initial bolus IV (4,000 U maximum), then 12 U/kg/hr (maximum 1,000 u/hr) infusion.
    Maintain aPTT 1.5–2.0 times control (50-70s).
    b. **NSTEMI dosing**: 60 U/kg initial bolus IV (5,000 U maximum), then 12 U/kg/hr (maximum 1,000 U/hr) infusion.
    Maintain aPTT 1.5–2.0 times control (50-70s).

OR

3. **Fondaparinux (Arixtra)**
    a. **STEMI dosing**: 2.5 mg IV x1 then 2.5 mg SubQ daily.
    b. **NSTEMI dosing**: 2.5 mg SubQ daily.

REPERFUSION THERAPY

Prompt reperfusion is the mainstay of STEMI care. Whenever possible, a patient presenting with STEMI should be transferred emergently to the nearest medical facility that can perform urgent PCI as it is considered the optimal treatment. This should be achieved within 2 hours of patient arrival.[13] In the event that door-to-balloon time cannot be achieved within 2 hours, fibrinolytics should be given instead. In the deployed setting, PCI, may be prohibitively far away, and thus rapid delivery of fibrinolytic therapy is the method of choice for revascularization. Fibrinolytic therapy should be administered within 30 minutes of STEMI diagnosis. There are absolute and relative contraindications to administration of fibrinolytics (listed in Appendix A). This list should be meticulously reviewed step by step prior to administration of these agents. The two most common agents available are listed below with their dosing. Fibrinolytics should be offered to patients with symptom duration of 12 hours or less. It is recommended that expert consultation with the theater cardiology consultant be used prior to administering fibrinolytics in the 12 to 24 hour period.

***Note**: Dual antiplatelet therapy, systemic anticoagulation, beta blocker and high dose statins should still be given even when fibrinolytics are given.*

1. **Tenecteplase (TNKase) – *preferred agent***
   a. < 60 kg: 30mg IV bolus x 1
   b. 60-69 kg: 35mg IV bolus x 1
   c. 70-79 kg: 40mg IV bolus x 1
   d. 80-89 kg: 45mg IV bolus x 1
   e. ≥ 90 kg: 50mg IV bolus x 1

OR

2. **Alteplase (Activase)**
   a. ≤ 67 kg: 15mg IV bolus over 1-2 minutes, then infusion of 0.75 mg/kg (maximum 50mg) over 30 minutes, then infusion of 0.5 mg/kg (maximum 35mg) over 60 minutes.
   b. 67 KG: 15mg IV bolus over 1-2 minutes, then infusion of 50mg over 30 minutes, then infusion of 35mg over 60 minutes (maximum total dose 100mg).

NOTE: In contrast to STEMI, fibrinolytics are contraindicated in UA/NSTEMI patients.

STATIN THERAPY

Regardless of the patient's blood lipid levels, high dose Statin therapy should also be administered during the initial presentation (within the first 2 hours) of ACS. The following are the two therapy options to administer:

1. **Atorvastatin (Lipitor)** 80mg PO x 1 then 80mg nightly.

OR

2. **Rosuvastatin (Crestor)** 20-40mg PO x1 then 20-40mg PO daily.

SUPPORTIVE THERAPY

Supplemental Oxygen

Administer ring oxygen in patients with ACS is recommended only when oxygen saturation levels are < 90%, to avoid potential harm from oxygen free radicals and further hyperoxia coronary vasoconstriction.

Analgesia

Decreasing the pain response can block sympathetic activity and relieve anxiety, which decreases myocardial oxygen consumption. The routine use of morphine for pain control is not recommended since this has been shown to increase the risk of mortality in ACS.[34] Nitroglycerin a vasodilator, opens blood vessels to improve blood flow; treating angina symptoms, such as chest pain or pressure that

happens when there is not enough blood flowing to the heart. Nitroglycerin dilates coronary arteries and relaxes vascular smooth muscles, resulting in decreased preload/afterload and decreased myocardial oxygen demand.

Nitroglycerin

1. Initial dose of 0.3 – 0.4 mg sublingually every 5 minutes for 3 doses; afterwards an intravenous infusion may be considered.
2. If a nitroglycerin IV infusion is administered, start at 5-10 mcg/ minute (min) and titrate as needed to relieve anginal symptoms in increments of 5 mcg/min every 5-10 minutes up to 20 mcg/min; if angina persists at a dose of 20 mcg/min then increase the dosage by 10-20 mcg/min every 3-5 minutes to a maximum dose of 400 mcg/min.
3. Nitroglycerin is contraindicated in patients who have a systolic blood pressure < 100 mmHg, who have used phosphodiesterase inhibitors in the last 24 hours, or who have evidence of inferior STEMI on ECG.

Beta Blockers

Beta blockers block sympathetic stimulation and decrease the heart rate. They have been shown to decrease early development of lethal ventricular dysrhythmias as well as improve long term left ventricular remodeling. Although the American Heart Association and American College of Cardiology recommend that beta blockers be initiated in the first 24 hours of NSTEMI, there have been studies that show early administration of beta blockers in the ED was associated with higher rates of shock or death than later administration.[35] Initiate this medication after the patient has been hospitalized in either the Role 2 or Role 3. Beta blockers should be withheld in patients with systolic blood pressure < 100 mmHg, heart rate < 60 beats per minute, evidence of pulmonary edema, second or third degree heart block, severe reactive airway disease, or elevated risk of cardiogenic shock.

1. **Metoprolol tartrate** (immediate release) – 12.5 mg PO every 6 – 12 hours.

OR

2. **Metoprolol succinate** (extended release) – 25-50 mg PO once daily.

OR

3. **Atenolo**l – 50-100 mg PO every 12 to 24 hours.

ACE Inhibitors and ARBs

Angiotension converting enzyme (ACE) inhibitors that are initiated within 24 hours to 16 days after an acute MI show improved patient survival as well as improved left ventricular ejection fraction. ACE inhibitors are contraindicated in systolic blood pressure < 100 mmHg, history of bilateral renal artery stenosis, hyperkalemia, or prior worsening renal function with ACE inhibitors. It is reasonable to administer angiotensin receptor blockers (ARBs) in patients who cannot tolerate ACE inhibitors.

1. **Captopril** - initial dose of 6.25 mg PO, which is increased at 6 to 8 hour intervals to a maximum of 50 mg PO three times daily as long as the systolic blood pressure is above 90 to 100 mmHg.

OR

2. **Enalapril** - initial dose 2.5 mg PO daily increased up to 20 mg PO twice daily.

OR

3. **Lisinopril** - initial dose 2.5 mg PO daily increased to a maximum of 10 mg PO daily.

OR

4. **Losartan** - 25 to 50 mg PO once daily depending on initial blood pressure.

Other Therapies

If indicated continue to utilize Proton pump inhibitors. Nonsteroidal anti-inflammatory drugs (NSAIDs) on the other hand should be avoided in these patients due to their increased risk of adverse cardiovascular events and the increased risk of bleeding when combined with the other mainstay treatments of ACS such as antiplatelet and anticoagulant therapy. Routine use of blood transfusion in the setting of ACS is associated with increased mortality.[13,36,37] It is recommended to avoid transfusion unless the hemoglobin level is < 8 g/DL .

COMPLICATIONS TO THERAPY

BLEEDING COMPLICATIONS

Bleeding complications are likely to be rare but they may be encountered and should be closely watched. Neurological checks should occur every 15 minutes for the first hour and then 30 minutes for the next 6 hours to monitor for intracranial bleeding. Normotension should be strived for while SBP > 160 mmHg should be avoided. Once life-threatening bleeding is identified, stop all antiplatelet agents, anticoagulants, and fibrinolytics. Specific reversal therapies unique to life-threatening bleeding due to thrombolysis are listed below.[38] In the case of suspected intracranial bleeding, obtain an emergent CT head and consult neurosurgery if an intracranial bleed is actually present.

1. Administer Cryoprecipitate - 10 units IV.
2. Administer tranexamic acid (TXA) – 10-15 mg/kg IV.
3. Stop all antiplatelet, anticoagulant, and fibrinolytics.

COMPLICATIONS ASSOCIATED WITH MI

There are both early and late complications associated with MI. Early complications will be the focus here as these are the ones likely to be seen in the deployed setting during the initial hours to days of treatment.

CARDIOGENIC SHOCK

Cardiogenic shock is best defined as SBP < 90 mmHg for 30 minutes or SBP > 90 mmHg on vasopressors with evidence of end organ damage from a cardiovascular origin. The patient may have evidence of pulmonary edema (worsening dyspnea, crackles, worsening chest x-ray) or evidence of peripheral hypoperfusion (weak pulses, cold extremities). Listed below are the vasopressor/inotropic agents that should be utilized in this situation.[39]

1. **Norepinephrine** (preferred first line agent) - 5 – 20 mcg/min IV. Start at 5 mcg/min and increase by 2-5 mcg/min q15 minutes.
2. **Dopamine** - 0.5 to 20 mcg/kg/minute IV (lower doses preferred).

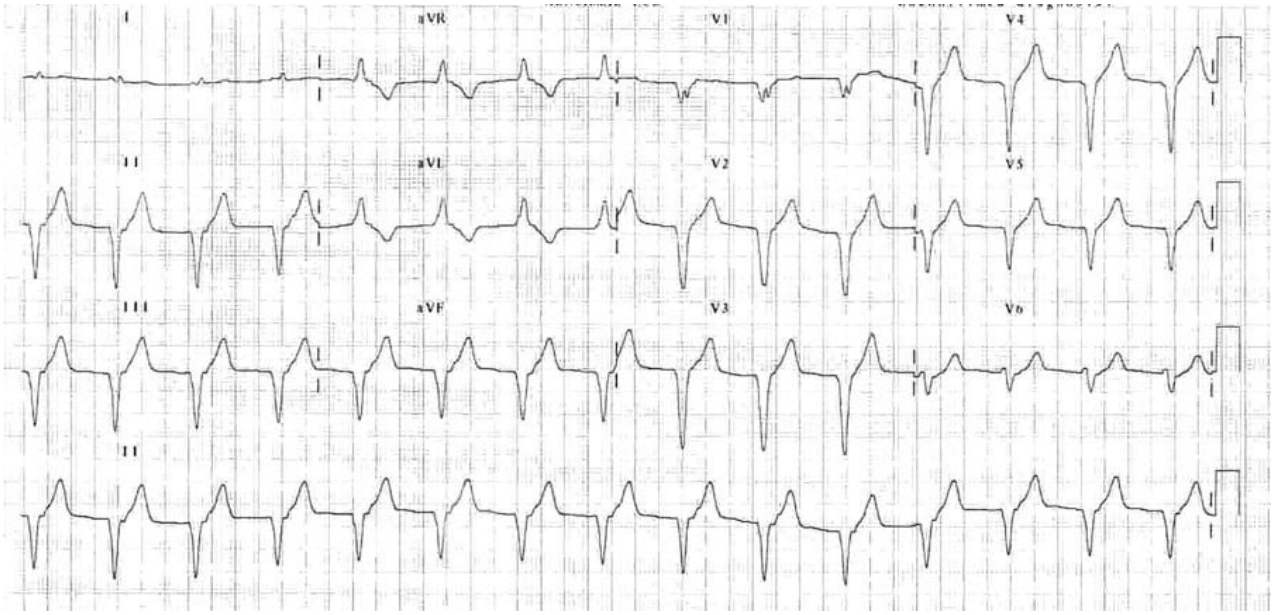
MECHANICAL COMPLICATIONS

Mechanical complications primarily manifest as either acute chordal rupture of the mitral valve, ventricular septal rupture, or left ventricular free wall rupture. These typically occur within the first 24 hours but can present within a week as well. The use of fibrinolytics is also a risk factor for an acute ventricular septal rupture and left ventricular free wall rupture. Medical therapy is only temporary with a high mortality rate for those unable to undergo surgical correction. Echocardiography is the modality of choice for rapid diagnosis. This can show pericardial effusion when associated with left ventricular free wall rupture.

ARRHYTHMIAS

There are multiple different tachy- and brady-dysrhythmias that can occur with acute MI. Most can be addressed using ACLS protocols. The most common rhythm following reperfusion is accelerated idioventricular rhythm (Figure 14). This manifests as a wide complex QRS rhythm (as you would see with ventricular tachycardia) with heart rates in the 50 – 110 bpm; it is benign and requires no intervention.[40] Patients are typically asymptomatic with normal vital signs otherwise, and it should resolve spontaneously.

Figure 15. 12-lead ECG STEMI Accelerated idioventricular rhythm after STEMI reperfusion[40]



CONSIDERATIONS DURING MECHANICAL VENTILATION

MEDICATIONS

If the patient with ACS begins to develop shock and is unable to protect his/her airway, then you might have to perform endotracheal intubation (Refer to Airway Management CPG). The following considerations should be made regarding induction and post-intubation sedation medications:[41]

1. **Ketamine** should be avoided in patients with acute MI or heart failure due to increased myocardial oxygen consumption and negative inotropic effects.
2. **Etomidate** is recommended for induction of anesthesia given its neutral effects on hemodynamics.
3. **Propofol** should be used with caution in induction, given its ability to cause acute hypotension. It is generally safe for maintenance of anesthesia, however, caution should be used in patients with severe cardiac disease as propofol has also been shown to exacerbate cardiac dysfunction through its negative inotropic effects.
4. **Versed/Fentanyl** is acceptable for induction/maintenance of anesthesia.

VENTILATOR SETTINGS

High intrathoracic pressures affect the right and left ventricles differently. High intrathoracic pressures impair right ventricular filling, yet reduces left ventricular afterload. In a patient's post-MI that require mechanical ventilation, minimization of intrathoracic pressures for a patient with inferior/right ventricular infarction is essential to maintain cardiac output. PEEP and tidal volume should be minimized as much as possible.

CONSIDERATIONS DURING EVACUATION

The care of ACS patients at deployed locations is limited by the fact that cardiac care is provided in austere locations without rapid access to higher levels of care. While medical therapies can stabilize patients for short periods of time, any complications that arise do not have the benefit of being treated urgent/emergently in a cardiac catheterization laboratory. A plan for urgent MEDEVAC/aeromedical evacuation to a PCI capable center should be initiated as soon as they are diagnosed with ACS and initiated medical therapy.

TELEMEDICINE/CARDIOLOGY CONSULTANT

The first cardiology consultant within the evacuation chain should be consulted immediately for patients diagnosed with a STEMI or UA/NSTEMI at deployed locations. Diagnosis and treatment of ACS should be guided by expert consultation, to include electronic transmission of ECGs (by whatever means available).

MEDEVAC

When possible, all patients diagnosed with or with high suspicion of ACS at Role 1 or 2 facilities should be moved to a Role 3 facility (preferably a facility with a cardiologist) as soon as possible.

HOST NATION

At times host nations (HNs) have facilities that meet U.S. standards for medical care. If relationships with these facilities have been established and have been utilized for emergent care of U.S. personnel, then their use should be considered for emergent PCI in the following situations:

1. **STEMI** – PCI at a HN facility is preferred over fibrinolysis since this is the primary treatment for STEMI. If the facility or total transport time exceeds 120 minutes, then administer fibrinolysis (with cardiology teleconsultation) and continue coordinating transport to the HN PCI lab.
2. **UA/NSTEMI** – refractory angina, ischemic ECG changes, hemodynamic instability (with or without cardiogenic shock) or refractory arrhythmias after receiving medical therapy.

AEROMEDICAL EVACUATION

Aeromedical evacuation of all ACS patients should adhere to the following principles when possible due to the evolving nature of the disease and limited ability to fully handle the complications that come with this disease process.

1. All aeromedical evacuation of ACS patients should be evacuated by CCATT or equivalent level of care.
2. Missions should be requested as URGENT unless otherwise designated with cardiology consultation.
3. All in flight treatment should follow the recommendations put forth in the previous sections of the CPG.

IN-FLIGHT CONSIDERATIONS

1. Maintain O_2 saturations > 90%.
2. Red blood cell transfusion should be avoided unless the hemoglobin level is < 8 g/dl in patients without active ischemia.[13,36]
3. In the event of an inflight STEMI (new vs. reinfarction), then thrombolytics should be given unless a contraindication exists (see Appendix A).
4. If the patient becomes unstable in-flight, consideration of diversion to the closest location with PCI capabilities should be strongly considered. An unstable patient is one who has ongoing ischemia and the presence of either:
    a. Hypotension/shock primarily due to suspected cardiovascular causes.
    b. Recurrent ventricular arrhythmias that are unable to be controlled with ACLS medications (e.g., beta blockers, amiodarone).

PERFORMANCE IMPROVEMENT (PI) MONITORING

POPULATION OF INTEREST

1. All patients presenting with a STEMI receive dual antiplatelet therapy, systemic anticoagulation and fibrinolytic therapy (unless contraindicated) within 30 minutes of arrival to a facility with these therapies available.
2. In facilities without fibrinolytics, dual antiplatelet therapy and systemic anticoagulation is administered before transfer to a higher level of care.

INTENT (EXPECTED OUTCOMES)

1. All patients with concern for STEMI or NSTEMI will have a documented consultation/teleconsultation with a cardiologist.
2. All patients diagnosed with STEMI are transferred to a PCI center.

PERFORMANCE/ADHERENCE MEASURES

1. Dual antiplatelet therapy and systemic anticoagulation are given within 30 minutes of arrival at a facility with these therapies available.
2. Fibrinolytic therapy is given within 30 minutes of confirmation of a STEMI in facilities that have these therapies, or door to PCI center within 120 minutes with facilities that can arrange for a transfer to a PCI center.
3. Consultation/teleconsultation with a cardiologist is documented for all patients with concern for STEMI or NSTEMI.

DATA SOURCE

- Patient Record
- Department of Defense Trauma Registry
- Theater Medical Data Store

SYSTEM REPORTING & FREQUENCY

The above constitutes the minimum criteria for PI monitoring of this CPG. System reporting frequency will be performed annually; additional PI monitoring and system reporting may be performed as needed.

The system review and data analysis will be performed by the JTS Chief, Program Manager and PI Branch.

RESPONSIBILITIES

It is the Chief of Trauma or equivalent's responsibility to ensure familiarity, appropriate compliance and PI monitoring at the local level with this CPG.

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APPENDIX A: FIBRINOLYTICS ABSOLUTE AND RELATIVE CONTRAINDICATIONS IN STEMI

The following table provides the absolute and relative contraindications health factors in regards to administering fibrinolytics in STEMI.

Table A-1. Absolute and Relative Contraindications

Absolute	Relative
Any prior intracranial hemorrhage	History of chronic, severe, poorly controlled hypertension
Known structural cerebral vascular lesion (e.g., arteriovenous malformation)	Significant hypertension on presentation (SBP >180 mm Hg or DBP >110 mm Hg)
Known malignant intracranial neoplasm (primary or metastatic)	History of prior ischemic stroke >3 months
Ischemic stroke within 3 months (EXCEPT acute ischemic stroke within 4.5 hours)	Dementia
Suspected aortic dissection	Known intracranial pathology not covered in absolute contraindications
Active bleeding or bleeding diathesis (excluding menses)	Traumatic or prolonged (>10 minutes) CPR
Significant closed-head or facial trauma within 3 months	Major surgery (< 3 weeks)
Intracranial or intraspinal surgery within 2 months	Recent (within 2 to 4 weeks) internal bleeding
Severe uncontrolled hypertension (unresponsive to emergency therapy)	Noncompressible vascular punctures
For streptokinase, prior treatment within the previous 6 months	Pregnancy
	Active peptic ulcer
	Oral anticoagulant therapy

Source: O'Gara PT, Kushner FG, Ascheim DD, et al. The American College of Cardiology Foundation/American Heart Association guideline for the management of ST-elevation myocardial infarction: executive summary. Circulation. 2013;127:529–555

APPENDIX B: MAJOR ADVERSE CARDIAC EVENT (MACE) HEART SCORE

This section provides the predictive HEART score for a 6-week risk of MACE.

MACEs are defined as the following:

- Acute myocardial infarction (MI)
- Percutaneous coronary intervention (PCI)
- Coronary artery bypass graft (CABG)
- Coronary angiography revealing procedurally correctable stenosis managed conservatively
- Death due to any cause

Table B-1. MACE Heart Score

History	Highly suspicious	2
	Moderately suspicious	1
	Slightly or non-suspicious	0
ECG	Significant ST-depression	2
	Nonspecific repolarization disturbance	1
	Normal	0
Age	≥ 65 years	2
	> 45 - < 65 years	1
	≤ 45 years	0
Risk Factors*	≥ 3 risk factors, *or* history of atherosclerotic disturbance	2
	1 to 2 risk factors	1
	No risk factors known	0
Troponin	≥ 3x normal limit	2
	> 1 - < 3x normal limit	1
	≤ normal limit	0

The following are MACE risk factors:

- Hypertension
- Hypercholesterolemia
- Diabetes mellitus
- Obesity (body max index > 30 kg/m2)
- Smoking (current, or smoking cessation < 1 month)

- Positive family history for coronary artery disease
- Atherosclerotic disease
- Prior MI, PCI, CABG, cerebrovascular accident/transient ischaemic attack or peripheral artery disease

The following are the score levels for MACE:

- 0 – 3: low risk; 1.7% found to have MACE
- 4 – 6: moderate risk; 16.6% found to have MACE
- 7 – 10: high risk; 50.1% found to have MACE

Sources:

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APPENDIX C: ACS ROLES

The following figures provide the ACS roles for care.

Figure C-1. ACS Role 1

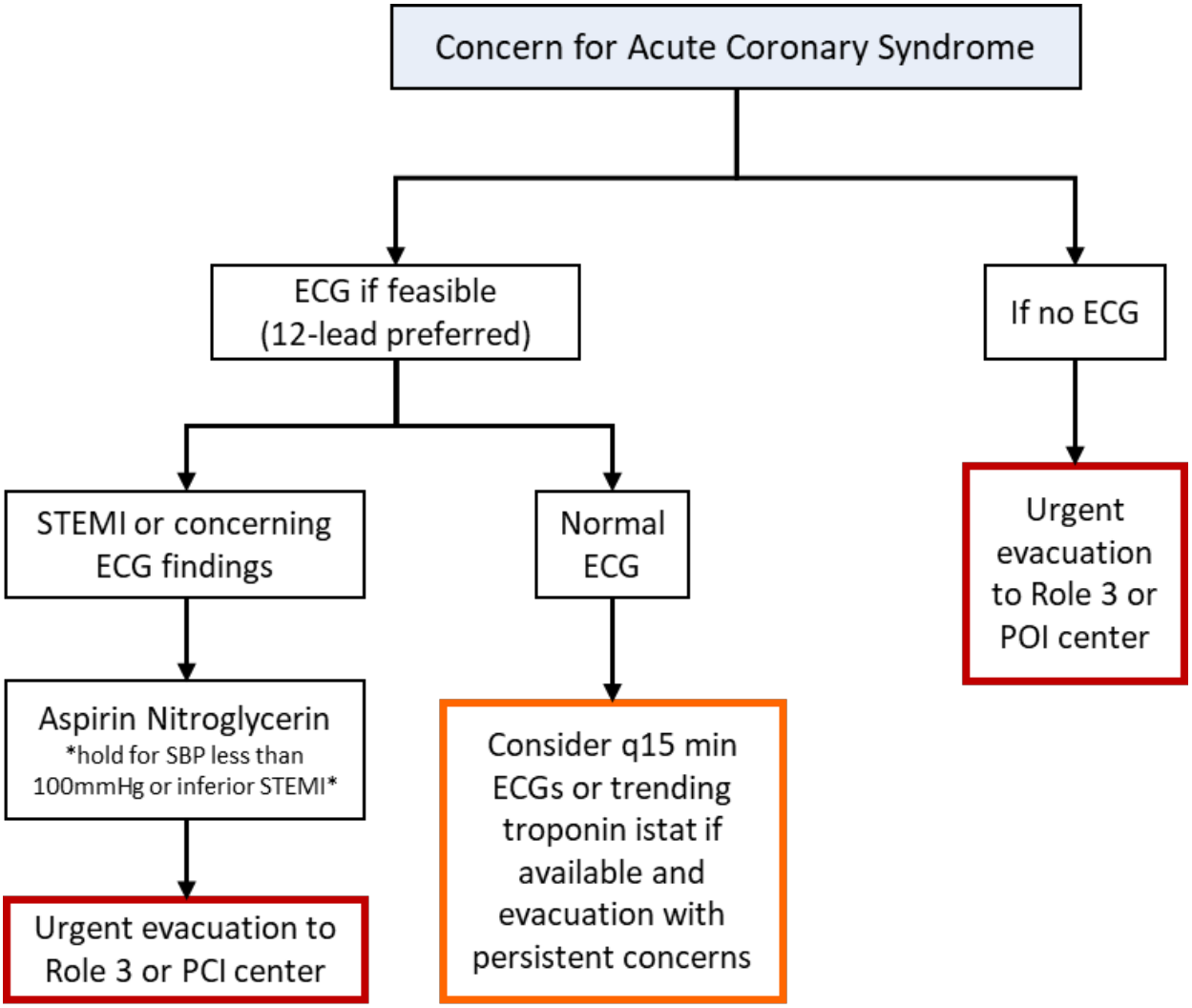


Figure C-2. ACS Role 2

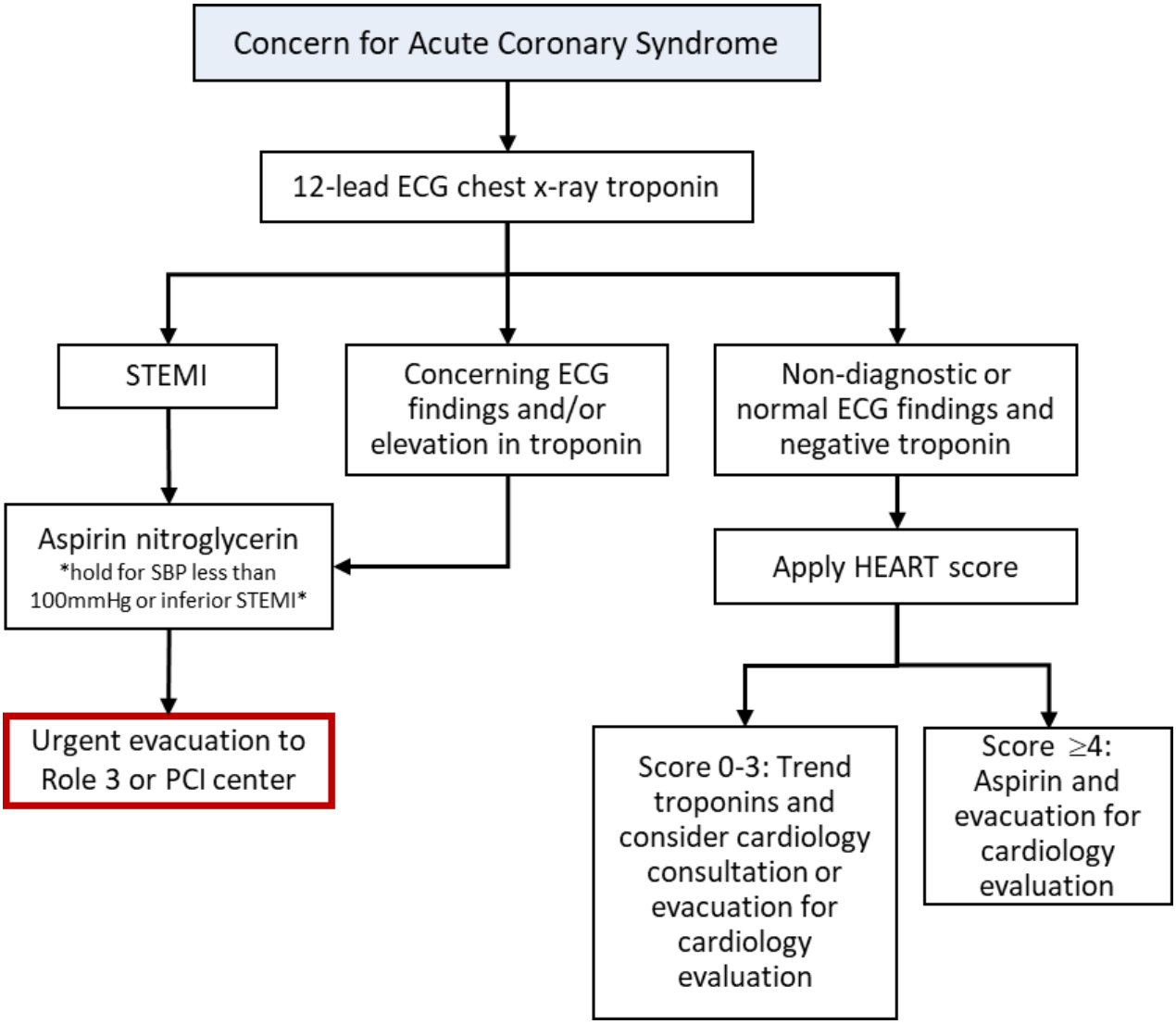
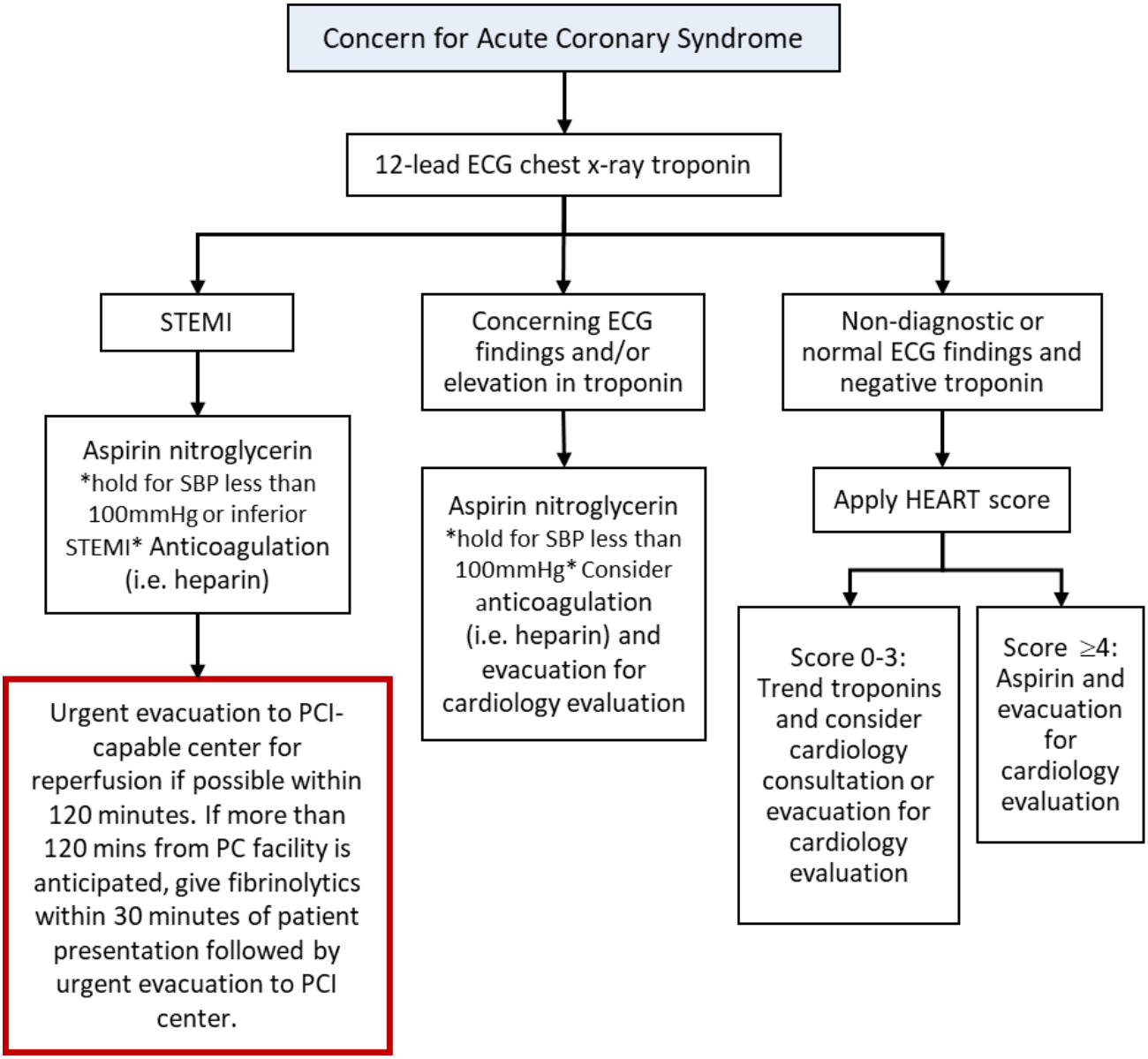


Figure C-3. ACS Role 3



APPENDIX D: ADDITIONAL INFORMATION REGARDING OFF-LABEL USES IN CPGS

PURPOSE

The purpose of this Appendix is to ensure an understanding of DoD policy and practice regarding inclusion in CPGs of "off-label" uses of U.S. Food and Drug Administration (FDA)–approved products. This applies to off-label uses with patients who are armed forces members.

BACKGROUND

Unapproved (i.e. "off-label") uses of FDA-approved products are extremely common in American medicine and are usually not subject to any special regulations. However, under Federal law, in some circumstances, unapproved uses of approved drugs are subject to FDA regulations governing "investigational new drugs." These circumstances include such uses as part of clinical trials, and in the military context, command required, unapproved uses. Some command requested unapproved uses may also be subject to special regulations.

ADDITIONAL INFORMATION REGARDING OFF-LABEL USES IN CPGS

The inclusion in CPGs of off-label uses is not a clinical trial, nor is it a command request or requirement. Further, it does not imply that the Military Health System requires that use by DoD health care practitioners or considers it to be the "standard of care." Rather, the inclusion in CPGs of off-label uses is to inform the clinical judgment of the responsible health care practitioner by providing information regarding potential risks and benefits of treatment alternatives. The decision is for the clinical judgment of the responsible health care practitioner within the practitioner-patient relationship.

ADDITIONAL PROCEDURES

Balanced Discussion

Consistent with this purpose, CPG discussions of off-label uses specifically state that they are uses not approved by the FDA. Further, such discussions are balanced in the presentation of appropriate clinical study data, including any such data that suggest caution in the use of the product and specifically including any FDA-issued warnings.

Quality Assurance Monitoring

With respect to such off-label uses, DoD procedure is to maintain a regular system of quality assurance monitoring of outcomes and known potential adverse events. For this reason, the importance of accurate clinical records is underscored.

Information to Patients

Good clinical practice includes the provision of appropriate information to patients. Each CPG discussing an unusual off-label use will address the issue of information to patients. When practicable, consideration will be given to including in an appendix an appropriate information sheet for distribution to patients, whether before or after use of the product. Information to patients should address in plain language: a) that the use is not approved by the FDA; b) the reasons why a DoD health care practitioner would decide to use the product for this purpose; and c) the potential risks associated with such use.