Acute Coronary Syndrome (ACS): From Door to Discharge

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Cardiovascular Nursing Education Associates / Key Choice

Acute Coronary Syndrome (ACS)

- No ST Elevation
- Non STEMI
- Unstable Angina
- ST Elevation
- STEMI
Hospitalizations in the U.S. due to Acute Coronary Syndromes

1,190,000 Hospital Discharges with primary or secondary diagnosis of ACS

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>NRMI-4*:</th>
<th>AHA Get with the Guidelines</th>
<th>GRACE** Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>UA/NSTEMI</td>
<td>71%</td>
<td>69%</td>
<td>62%</td>
</tr>
<tr>
<td>STEMI</td>
<td>29%</td>
<td>31%</td>
<td>38%</td>
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</tbody>
</table>

Acute Coronary Syndrome refers to any rupture of plaque or thrombotic event that leads to symptomatic ischemia or infarction.

Ruptured Plaque

STEMI

NonSTEMI / Unstable Angina
Pathophysiology of ACS

- Deposit of lipids, calcium, fibrin, and other cellular substances within the lining of the arteries.
- Initiates a progressive inflammatory response in an effort to heal the endothelium.
- End result of inflammatory process: the production of a fibrous atherosclerotic plaque.
- Plaque can progress to cause coronary stenosis
- Plaque can also rupture prior to causing significant stenosis

Plaque

- **Stable plaque of stable angina**
  - Thick fibrous caps separate the lipid core from the endothelium
  - Less complicated than vulnerable plaques
  - Tend to have smooth outlines

- **Vulnerable plaque of ACS**
  - Thin caps
  - Edge of the fibrous cap is a particularly vulnerable area and is commonly the location of ruptured plaque

- **Limitations of stress testing**
Non-Modifiable Risk Factors

- Previous history
- Family history
  - 1st degree relative (parents, siblings)
  - Men < 55; Women < 65
- Age
- Gender
- Socioeconomic Factors and Ethnicity

9 easily measured and potentially modifiable risk factors account for over 90% of the risk of an initial acute MI

- Smoking
- Hypertension
- Dyslipidemia
- Diabetes
- Obesity
- Metabolic Syndrome
- Inactivity
- Alcohol

Mortality Rate Age > 40 years:
1 year: F- 23%, M- 18%
5 year: F- 43%, M – 33%
## ACS Symptoms

<table>
<thead>
<tr>
<th>Classic Symptoms</th>
<th>Symptom Variations</th>
</tr>
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<tbody>
<tr>
<td>Stable angina</td>
<td>MI</td>
</tr>
<tr>
<td>Unstable angina</td>
<td>Women</td>
</tr>
<tr>
<td></td>
<td>Elderly</td>
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<tr>
<td></td>
<td>Diabetics</td>
</tr>
</tbody>
</table>

### Classic Symptoms
- Stable angina
- Unstable angina
- MI

### Symptom Variations
- Women
- Elderly
- Diabetics

### Characteristics of Angina

**Sensation of pressure, tightness, heaviness, burning, or squeezing.**

- Usually not described as a sharp or stabbing pain (women)
- Should not worsen with changes in position or respiration.

**Location behind the sternum and in the upper back, shoulder, arm, jaw, or epigastric area.**

- Not usually located in the middle to lower abdomen and does usually not radiate to the lower extremities.

**Associated symptoms (or stand alone symptoms) of dyspnea, nausea, palpitations, or diaphoresis.**

**Duration typically defined in minutes.**

- Not typically defined in seconds or hours.

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**CAUTION WHEN ASKING THE PATIENT ABOUT “PAIN”!**
Acute MI Symptoms

- Symptoms occur spontaneously and are not relieved by rest or nitroglycerin
- Chest pressure or discomfort may be accompanied by nausea, vomiting, or diaphoresis
- Patient may have hemodynamic instability or cardiac arrest from ventricular fibrillation

STEMI

- 29-38% of ACS patients
- Complete occlusion of a vessel by a thrombus
- Fibrin stable clot (red clot)
- Classified more specifically by the portion of the left ventricle suffering injury.
- Mortality is greatest within the first 24 to 48 hours of symptom onset

TREATMENT FOCUS = REPERFUSION
NSTE – ACS

Nationally under treated according to evidence based practice guidelines (Crusade Registry)
Pathophysiology often involves a platelet plug or white clot
Less stable clot
Opportunity for spontaneous reperfusion
Differentiated from unstable angina by troponin levels

TREATMENT FOCUS = ANTIPLATELET THERAPY

Supply and Demand Mismatch

- Increase myocardial oxygen demand:
  - Hyperthermia
  - Hypertension
  - Tachycardia
  - Conditions producing over stimulation of the sympathetic nervous system (cocaine use, hyperthyroidism)
- Decrease myocardial oxygen delivery:
  - Anemia
  - Pulmonary disease.
- Increase myocardial oxygen demand and decrease myocardial oxygen supply:
  - Aortic stenosis
  - Hypertrophic cardiomyopathy

Type 2 MI

Elderly are at risk for secondary coronary events related to supply and demand imbalance.
The Coronary Arteries

- **Left Main Coronary Artery (LM)**
  - Left Anterior Descending Artery (LAD)
    - Diagonal Branch
    - Septal Perforator
  - Left Circumflex Artery (LCirc or LCx)
    - Obtuse Marginal (OM)
    - Ramus intermedius
- **Right Coronary Artery**
  - Marginal Branch
  - Posterior Descending Artery (PDA)

Concept of dominance

The Coronary Arteries

- **LAD**
  - Anterior LV
  - High Lateral LV
  - Septum (anterior 2/3)
  - Bundle Branches
- **Left Circumflex**
  - Low Lateral LV
  - Posterior LV
  - Left Atrium
  - Anterolateral papillary muscle of the mitral valve
- **SA Node (45%)**
- **AV Node (10%)**

SVC
Superior Vena Cava
LA
LA
Aorta
Left Pulmonary
Artery
LV
RV
Aorta
LV
RV
LA
RA
Found only in cardiac muscle
Most sensitive indicator of myocardial damage
   - Capable of diagnosing small amounts of myocardial necrosis not measured by rises in CK-MB levels
Approximately 30% of patients with non-ST elevation and normal CKMB levels will test positive for Non-STEMI
Of equal sensitivity and specificity
Troponin remains elevated for a long period
   - Beneficial for late presentation
   - Challenging for re-infarction
Positive troponin + ECG changes of injury / ischemia or ACS symptoms = INFARCT
More on Troponin

- Non infarct cardiac causes of elevated troponin: heart failure, left ventricular hypertrophy, tachyarrhythmias, pericarditis, cardiac trauma
- Non CAD causes of troponin elevation (sepsis, pulmonary emboli, chronic kidney disease, chemotherapy, respiratory failure, burns, neurological disease)
- Troponin I more specific in renal dysfunction
  - Patients with ESRD commonly have elevated troponin T
    - Not a false positive - relates to overall dysfunction of the cardiorenal system
  - < 10% of patients with ESRD have elevated troponin I in absence of ACS
- Elevated troponin levels are marker of risk and associated with an increased mortality – even when diagnosis is not myocardial infarction
- Degree of troponin elevation correlates with risk of death
- New high sensitivity troponin T

Timing of Release of Various Biomarkers After Acute Myocardial Infarction


Anderson JL, et al. J Am Coll Cardiol 2007;50:e1–e157, Figure 5.
Medical Management of STEMI

- ASA: 325 mg (non enteric coated)
  - If fibrinolytic therapy – 162-325 mg
- P2Y₁₂ inhibitor (loading dose before or at time of PCI)
  - If fibrinolytic therapy - clopidogrel only
- Anticoagulants (related to reperfusion strategy)
  - If fibrinolytic – weight based heparin x 48 hours
- Oral beta blockers ASAP
  - IV if hypertensive or tachycardic
- NTG – Sublingual vs IV
- Morphine Sulfate (Class I)
- Oxygen if hypoxemic (arterial oxygen saturation < 90%)
- High intensity statin therapy
- D/C NSAIDS
- ACE Inhibitors (within 24 hours)
  - Greatest benefit in anterior wall MI, LVEF < 40%, HTN, diabetes or chronic kidney disease
- Aldosterone Antagonists
  - Initiate within 7 days in those with LVEF <40%, HF, or diabetes

Reperfusion is primary management strategy.

STEMI Management

- **Reperfusion is number one treatment strategy**
- Primary Coronary Intervention (PCI) preferred treatment strategy if within 90 minutes
  - Goal: 90 minutes from 1st medical contact
- Fibrinolytics within 30 minutes of hospital presentation (or 30 minutes from EMS to fibrinolytics)

Facilitated PCI with full dose fibrinolytics is not recommended.

Rescue PCI may be done after failed fibrinolytics
The Winner!

Fibrinolytic Therapy

Primary PCI

However:
Timely reperfusion is the priority over method of reperfusion.

Interventional Revascularization: PCI
(Primary Coronary Intervention (in STEMI) or Percutaneous Coronary Intervention)

- PTCA: Percutaneous transluminal coronary angioplasty
- Coronary Stent
  - BMS: Bare metal
  - DES: Drug eluting
- Coronary Extraction Atherectomy
Interventional Revascularization

**Indications**
- **Reperfusion in STEMI**
  - Strategy of choice if 90 minute reperfusion time
- **Unstable angina / NSTEMI**
  - High risk features
- ** Stable angina**
  - Courage Trial - 2007

**Contraindications**
- When antiplatelet therapy is contraindicated

**Complications**
- Abrupt closure
- Dissection
- In stent thrombosis (acute or late)
- Down stream embolization
- Emergency CABG
- Bleeding or hematoma
- Pseudoaneurysm*
- Retroperitoneal Bleed
- Arterial Embolus
- Contrast nephropathy
- Restenosis (late)
- Coronary artery aneurysm (late)
- MI
- Stroke
- Death
Each community should develop a STEMI system of care consistent with minimum standards of AHA's Mission Lifeline.

Door to device time alone is not sufficient to further reduce mortality.

The average time of presentation after symptom onset is 1.5 to 2.0 hours.

Patient populations with the longest delays are women, African Americans, and the elderly.

Nurses can make an impact through patient and community education and awareness campaigns.

60 minutes is the golden hour: Survival rates improve significantly.
Medical Management of NSTE – ACS

- Dual antiplatelet
- Anticoagulation
- Oxygen if SpO$_2$ < 90%
- NTG
  - IV in first 48 hours for persistent ischemia, HTN, HF
  - Should not interfere with mortality reducing beta blockers or ace inhibitors
- MS (if NTG unsuccessful and other anti ischemic drugs on board)
- Beta Blockers (within 24 hours)
  - Start PO when hemodynamically stable
  - May use IV if hypertensive
- ACE Inhibitors (within 24 hours)
  - In select patients – pulmonary congestion or LVEF ≤ 40% – may also be used in other patients
- High intensity statin
- DC – NSAIDS

**Priority Treatment is to Attack the Platelet**

Medical Supportive Therapy: Similar to STEMI

Treatment of NSTE - ACS

- Attacking Platelet is number one treatment strategy

- Two antiplatelets agents are indicated

- There are 3 types of antiplatelet agents
  - Aspirin
  - P2Y$_{12}$ Receptor Antagonists
  - Intravenous GP IIb/IIIa Inhibitors
Dual antiplatelet therapy for invasive strategies in medium to high risk patients:
- ASA (and one of the following)
- P2Y12 / ADP Receptor blockers
  - Clopidogrel
  - Prasugrel
  - Ticagrelor
- GP II b / III a Inhibitors (*eptifibatide, *tirofiban, abciximab)
  - * preferred agents
  - Used only in special circumstances

Antiplatelet therapy also in conservative treatment:
- Prasugrel not unless PCI is planned
- Abciximab not unless PCI is planned

Dual antiplatelet therapy is also used after STEMI and after any coronary intervention.

Stent Restenosis Compared to Stent Thrombosis
**Early Invasive Option in NSTE-ACS**

**Versus Ischemia Guided Treatment**

- What is it?
  - Not waiting for failed medical treatment
  - Not waiting for + noninvasive test
  - Angiography with intent of revascularization
  - Done within 12 to 24 hours

**Overall reduction in mortality and increased quality of life.**

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**Early Invasive Option in NSTE-ACS**

- When to do it?
  - Refractory angina
  - Hemodynamic instability
  - Electrical instability
  - Initially stable patients with a high risk for clinical events

- **Excluded:**
  - Very frail elderly, severe hepatic, renal or pulmonary disease / active or inoperable cancer
  - Early invasive therapy is not recommended in patients with acute chest pain with a low likelihood of ACS
  - Early invasive therapy is not recommended in patients who do not want to consent to revascularization.
High Risk Features in UA / NSTEMI

- Recurrent angina / ischemia
  - Rest or low level activity with medical treatment
- Troponin +
- New or presumed new ST depression
- S&S HF or worsening mitral regurgitation
- High risk findings on noninvasive testing
  - EF < 35%, large anterior perfusion defect, multiple perfusion defects)
- Hemodynamic instability
- Sustained VT
- PCI within 6 months
- Prior CABG
- Reduced LV Function
- High risk TIMI or GRACE Score

Population > 75 years: 80% are high risk

Elderly:
cancer, renal insufficiency,
lung disease, anemia,
and heart failure are common
corollary conditions

Long Term Management of ACS

Medications to improve prognosis

- Aspirin
  - ASA benefits > in those > 65 years
  - Long term benefit with 81 mg
- Clopidogrel / Prasugrel / Ticagrelor
  - Dual antiplatelet therapy in conservative management for 12 months
  - Higher risk of bleeding with dual antiplatelet therapy
    - No elderly sub group data for clopidogrel
- Statins
  - Have greater benefit in elderly for reduction of future MI and death than in younger patient populations
**Medications to improve prognosis**

- Beta-blockers
- ACE inhibitors
  - Definite in select patients / reasonable in all
  - ARBs if ACE-I intolerant
- Aldosterone antagonists
  - EF ≤ 40 with HF or diabetes

**Impact long term ventricular remodeling**

**SL NTG Instruction**

- No more than 1 dose of SL NTG
  - If chest discomfort is unimproved or is worsening 5 min after 1 NTG call 9-1-1 immediately before taking additional NTG.
  - May take additional NTG while waiting EMS.
  - Chew ASA while waiting EMS.

- In chronic stable angina if symptoms are significantly improved by 1 dose of NTG may repeat NTG every 5 min for a maximum of 3 doses and call 9-1-1 if symptoms have not resolved completely.
Secondary Prevention: ACS and Stable CAD

- Smoking cessation
- Reduction of hyperlipidemia
  - LDL < 100 mg/dL or < 70 mg/dL (optimal)
- Hypertension control
  - <130/80 for kidney disease or diabetes
- Diabetes control Hb Alc < 7
- Physical activity minimum of 5 days / per week
  - 7 days recommended
- BMI 18.5 – 24.9 kg/mm²
- Phase II Cardiac Rehab
- Influenza Vaccine / Pneumonia Vaccine
Key Nursing Care Considerations

- Use oxygen for hypoxemia
- Assess response to beta-blocker therapy.
  - HR / BP
  - Arrhythmia control
- Assess for complications related to specific type of MI
  - Assess heart sounds for new holosystolic murmurs
    - Risk for myocardial rupture
  - Observe for signs of left ventricular dysfunction, including hypotension or clinical signs of heart failure.
  - Monitor ECG for conduction disturbances and arrhythmias
  - Assess for presence of RV infarct

Key Nursing Care Considerations

- Management of arterial access site
- Assessment for contrast nephropathy
- Restrict activity for the first 12 hours, and then begin Phase I Cardiac Rehabilitation (progressive mobility)
  - Referral to Phase II Cardiac Rehabilitation
- Utilize cardiac monitoring
  - ST-segment monitoring
  - Uninterrupted monitoring for first 24-48 hours
- Address addiction to nicotine
  - Consideration for nicotine withdrawal
- Focus on holistic approach to anxiety reduction
  - Include the family. Family visits do not have a negative impact on vital signs or cardiac rhythm
Complications of MI

- Cellular edema produces an inflammatory response.
- Recruitment of some stem cells leads to some tissue regeneration.
- Damaged tissue is bruised and cyanotic.
- Catecholamines are released from myocardial cells, thus increasing the risk of arrhythmias.
- Cardiac biomarkers are released.
- White blood cells invade the necrotic tissue within 2 to 3 days.
- Scavenger cells release enzymes to break down necrotic tissue.
- The necrotic wall can become very thin during this phase, and the patient is at risk for cardiac rupture.

**LINKING KNOWLEDGE TO PRACTICE:** Beta blockers are particularly important in suppressing cardiac arrhythmias in ischemic tissue during the acute remodeling phase because they suppress catecholamine release.
A weak collagen matrix forms by second week, myocardium is still vulnerable to re-injury.

Scarc formation has started by third week.

Necrotic area is completely replaced with scar tissue by week 6. Scar tissue does not contribute to the contractile function of the myocardium.

Myocardial necrosis (transmural and non transmural) and stunned or hibernating viable myocardium adversely affect the synergy of left ventricular contraction.

Surviving myocytes hypertrophy in an attempt to compensate for damaged tissue.

Excessive non-contractile collagen is present in the newly hypertrophied myocardium, leading to a ventricle that is stiff and noncompliant.

Regional wall motion dysfunction may improve due to recovery of post-ischemic viable myocardium. Does not necessarily correlate with an improvement in overall left ventricular ejection fraction.

Non-uniform left ventricular dilatation occurs. Occurs even in patients with recovery of regional wall motion abnormalities.

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**Left Ventricular Remodeling Following Myocardial Infarction**

*Acute Infarction, hours*

*Infarct Expansion, hours to days*

*Global Remodeling, days to months*
### Hemodynamic Alterations

#### Pre Shock Hypoperfusion
- Cold extremities
- Cyanosis
- Oliguria
- Decreased mentation

#### Shock
- Large LV infarction (> 40% myocardium)
- Right ventricular infarct
- Mechanical complication

**Heart Failure:**
- Cause: Ischemic, stunned, hibernating, or injured myocardium.
- HF after a STEMI is a predictor of mortality.
- Functional mitral valve regurgitation can co-exist.

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### Ventricular Arrhythmias

**Early**
- V-fib preventable cause of death
- 90% of sustained arrhythmias occur in first 48 hours

**Treatment:**
- Reperfusion
- Beta blocker therapy
- Correction of electrolytes
- Treatment of hemodynamic alterations

**No treatment needed:**
- AIVR, PVCs, & non-sustained VT.

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**> 48 hours post MI**
- Higher mortality than arrhythmias early in course
- ICD consultation if no reversible cause
Cardiac tamponade from free wall rupture
Formation of left ventricular diverticulum or pseudoaneurysm from free wall rupture
Left to right shunt from septal rupture
Acute mitral regurgitation from papillary muscle rupture.

10% of MIs
15% of in hospital deaths after MI
Without surgical intervention, the mortality rate for rupture is > 80% at two weeks.
Two high risk periods
  - 1st 24 hours
  - Within 1st week (3 to 5 days)
Associated with delayed fibrinolytics and late presentation
Activity

- After the patient achieves a rehabilitation level equivalent with activities of daily living, he/she can begin a walking program
  - 3 to 4 METS
  - Should be by time of discharge
  - Begin walking 5 to 10 minutes at a time
  - Patients should rate activity as moderate
- Shortness of breath means overexertion. Other signs of activity intolerance include: angina, dizziness, diaphoresis, prolonged fatigue, and nausea.
- The use of force to open windows or tight jar lids should be avoided in patients with lifting restrictions.

Linking Knowledge to Practice

✓ When providing instructions regarding weight lifting restrictions it is helpful to know the weight of common household items.

A gallon of milk = 8 pounds,
A bag of groceries between 5 & 10 pounds
A large basket of laundry 20 pounds or more.
Typical MET Levels of Common Activities

<table>
<thead>
<tr>
<th>Activity</th>
<th>MET Level</th>
<th>Activity</th>
<th>MET Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Washing, dressing, grooming</td>
<td>2.1</td>
<td>Grocery shopping</td>
<td>2.1</td>
</tr>
<tr>
<td>Light housework</td>
<td>2.51</td>
<td>Heavy equipment operation</td>
<td>6.0</td>
</tr>
<tr>
<td>Interior cleaning</td>
<td>3.01</td>
<td>Vehicle repair</td>
<td>2.93</td>
</tr>
<tr>
<td>Laundry</td>
<td>2.07</td>
<td>Walking (moderate to brisk)</td>
<td>3.0 to 5.0</td>
</tr>
<tr>
<td>Food preparation and clean up</td>
<td>2.54</td>
<td>Jogging / Running</td>
<td>6.3 to 8.0</td>
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<tr>
<td>Light shoveling (&lt;10 pounds)</td>
<td>6.0</td>
<td>Golfing (Pulling Clubs)</td>
<td>3.75 (4.3)</td>
</tr>
<tr>
<td>Lawn and garden</td>
<td>3.45 to 3.66</td>
<td>Strength training</td>
<td>3.0</td>
</tr>
<tr>
<td>Physical care for children</td>
<td>2.67 to 2.72</td>
<td>Dancing</td>
<td>4.5</td>
</tr>
<tr>
<td>Physical care for adults</td>
<td>2.89</td>
<td>Biking</td>
<td>8.0 (2015)</td>
</tr>
</tbody>
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Source: National Cancer Institute; Sanderson, 2010

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Driving

- Driving requires only 1.5 to 3.0 METS.
- Most patients with an uncomplicated hospital course can drive 1 week after discharge. Driving instructions should be compliant with any existing state regulations.
- Patients should be accompanied when they resume driving and should avoid stressful driving situations such as night driving, rush hour, high speeds, and driving during heavy rain or snow.
- Driving should be delayed for 2 to 3 weeks in patients with a complicated myocardial infarction. This includes patients who had a cardiac arrest, hypotension, arrhythmias, or heart failure during hospitalization.
**Travel**

- Patients can usually travel by air within 2 weeks if accompanied by a travel companion, and if the patient has sublingual nitroglycerin
  - If free of all angina symptoms and complications of their myocardial infarction

- Patients should also have airport transportation assistance to avoid excessive stress and rushing in the airport

- Patients should also take precautions when traveling to avoid the development of deep vein thrombosis

**Sex**

- After an acute coronary syndrome, stable patients can resume sexual activity with their usual partner in one week to 10 days (Anderson et al., 2011).

- Patients are uncomfortable asking about resuming sexual relationships, so instructions regarding sexual activity should be included as a routine part of all discharge instructions.

- Patients with a history of angina during sexual relationships may be instructed to take nitroglycerin prior to engaging in sexual activities.

- The average intimate session ranges from 2.5-4 METS for most people.
  - Walking at 2 mph on level ground is 2.5 METS. Mowing the lawn with a power mower or walking at 3.5 mph is 4 METS. Climbing up a flight of stairs is 8 METS.

- The biggest risk with sex in the cardiac patient is the possibility of arrhythmias, which is associated with sympathetic activity increased during arousal. Patients with uncontrolled or untreated hypertension need to discuss specific guidelines with their physician (Sotile & Cantor-Cooke, 2003).
Return to Work

- Low risk myocardial infarction (LVEF > 45%, successful revascularization with PCI, age < 70 years) can generally return to work after 2 weeks.

- Most myocardial infarction adverse events reach a low steady state at 10 weeks. This may guide decision making in some types of employment.

- Patients who need to return to physically demanding activities can have an exercise stress test that compares their performance on the stress test to the METs required for the activity. This will provide information about the ability and safety of engaging in activities based on the MET level achieved during exercise stress test. (Anderson et al., 2011).

Cardiac Rehabilitation

- Goals:
  - Increase functional capacity
  - Reduce disability
  - Improve quality of life
  - Modify cardiac risk factors
  - Reduce morbidity and mortality.

- Pooled data from a meta-analysis of studies involving the exercise portion of cardiac rehabilitation show a benefit of reduced all-cause mortality of approximately 25% when compared to usual care.

- In one study of over 600,000 Medicare patients, mortality rates were 21% to 34% lower in patients who participated in cardiac rehabilitation (Suaya, Stason, Ades, Normand, & Shepard, 2009).
Cardiac Rehabilitation

- Low-risk patients can implement an exercise prescription at home or in a community setting. Low-risk patients include those with absence of ischemia or arrhythmias on a stress test.

- High-risk patients should be in medically supervised exercise programs. They are defined as patients with ischemia or serious arrhythmias on a stress test.

- Underutilization of cardiac rehabilitation.

Treating the Whole Patient

- **Depression**
  - Approximately 1 in 5 patients hospitalized with MI have major depression. There is also evidence that depression continues for several months after discharge (Fihn et al., 2012; Bush et al., 2005).
  - There is strong evidence that patients who are depressed post MI have a higher rate of mortality from both cardiac and non-cardiac causes (Bush et al., 2005).

- **Anxiety and Stress**
  - In post MI patients, interventions to reduce stress can reduce recurrent cardiac events by as much as 35-75% (Gibbons et al., 2002).

- **Social Support**
  - **Role Identity**
Our Vision:

Impact every patient and family on their journey and provide safe passage by meeting them where they are, connecting with them in a meaningful way, and delivering care with wisdom and intention.

- Cindy and Karen

BE THE BEST THAT YOU CAN BE EVERY DAY. YOUR PATIENTS ARE COUNTING ON IT!

Contact me for questions at: www.cardionursing.com