Assessment of Chest Pain
Cardiac-Specific Assessment
Angina and MI

Objectives
• Identify the cardiac and non-cardiac causes for chest pain
• Identify the general physiology involved in the development of cardiovascular disorders
• Outline a cardiac-specific assessment flow for a patient with chest pain

Objectives
• Differentiate between the presentations of stable angina, unstable angina and acute MI
• Explain the pathophysiology involved in the development of acute coronary syndromes

Objectives
• Discuss the “window of opportunity” in terms of reperfusion therapy for an MI
• Discuss the hemodynamic complications of an MI
• Identify general treatment strategies to minimize or prevent complications related to an acute MI

Exercise

Causes of Cardiovascular Disorders
• Atherosclerosis
  - Stable angina
  - Aortic aneurysm
• Issues with blood clotting
  - Unstable angina
  - Myocardial infarction
• Problems with cardiac output
  - Congestive heart failure
• Issues with electrical cell potentials
  - Dysrhythmias
The Cardiovascular Assessment

Additions to the Basic Assessment Flow

• General Impression and Initial Assessment
  - Focus on respiratory or circulatory compromise
  - Scene clues for chronic conditions

The Focused History

• This is the most helpful stage for determining potential causes for the patient’s presentation
  - Respiratory v. cardiac causes
  - AMI or CHF?
  - Pneumonia or CHF?
  - Dysrhythmias or chronic cardiac problem?
• Allows for better decisions regarding drug therapy

The Focused History

• This should include a combined assessment of chest pain and dyspnea
• Past medical history and medications will provide valuable information on baseline cardiac or respiratory diseases
  - Use of the OPQRST can compare chronic to acute symptoms and changes
• Associated symptoms may help narrow the cause for the patient’s presentation today

The Focused History In Detail

• Onset
  - When the pain started and the potential origin of the pain
  - Relate this to the patient’s pertinent past medical history
• Provocation/Palliation
  - Is the pain caused or increased with exertion?
  - Does the pain subside with rest?

The Focused History In Detail

• Quality
  - Pleuritic pain v. cardiac organ pain
  - “Chest tightness” from bronchoconstriction
• Severity
  - Used as a baseline
    • Compare to previous episodes
    • To determine if treatments are working
• Timing
  - Duration of pain
  - Continuous or intermittent pain
The Focused History In Detail

• “SHOPS”
  - Street drugs
    • Especially stimulants
  - Herbal remedies
  - Over-the-counter medications
  - Prescription medications
    • Compliance and changes in medications
  - Sexual enhancement drugs
    • Cialis, Levitra, Viagra

The Focused History in Detail

• The dyspnea assessment
  - Continuous v. intermittent
  - Exertional v. non-exertional
  - Orthopnea or paroxysmal nocturnal dyspnea (PND)
  - Cough
    • Wet v. dry
    • Productive
    • Frothy or bloody

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The Focused History In Detail

• Associated Symptoms
  - Nausea and/or vomiting
  - Fatigue
  - Limitations in daily activities
  - Palpitations
  - Edema
  - Headache
  - Syncope
  - Traumatic cause?

The Focused History in Detail

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Pertinent Past Medical History

• Coronary artery disease (CAD)
• Angina
• Previous MI
• Hypertension
• Congestive heart failure (CHF)
• Valve disease
• Vascular disease
• Aortic aneurysm
• Pulmonary disease
• Diabetes
• Previous cardiac surgery
• Congenital heart problems

The Physical Assessment

• Focus areas:
  - Neck
    • Signs of trauma or JVD
  - Chest
    • Inspection, auscultation
    • Palpation if history suggests trauma
  - Abdomen
    • Inspection and palpation
  - Extremities
    • Temperature changes, color changes, presence of edema

Chest Assessment
Extremities – Pedal Edema

Additional Components of a Cardiovascular Assessment
- EKG monitoring and interpretation
- $\text{SaO}_2$
- Vital signs
  - Orthostatic changes

Application of Cardiovascular Assessment

Questions:
- Stable or critical?
- What elements would you like to include in your scene assessment and general impression?

Initial Assessment Findings
- Breathing is shallow but unlabored
- Patient able to speak 5 word-sentences but is a bit winded when finished speaking.
- Radial pulse is present but irregular
  - Rate is between 45-65
- Skin color is pale but no diaphoresis noted
- Patient is conscious and alert

More on the Patient Scenario
- Her chief complaint is dyspnea
  - What elements of the dyspnea assessment should you include?
  - What other questions should you include in your assessment?
- She also has had general weakness for the past 5 days.
  - Is this important to pursue with the history?
- Oh yeah – she has some chest pain, too.

Dyspnea Assessment
- Continuous or intermittent?
  - “It’s with me almost always.”
- Does the dyspnea change with exertion?
  - “Yes, it does.”
- Do you get more short of breath when you lie flat or sleep?
  - “Yes. I’m sleeping on 2 pillows at night now.”
- Cough assessment
  - “I have had a minor cough, but it is non-productive.”
Chest Pain Assessment

• Onset
  - “I feel it when I take a deep breath.”
  - “I feel it now, though.”
• Provocation
  - “It is more noticeable when I take a deep breath.”
• Quality
  - “It is a nuisance, mainly. It feels tight to me.”

Radiation of Pain
- “None.”

Severity of Pain
- “It’s there.” Pain is rated at a 3-4.

Time
- The dyspnea has worsened over the past 5 days.
- “The chest pain did come with the dyspnea and comes mainly with deep breaths.”

Discuss the causes of dyspnea and chest pain and which causes may be the potential culprits for Gertrude’s symptoms.

Time for small groups

Associated Symptoms

• No nausea or vomiting
• Fatigue is more noticeable, but she thinks it is from feeling so weak.
• She doesn’t do as many daily activities because she feels so weak.
• Denies palpitations, but she normally has an irregular pulse.

• The swelling in her feet has been increasing.
• No headache.
• No syncope.
• No recent chest trauma.

Small groups: discuss how this information may change or confirm your possibilities.

More Assessment

• “SHOPS”
  - She takes Aleve, Lasix and digoxin
    • She has been increasing her dose of Lasix from 1x/day to 3x/day (40mg to 120mg) in order to improve her edema and dyspnea.
    - She takes no other medications or drugs
• Past Medical History
  - CHF
  - Diverticulitis
  - Gall bladder surgery 1 year ago
Vital Signs

- Vital signs
  - SaO2 is 93% on room air
  - Increases to 97% with oxygen at 4 LPM
  - Respiratory rate drops only slightly with oxygen
  - BP is 164/78
  - Pulse is irregular at a rate of 45-65
  - EKG reveals atrial fibrillation

Physical Assessment Findings

- Moderate JVD noticed as the patient is seated upright
- No barrel chest noted
- No scars noted on chest
- Lung sounds reveal crackles in all fields
- No pulsations or masses noted to the abdomen
- Pitting edema noted to the patient’s feet and ankles

Treatment Options for the Patient

- Treatment options:
  - Chest pain
  - Dyspnea
  - Rate problem
  - CHF
- Which of the above elements will you treat? What components of your assessment led you to choose this?

The Findings in the Emergency Department

- The X-ray reveals an enlarged heart with fluid in the lungs.
  - The patient has a documented history of left ventricular enlargement
- Her digoxin level was normal
- ED Diagnosis: “Exacerbation of CHF.”

Scope of Practice Changes for Chest Pain Management

- Nitroglycerin and morphine sulfate
  - The “for pain” limitation is now removed
  - This provides a greater potential to treat pulmonary edema as a result of CHF
- Furosemide
  - Potent diuretic used to treat pulmonary edema as a result of CHF
• Aspirin
  – No change in dosing
• Atropine
  – No changes
• Antidysrhythmic Drugs
  – Addition of amiodarone
  – No change to lidocaine
  – Emphasis on identifying the underlying cause for the dysrhythmia and determining whether the antidysrhythmic is appropriate to give

Implications of the New Scope
• Most of the new drugs carry significant side effects
  – The EMT-I must increase their knowledge on the specific actions of drugs
  – The EMT-I must possess a greater understanding of cardiac diseases
    • It is just important to know when not to give the drug as it is to know when to give it

Chest Pain Profiles: Angina and MI

Chest Pain Profile In Angina
• What are the OPQRST findings for stable angina?
  – Onset with exertion
  – Relief with rest and/or medication
  – Substernal pain, with or without radiation
  – Crushing, pressure or squeezing-type pain
  – Predictable profile for the patient

From Angina to MI: Step 1 - Angina
A. Atherosclerosis builds in the coronary artery
  • Progressive build-up of plaque which will narrow the diameter of the artery
  • Reduced oxygen delivery during increased workload of the heart
  • Rest will decrease workload and resultant demand for oxygen
  • Medication (nitroglycerin) widens vessel in order to deliver more oxygen to the heart

Atherosclerosis and Angina Connections
How will this vessel handle a demand for more blood flow by the heart during a period of increased workload?
Chest pain profile in unstable angina

• How is the pain profile different in unstable angina?
  - Onset may occur either at rest or with exercise
  - Pain quality may be similar to stable angina
  - It may be worse, however
  - Pain does not go away as easily
  - More nitroglycerin required to reduce or eliminate the pain

From Angina to MI: Step 2 – Unstable Angina

• Plaque hardens and creates turbulence in the vessel
• Plaque “catches” and tears
• Initiates an injury response

The injury response: a quick review of blood clotting

Steps of the Injury Response

• Vascular spasm
• Platelet plug

Steps of the Injury Response

• Coagulation
  - Fibrin net forms around the injury
    • Contains the platelets already at the site
    • Traps more platelets and the blood clot builds

• Clotting and Repair
  - Fibrinolysis
  - Eventual breakdown of the clot by plasmin
Development of Unstable Angina

- Tear in plaque stimulates the injury response
- Blood clot further narrows artery
- Reduction of blood flow begins to impact the heart under normal workload conditions
- Myocardium is very ischemic

From Angina to MI

- Similar progression as that of unstable angina
- Differences
  - More significant occlusion or complete occlusion
  - Heart muscle is injured and/or dying

AMI Example

Occlusion Timelines

- Ischemia occurs immediately after occlusion
- Injury will continue as time progresses
  - 30 minutes after occlusion, myocardial cells in the injured area begin to die
  - 2 hours after occlusion, half of the cells in the area of injury are now dead
  - 4-6 hours after occlusion, 90% of the initially injured area is dead

Reperfusion Therapy

- Goal is to reverse injury and ischemia in the myocardium
  - “Time is muscle”
- Reperfusion therapy options
  - Fibrinolytics
  - Angioplasty
- “Window of opportunity”
  - Timeline for maximum benefit from reperfusion therapy
  - 6 hours from the onset of symptoms to the therapy
Fibrinolytics

- Ateplace, Retaplace, t-PA
- Accelerates the disintegration of the clot
  - Encourages plasmin to “eat” the fibrin mesh
- Non-specific target sites
  - It will dissolve all clots that are present anywhere in the body
  - It will not allow clots to develop while the drug is in the patient’s system
  - IV therapy, blood draws
- Need for screening criteria for this therapy

Angioplasty

- Insertion of a flexible catheter into the coronary artery
- Inflation of the attached balloon against the artery wall
- Plaque is crushed and the vessel opens

Patient Presentation in AMI

- Similar symptoms to unstable angina
  - Pain is dull, crushing, pressure
  - Onset with rest
  - Unrelieved by nitroglycerin, rest and oxygen
    - Pain may not even dissipate with morphine
  - Radiation of pain

- Other symptoms
  - Nausea, vomiting
  - Epigastric pain
  - Frequent belching
- Jaw pain, bilateral shoulder pain
- Shortness of breath
  - May be the only symptom
  - Suspect MI with this symptom for females and patients with diabetes

Complications of an MI: Reduced Cardiac Output

- Reduction of heart rate
  - Dysrhythmias
  - “Protection” by parasympathetic NS
- Reduction of stroke volume
  - Physical damage to myocardium
    - Inability to push out blood in severe MIs will cause a back-up of fluid into the lungs
  - Dysrhythmias change the flow of ventricular contraction
- Poor cardiac output may cause systemic hypoperfusion
Signs of Cardiovascular Compromise

• Restless, agitation or anxiety
• Changes in skin signs
  – Pallor, grey skin, cool extremities, and/or diaphoresis
• Patient sense of impending doom
• New-onset ventricular dysrhythmias

Management Strategies for MI

• Rapid recognition!
  – Signs and symptoms
  • Differentiation between angina and MI
  • Changes to baseline angina/CHF profiles

• Prevent or minimize complications
  – Watch for new-onset dysrhythmias
  – Emphasize pain control
  • Oxygen: flow based upon overall perfusion
  • Aspirin
    – Prevent further clot development by making the platelets less sticky and reducing their ability to stimulate further clotting
  • Nitroglycerin and morphine
    – Vasodilation to provide more blood flow
    – Reduce preload to decrease workload on the myocardium

The End