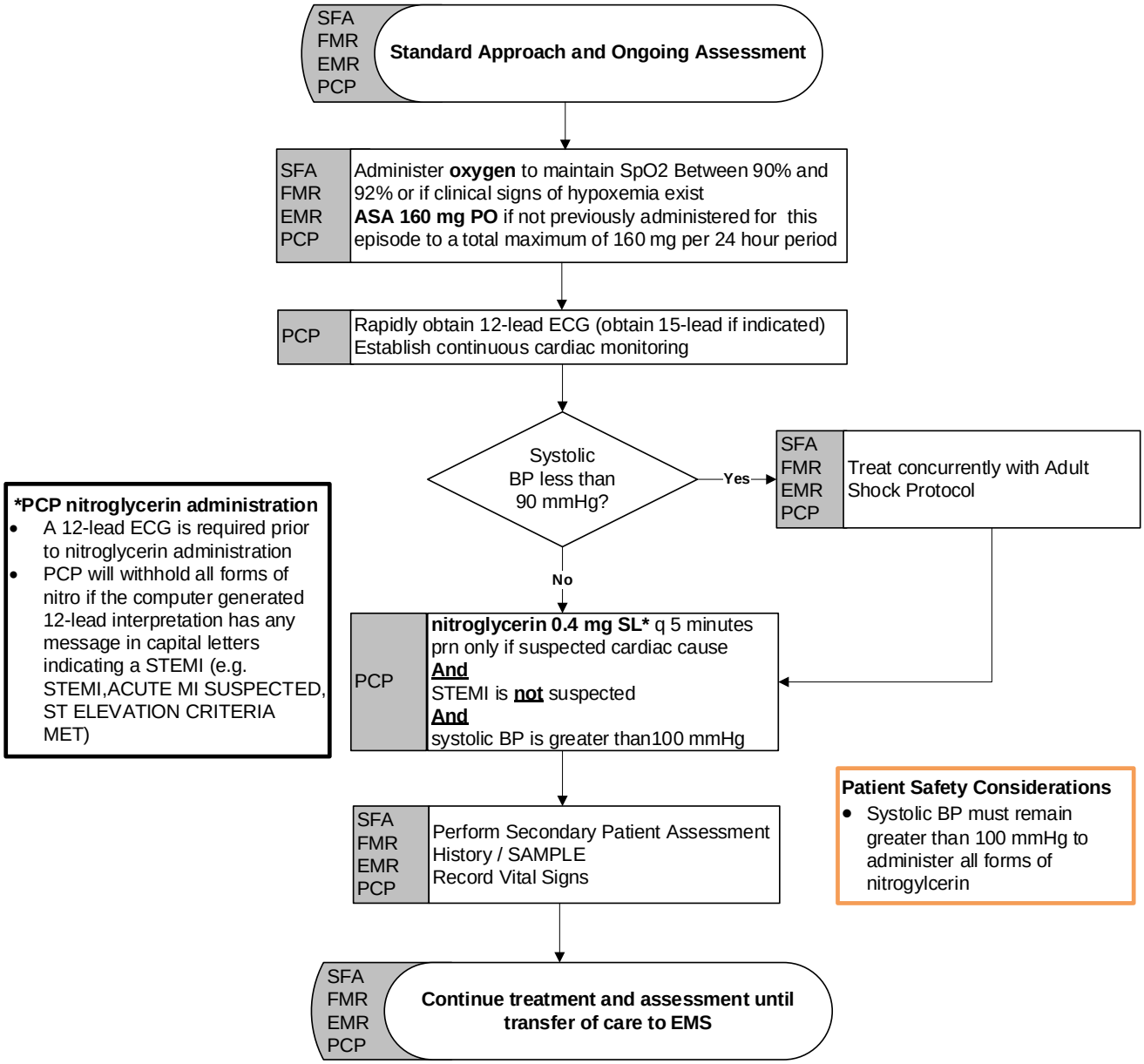
Adult Chest Pain



Chest Pain (Acute Coronary Syndrome Suspected)

Although there are many potential causes of chest pain, all patients with acute onset chest pain should be approached as having a myocardial infarction. Some of the life-threatening causes of chest pain are:

- Unstable angina
- Acute myocardial infarction
- Aortic dissection
- Pulmonary embolus
- Spontaneous pneumothorax
- Esophageal rupture

Symptom Recognition

Prompt recognition that an **acute myocardial infarction (AMI)** or heart attack is occurring is critical, since most deaths associated with AMI are due to electrical instability and occur suddenly, often before arrival at the hospital. It is important to aggressively treat patients with symptoms of myocardial infarction since ventricular fibrillation is more likely to occur during the first hour after onset of symptoms than at any other time.

About 20% of AMI's are not accompanied by chest pain, especially in elderly persons, females, and/or diabetic patients. When pain is present, it generally has a retro-sternal component; and it may radiate to the neck, shoulders, lower jaw, back, or down the inside of either or both arms. This pain is typically described as a heavy or squeezing sensation. It may be mild to severe, but it tends to increase in severity over a period of minutes. In some patients, high epigastric discomfort may be a symptom of AMI and is often dismissed by the patient as indigestion.

The pain of AMI generally lasts longer than 15 - 30 min. The pain of AMI typically builds to its maximum, whereas pain from aortic dissection or pulmonary embolus is usually most severe, rapidly after onset.

Angina Pectoris (Chest Pain)

Is induced by exertion, usually lasts 5 to 15 min and is relieved by rest or by nitroglycerin.

Any angina pain that lasts longer than 15 min, is not relieved by the patient's own nitroglycerin, or is accompanied by diaphoresis, dyspnea, nausea, or vomiting, suggests an AMI.

The following are at highest risk for sudden death:

- Patients with a new onset of chest pain either at rest or with ordinary or usual activity.
- Patients who experience a sudden change in a previously stable pattern of angina pain, such as an increase in frequency or severity, or occurrences at rest for the first time.

- Patients who are experiencing chest pain and have known coronary heart disease
- Chest pain or discomfort that is unrelieved by rest and/or nitroglycerin.

Questions to Consider

- Did the pain/discomfort begin suddenly?
- What was the patient doing when the pain/discomfort began?
- Has the patient ever had the pain/discomfort before?
- Has the pain/discomfort become better or worse?
- Has the patient ever had a heart attack?

Oxygen Administration

Supplemental oxygen helps reduce both the magnitude and extent of damage in patients with AMI. Patients should be allowed to remain in the position of greatest comfort and ease of breathing. If supplemental oxygen is administered, the flow rate should be titrated to maintain a SpO2 value of no greater than 92%.

Oxygen delivery should start with nasal cannula at 2 - 4 LPM and progress to higher concentrations as required.

- Excessive oxygen administration has been shown to be potentially harmful to the ACS and STEMI patient populations
- Supplemental oxygen administration should be avoided in ACS and STEMI patients with a SpO2 value of greater than or equal to 90% unless clinical signs or symptoms of hypoxemia are observed

Acetylsalicylic Acid (ASA)

ASA administration is the single most important medication to limit both morbidity and mortality in AMI, equal to the benefit of thrombolysis

Refer to the History Taking Guidelines

Myocardial Infarction	
Signs	**Symptoms**
Tachypnea Dysrhythmias Cyanosis Diaphoresis Vomiting Agitation Cardiac arrest Cardiogenic shock	Chest and/or abdominal pain Palpitations Shortness of breath Sweating Nausea Light-headedness / Pre-syncope Confusion Weakness Anxiety / fear Feeling of impending doom
Differential Diagnosis Limited differential diagnoses for chest pain include:	
Angina Pulmonary embolism Dissecting thoracic aortic aneurysm Gastrointestinal cause (e.g. esophageal spasm) Hyperventilation Musculoskeletal cause (e.g. chest wall pain)	Myocardial infarction Pericarditis Pneumonia Pneumothorax Pleurisy Dermatologic cause (e.g. shingles)

Life Threatening and Serious Causes of Non-ACS Chest Pain		
Life Threatening	**Potential Life-threatening**	**Less Serious**
Aortic dissection Acute pericardial effusion and tamponade Acute pulmonary embolism Tension pneumothorax	Peptic ulcer, perforated Esophageal rupture Acute pneumonia Aortic stenosis (chest pain, syncope, exertional dyspnea) Acute cholecystitis, cholelithiasis, ruptured gall bladder Acute pancreatitis	Gastroesophageal reflux disease (GERD) Esophagitis, gastritis Hiatal hernia Musculoskeletal chest pain Costochondritis

Patient Safety Considerations
25% of AMI patients present with reproducible chest wall tenderness.

Additional Notes for Primary Care Paramedics

Acute Coronary Syndromes (ACS) Suspected

Patients suffering from ACS may experience a broad range of syndromes that result from varying degrees of coronary artery occlusion. These syndromes include:

Unstable Angina (UA)

- Chest pain due to angina is caused by a transient increase in myocardial oxygen demand beyond the coronary supply; this may be due to atherosclerotic plaque that has narrowed the coronary artery or abnormal coronary arterial spasm
- Thrombus is platelet-rich at this stage and treatment with aspirin is most effective
- Occurs at rest and may not respond to treatment in contrast to stable angina which occurs during physical or emotional stress and responds quickly to rest and medications

Non-ST-Segment Elevation Myocardial Infarction (NSTEMI)

- A partial occlusion of a coronary artery, or a combination of coronary arterial spasm with a thrombus, can cause infarction or cell death to some of the heart tissue supplied by that artery
- ECG interpretation shows ST-segment depression or dynamic T-wave inversion; transient ST-segment elevation of less than 20 minutes is also included in this syndrome

ST-Segment Elevation Myocardial Infarction (STEMI)

- Death of heart tissue due to a complete occlusion of a coronary artery preventing blood flow to an area of the heart
- Characterized by the following ECG changes:

Anterior MI	In men, new ST elevation at the J point greater than or equal to 2mm (0.2mV) in leads V2-V3 In WOMEN, new ST elevation at the J point of at least 1.5mm (0.15mV) in leads V2-V3
Inferior, Septal, Lateral MI	In men OR women, new ST elevation at the J point of greater than or equal to 1mm in other contiguous chest leads or limb leads
Posterior MI	In men OR women, new ST Depression at the J point of greater than or equal to 1mm in leads V1-V2 and ST elevation greater than 1mm in a posterior lead V7-V9

1. Common presentations of acute myocardial infarction (AMI) includes:
   - Prolonged, severe substernal chest pressure, radiating down the left arm and up into the jaw
   - Shortness of breath
   - Diaphoresis
   - Nausea and vomiting

2. The following patient groups are more likely to present with atypical signs and symptoms:
   - Elderly (greater than 75 years old)
   - Women
   - Diabetics
   - Young adults who abuse cocaine or other sympathomimetic drugs (e.g. speed)

3. ECG changes are not always present in ACS; treat the patient regardless of how the ECG is interpreted

4. Sympathomimetic Induced Chest Pain
   - Chest pain that is a result of sympathomimetic use (e.g. cocaine, amphetamines) is to be treated according to the Adult Sympathomimetic Overdose Protocol

5. Anxiety and agitation can occur as a result of ischemic chest pain. Optimal treatment of this condition is effectively managing the ACS.

Differential Diagnosis

Limited differential diagnosis for chest pain included:

- Angina
- Pulmonary embolism
- Dissecting thoracic aortic aneurysm
- Gastrointestinal cause (e.g. esophageal spasm)
- Hyperventilation
- Musculoskeletal cause (e.g. chest wall pain)
- Myocardial infarction
- Pericarditis
- Pneumonia
- Pneumothorax
- Pleurisy
- Dermatologic cause (e.g. shingles)

	Process	Location	Quality	Severity	Timing	Factors
Pericarditis	Irritation of parietal pleura adjacent to pericardium	Precordial, may radiate to tip of shoulder and to neck	Sharp, knifelike	Often severe	Persistent	Worsen: Breathing changing positions, coughing, lying down, swallowing Relieve: Better sitting forward
Dissecting Aortic Aneurysm	A splitting within the layers of the aortic wall	Anterior chest, radiating to neck, back or abdomen	Ripping, tearing	Very severe	Abrupt onset	Worsen: Hypertension
Pleural Pain	Inflammation of the parietal pleura (pleurisy, pneumonia)	Chest wall overlying the process	Sharp, knifelike	Often severe	Persistent	Worsen: Breathing, coughing, movements of the trunk make it worse Relieve: Lying on involved side
Reflex Esophagitis	Inflammation of the esophageal mucosa by reflux of gastric acid	Retrosternal, may radiate to back	Burning, may be squeezing	Mild to severe	Variable	Worsen: large meal, bending over, lying down Relieve: antacids, belching
Chest Wall Pain	Variable, often unclear	Often below left breast or along costal cartilages	Stabbing, sticking, or dull, aching	Variable	Fleeting to hours or days	Worsen: movement of chest, trunk, arms
Anxiety	Unclear	Precordial, below left breast, or across anterior chest	Stabbing, sticking, or dull, aching	Variable	Fleeting hours to days	Worsen: may follow effort, emotional stress

Interventions

Nitroglycerin Administration

Nitroglycerin does not alter morbidity and mortality in Acute Coronary Syndrome (ACS), it is for symptom relief. Administration of nitroglycerin **WILL NOT** be performed if a 12 lead ECG is unavailable. This is due to the increased risk of creating profound hypotension in patients experiencing a Right Ventricular Infarct (RVI).

PCPs will withhold all forms of nitroglycerin if not equipped to obtain a 12-lead ECG or the computer generated 12-lead interpretation has any message in capital letters indicating a STEMI (e.g. STEMI, ACUTE MI, SUSPECTED, ST ELEVATION CRITERIA MET)

Establish IV access prior to administration of nitroglycerin. If possible, start IV in patient's left forearm.

Vascular Access

- Vascular access is mandatory in patients receiving nitrate therapy
- Obtain IV access in the left arm
- Do not use the right arm unless absolutely necessary as some physicians use the right radial artery for interventional procedures
- Tape the catheter / administration set or saline lock in such a manner that the hospital can easily change administration sets as necessary

ECG Monitoring / 12-lead / 15-lead

Lead Placement

- Leads can be placed anywhere on limb (not trunk) but avoid bone
- Chest lead placement must be consistent
- In female patients place chest leads under the breast tissue
- Never use nipples as a reference point for locating electrodes on men or women as locations vary

Be sure to prep the skin first by a brisk rub with a 4 x 4 – this will result in an ECG with less artifact

Lie patient flat if possible, to obtain the ECG

- V1 - 4th intercostal space - Right of sternum
- V2 - 4th intercostal space - Left of Sternum
- V3 - Midway between V2 and V4
- V4 - 5th intercostal space - Left midclavicular line
- V5 - At the same level as V4 - Left anterior axillary line
- V6 - At the same level as V4 - Left midaxillary line
- V4R - 5th intercostal space - Right midclavicular line

12 / 15-lead Placement and Acquisition

- Acquire standard 12-lead ECG
- Remove leads V4, V5, V6
- V4 to V4R on corresponding R anterior chest
- V5 to V8 at the same level as V4 posterior left back midscapular line
- V6 to V9 at the same level as V4 posterior left back, paraspinal line (next to spine) next to V8
- Run another 12-lead. Relabel V4 to V4R, V5 to V8 and V6 to V9

ST-segment elevation in the following leads may be indicative of injury in the corresponding area:

II, III, and aVF	Inferior
I, V5, V6, and aVL	Lateral
V1 and V2	Septal
V3 and V4	Anterior
V8, and V9	Posterior
V3R – V6R	Right Ventricular

Measure ST-segment changes at the J point (i.e. the end of the QRS complex and start of the ST-segment)

Certain conditions can produce ST-segment elevation which can mimic an AMI (i.e. intracranial hemorrhage, pericarditis, hypothermia, benign early repolarization); however, these are diagnoses of exclusion

15-lead ECG is only required when there is inferior ST elevation (leads II, III, or aVF) or ST depression in V1-V4 (Is this referenced in the algorithm? Why would we ask PCPs to do this?)
Repeat serial ECGs every 15 minutes looking for evolution of ECG changes

Leads with ECG Change	Injury/Infarct – Related Artery	Area of Damage	Associated Complications
VI-V2	LCA - LAD Septum branch	Septum, His Bundle, bundle branches (Septal MI)	Infranodal block and BBBs
V3-V4	LCA – LAD diagonal branch	Anterior wall LV (Anterior MI)	LV dysfunction, CHF, BBBs, complete heart block, PVC's
V5-V6, I aVL	LCA - circumflex branch	High lateral wall LV (Lateral MI)	LV dysfunction, AV nodal block in some
II, III, aVF	RCA- posterior descending branch (PDA)	Inferior wall LV, posterior wall LV (Inferior MI)	Hypotension, bradycardia
V4R (II, III, aVF)	RCA — proximal branches	RV, Inferior wall LV, posterior wall LV (RVI)	Hypotension, sensitivity to nitro, morphine, AV nodal blocks, atrial fib/flutter, PAC's
V8-9 elevation VI-V4 depression	RCA — PDA LCA - circumflex	Posterior LV	LV dysfunction
ECG Changes (Note: other conditions may cause these to occur)			
ST depression — ischemia		T wave inversion — ischemia	
ST elevation — acute injury		Pathological Q waves — infarction	

Infarct Imposters

Left Ventricular Hypertrophy (LVH)

LVH may cause ST elevation that mimics ACS. LVH results in an enlarged left ventricle which increases the amplitude but not the width of the QRS.

Pericarditis

Normally one of the first clues of pericarditis on the ECG is that ST elevation does not follow a normal pattern of coronary artery distribution. The ST elevation in pericarditis is due to the pericardium being inflamed.

Bundle Branch Block

Left bundle branch block (LBBB) may cause ST elevation due to abnormal depolarization