Clinical Practice Guideline:

ACUTE CORONARY SYNDROME (ACS)

Type: Medical Diagnosis/Treatment/Procedure

Target Population: Adult/Geriatric

PROFESSIONAL PROCESS

GOALS/OUTCOMES:

A. Signs and symptoms of listed potential problems will be absent or manageable.
   1. Ischemia leading to Infarction
   2. Dysrhythmia/Arrhythmia
   3. Hemodynamic Instability
   4. Pericarditis leading to Pericardial Effusion
   5. Cardiovascular Structural Defects
   6. Mural Thrombosis leading to Embolism
   7. Acute Pain
   8. Situational Response

B. Patient/family/significant other (S.O.)/caregiver will verbalize and/or demonstrate an understanding of teaching/learning goals listed below: (Refer to Education Outcome Record)
   1. Acute coronary syndrome overview [e.g., description, anatomy, physiology, cause(s)].
   2. Signs/symptoms: Angina [e.g., discomfort (uncomfortable pressure, squeezing, fullness, pain) in chest lasting greater than 2 minutes or leaves and returns; discomfort in 1 or both arms, upper back, neck, jaw, or stomach; shortness of breath; diaphoresis; lightheadedness; nausea, vomiting].
   3. Signs/symptoms: Myocardial Infarction [e.g., mild or severe discomfort (pressure or pain in middle of chest, may extend into shoulder and arm, abdomen or jaw); shortness of breath; diaphoresis; nausea/vomiting; weakness or lightheadedness; paleness; burning feeling in upper abdomen; upset stomach; sudden weakness or unexplained tiredness; more intense than angina and lasts longer than 15 minutes].
   4. Treatment plan (e.g., Medications: nitrates, beta-blockers, calcium channel blockers, ACE inhibitors, aspirin, cholesterol-lowering medicines; Heart Healthy Diet: low saturated fat; Activity: regular exercise, stop activity if experiencing pain, weight-loss program if overweight).
   5. Modifiable risk factors/triggers: Angina (e.g., exercise, heavy physical labor, stress/upset, in cold air, digesting a big meal, high blood pressure, high cholesterol, high blood sugar).
   6. Modifiable risk factors/triggers: Myocardial Infarction (e.g., cigarette smoking, family history of heart attack, diabetes, overweight, high blood pressure, high blood cholesterol, low HDL cholesterol, stress, lifestyle without physical activity).
   7. Self-management (e.g., tobacco cessation; regular blood pressure checks; diet consistent in vitamin K if taking warfarin; regularly scheduled INR if nonvalvular atrial fibrillation/flutter; pacemaker checks with cardiologist if pacemaker; CPR education for family/caregiver).
   8. Rehabilitation to improve functional independence (e.g., cardiac rehabilitation).
   9. When to seek medical attention (e.g., contact EMS or 911 if chest pressure, squeezing or pain that lasts greater than five minutes or leaves/returns; pain or discomfort in one or both arms, neck, jaw or upper back that lasts more than five minutes or leaves/returns AND lightheadedness, trouble breathing, nausea or sweating; pain remains after taking three nitroglycerin tablets five minutes apart).
   10. General goals (room/unit routine, pain, medication, diagnostic tests/procedures, dietary modifications, hygiene, infection prevention, rehabilitation, medical equipment/supplies, tobacco cessation, resources for support).

ASSESSMENT/INTERVENTIONS/CLINICAL REASONING/DECISION-MAKING:

A. Assess and document readiness and ability to learn, learning needs and preferences. (Refer to Pre-Teaching Assessment on Education Outcome Record)

B. Collaborate with interdisciplinary resources related to significant changes in patient status and for the continuum of care (e.g., Physician, Nursing, Social Work/Services, Dietitian/Nutrition Services, Pastoral Care, Pharmacy, Respiratory Therapy, Occupational Therapy, Physical Therapy, Child Life, Speech Language Pathology, Home Care Services).

C. Assess, Monitor, and Detect:
   • The impact of other preexisting health problems.
   • The impact on psychosocial, cultural, sexual, developmental and spiritual well-being.
   • The impact of common diagnostic studies/laboratory values: [e.g., electrocardiogram (ECG); laboratory values (electrolytes, blood urea nitrogen (BUN), creatinine (Cr), magnesium, potassium, glucose, glycosolated hemoglobin (A1C) for diabetics, CBC, Troponins I and T, myoglobin, C-reactive protein (CRP), creatine kinase of the cardiac muscle (CK-MB), lactic dehydrogenase (LDH), chest x-ray (CXR), graded exercise test (GXT), pharmacologic stress echocardiogram, percutaneous coronary intervention (PCI), percutaneous transluminal coronary angioplasty (PTCA); transesophageal echocardiography (TEE), magnetic resonance imaging (MRI), computed tomography scan (CT scan)].
   • Baseline vital signs and trends.
D. Assess, Monitor, Detect, Prevent and/or Modify the listed potential problems and implement interventions as appropriate:

**Ischemia leading to Infarction AEB (As Evidenced By):** (5; 6; 33; 79; 84)

- **Ischemia:** substernal chest pain/discomfort/pressure/angina (usually less than 15 minutes duration), may radiate to arm/neck/back/jaw; dysrhythmias; discomfort of other areas in upper body; shortness of breath (SOB); decreased exercise tolerance; nausea; normal cardiac enzyme/biomarker levels. Atypical presentation: fatigue, syncope, weakness, SOB (most common in women, diabetic and geriatric patients).

Additional Geriatric considerations: altered mental status. If pre-existing altered mental status or dementia is present the patient may have no recollection of recent symptoms or have no complaints.

- **Acute Myocardial Infarction:** change in heart rate (HR) and rhythm/BP/cardiac output; systolic murmur; chest pain/discomfort/pressure (usually greater than 15 minutes duration), may radiate to arm/neck/back/jaw, unrelated by nitroglycerine; diminished heart sounds (especially S1), S4 with left ventricular dysfunction; decreased peripheral pulses, slow capillary refill; tachypnea, SOB; nausea/vomiting (NV); diaphoresis; restlessness, anxiety, confusion, agitation, denial, anger, feeling of impending doom; weakness; decrease in urine output

- **ST-segment elevation myocardial infarction or presumed new left bundle branch block (LBBB):** ST-segment elevation greater than 1 mm in two or more contiguous precordial leads or two or more adjacent limb leads

- **High-risk UA/Non-ST-elevation myocardial infarction/new or presumably new left bundle branch block (LBBB):** ischemic ST-segment depression greater than or equal to 0.5 mm or dynamic T-wave inversion with pain or discomfort; nonpersistent or transient ST-segment elevation greater than or equal to 0.5 mm for less than 20 minutes is also included in this category. Laboratory values: positive cardiac markers (increased CK-MB/Troponin I or T/myoglobin)

- **Right ventricle (RV) infarction:** Jugular venous distension (JVD); decreased BP; clear breath sounds; heart block; right ventricular (RV) infarction

Risks to safety.

- **Ischemia:** Rapid onset and acceleration of rapid beating of sinus node (100 to 180 bpm in adults, up to 200 with extreme exertion, 100 to 140 in older adults) leading to decreased cardiac output (2)

- **Supraventricular tachycardia:** rapid beating of sinus node that starts and stops abruptly (2)

- **Sinus node dysfunction:** symptomatic atrioventricular (AV) pauses greater than or equal to three seconds or sinus bradycardia with rates less than 50 bpm; may lead to syncope (30)

**Dysrhythmia/Arrhythmia AEB:**

- **Labile heart rhythm/rate/pulse amplitude:** palpitations; decreased capillary refill; change in BP; dizziness; syncope; N/V; change in mental status; increased anxiety; increased confusion; increased agitation; change in level of consciousness; cool, clammy skin; change in ECG, increased ectopy; change in cardiac output; change in cardiac index; change in CVP; change in pulmonary artery wedge pressure (PAWP); change in left arterial pressure (LAP); change in systemic vascular resistance (SVR); decreased SpO2; decreased percent venous O2 saturation (SvO2); decreased urinary output

- **Atrial:**
  - Symptomatic bradycardia: HR less than 60; syncope or near syncope, transient dizziness or lightheadedness, confusion; fatigue, exercise intolerance; may lead to heart failure (30)
  - Sinus tachycardia: gradual onset and acceleration of rapid beating of sinus node (100 to 180 bpm in adults, up to 200 with extreme exertion, 100 to 140 in older adults) leading to decreased cardiac output (2)
  - Supraventricular tachycardia (SVT): rapid beating of sinus node that starts and stops abruptly (2)
  - Sinus node dysfunction: symptomatic atrioventricular (AV) pauses greater than or equal to three seconds or sinus bradycardia with rates less than 50 bpm; may lead to syncope (30)
12. Correlate cardiopulmonary status to medications, treatments/diagnosis/procedure, oxygenation, electrolyte values (e.g., potassium, sodium, calcium and magnesium); hydration status, age and baseline assessment data. (22; 55; 88) {Grade C}

13. Correlate peripheral pulses to atrial HR for one minute. (55; 57) {Grade A}

14. Correlate dysrhythmias to ischemias; evaluate/anticipate need for respiratory support to maintain O₂ saturation above 90 percent [e.g., O₂ therapy, airway placement, noninvasive/invasive ventilation]. (5; 40) {Grade C}

15. Anticipate need for peripheral vascular access, IV infusion/bolus. (22; 38; 57; 62) {Grade C}

16. Promote hemodynamic stabilization via nonpharmacologic methods [e.g., decrease head of bed (HOB), promote calm environment]. (55) {Grade C}

17. Anticipate need for baseline diagnostic/laboratory studies [e.g., 12-lead ECG, arterial blood gases (ABGs), electrolytes, cardiac enzymes, baseline coagulation studies]. (5; 22; 37; 55; 62) {Grade C}

18. Evaluate need for monitored/critical care bed. (55; 88) {Grade A}

19. Screen for anxiety, depressive disorders and/or psychosocial implications related to diagnosis of heart disease. (22) {Grade C}

20. Refer patient to smoking cessation program, if indicated. (22; 37; 55) {Grade C}

Atrial:

21. Mediate underlying cause of dysrhythmia to control sinus rhythm/ventricular rate. (55; 82) {Grade A} ATRIAL: rate versus rhythm control

22. Anticipate administration of medications (e.g., electrolytes; diuretics, beta-blockers, digoxin, calcium channel blockers, antidysrhythmics, anticoagulants). (15; 24; 55; 57; 62; 87) {Grade A}

23. Anticipate discontinuation of any medications that would contribute to symptomatic bradycardia (e.g., beta-blockers, digoxin, verapamil). (22) {Grade C}

24. Anticipate antithrombotic/anticoagulant therapy (in nonvalvular A. fib/flutter) for prophylaxis of VTE [e.g., low molecular weight heparin (LMWH), heparin, aspirin, vitamin K antagonist (warfarin)]. (32; 38; 55) {Grade A}

25. Monitor for bleeding and correlate laboratory results to dose of antithrombotic medications [e.g., prothrombin time/partial thromboplastin time (PTT)/international normalized ratio (INR)] and dietary intake of vitamin K. (22; 32; 38; 55) {Grade A}

26. Anticipate need for emergency cardiac intervention (e.g., cardioversion, radiofrequency ablation). (15; 36; 57) {Grade A}

27. Anticipate need for temporary pacing (transcutaneous or transvenous) in patients with symptomatic bradycardia [e.g., unresponsive to atropine, presence of anterior myocardial infarction, third degree AV block in inferior myocardial infarction, alternating left/right BBB]. (22) {Grade C}

Junctional/Ventricular:

28. Anticipate need for physiologic monitoring [e.g., continuous ECG, CVP/pulmonary artery pressure, echocardiography (especially in geriatric population)], respiratory support (e.g., airway management, noninvasive or invasive mechanical ventilation), fluid balance and hemodynamic support (e.g., fluids, electrolytes, inotropic agents, antidysrhythmics, beta-blockers, anticholinergics, vasopressors). (24; 41; 80; 84; 88) {Grade A}

29. Anticipate need for emergency cardiac intervention (e.g., defibrillation, pacing, cardioversion). (88) {Grade A}

30. Assess for potential of deteriorating cardiac status/impending cardiac arrest with first priority being activation of rapid response team. (24; 88) {Grade A}

31. Anticipate need for emergent procedure [e.g., coronary angiography, pacemaker/automatic implantable cardiac defibrillator (AICD)]. (5) {Grade C}

Hemodynamic Instability AEB: (6; 43; 60; 64)

- Heart failure: decreased cardiac index/SpO₂; angina; cool, pale, diaphoretic skin; decreased capillary refill/quality of peripheral pulses; S₃, S₄, pericardial rub; increased weight; positive intake and output balance; decreased urinary output; fatigue

- Systolic (left heart): decreased BP, increased HR/pulmonary artery pressure (PAP)/pulmonary artery wedge pressure (PAWP)/systemic vascular resistance (SVR); change in RR, wheezes, crackles, pink frothy sputum, increased work of breathing, orthopnea, cyanosis

- Diastolic (right heart): JVD; increased CVP; pleural effusion; hepatomegaly; ascites; abdominal tenderness; positive hepatogjugular reflex; N/V; dependent peripheral edema

- Cardiogenic shock: signs/symptoms of heart failure plus restlessness, confusion, sense of impending doom; change in level of consciousness; peripheral vasoconstriction, narrowed pulse pressure; core temperature less than 36 degrees Celsius

32. Correlate cardiovascular/cardiopulmonary status to disease process, precipitating events, fluid status, medications/treatments, laboratory values, neurologic status, abdominal girth, intra-abdominal pressure, activity tolerance, sleep/rest patterns, visible bleeding, ECG, echocardiography and baseline assessment data. (43; 48; 60; 64) {Grade A}

33. Promote hemodynamic stability (e.g., lower HOB for hypotension, control of hypertension, blood/fluids, quiet environment). (60; 64) {Grade A}
34. Anticipate need for electrolyte replacement (e.g., potassium, calcium, magnesium) especially if patient is undergoing diuresis. (60; 64) (Grade A)
35. Evaluate impact of medication therapy on myocardial workload. (54) {Grade A}
36. Titrate vasodilators to decrease myocardial workload and cardiac index. (54) (Grade C)
37. Use vasopressors and inotropes cautiously to avoid increased myocardial O₂ demand. (33) (Grade C)
38. Evaluate need for active/passive rewarming techniques (e.g., warm blankets, water/air hyperthermia system, increased ventilator temperature). (60) (Grade C).
39. Prepare for possible intervention [e.g., intraaortic balloon pump (IABP)]. (6) (Grade B)
40. Promote rest and minimize O₂ consumption (e.g., allow rest periods, coordinate/cluster care, neutral-thermic environment). (60) (Grade A)

Pericarditis leading to Pericardial Effusion AEB:
- **Pericarditis**: pain described as being different from angina/myocardial infarction pain: sudden or gradual onset, sharp, stabbing, substernal; movement or deep inspiration aggravates pain; alleviated by sitting up/leaning forward; cough; dysphagia; pain may radiate to left upper back, shoulder or arm; pericardial friction rub; dysrhythmias; increased HR/respiratory rate (RR)/erythrocyte sedimentation rate (ESR)/white blood cell (WBC); low grade temperature; diffuse ST-segment elevation; complaints of dyspnea; chills; weakness (85)
- **Pericardial effusion**: muffled heart tones; paradoxical pulse; increased HR; decreased BP; disappearance of pericardial friction rub; pericardial fluid seen on echocardiogram; enlarged cardiac silhouette on CXR (83)

Cardiovascular Structural Defects AEB: (6; 84)
- **Left ventricular aneurysm**: paradoxical pulse; bulging of precordium; change in HR; ventricular ectopy
- **Septal rupture**: acute left ventricular failure; holosystolic murmur with precordial thrill; acute pulmonary edema
- **Mitral valve/Papillary muscle rupture**: acute left ventricular failure; rapid progression to pulmonary edema and often cardiogenic shock; early or holosystolic murmur
- **Ventricular septal rupture**: tamponade; cardiogenic shock with narrow pulse pressure; pulseless electrical activity (PEA) leading to cardiac arrest

41. Coordinate planned activities with pain management plan and position for comfort (e.g., upright). (83; 85) (Grade C)
42. Verify need to continue antplatelet/anticoagulation therapy. (83) (Grade C)
43. Anticipate need to prepare patient for diagnostic procedures [e.g., CXR, transesophageal TEE, MRI, CT scan] and urgent interventions (e.g., pericardiocentesis, balloon pericardotomy, pericardial sclerosis). (83) (Grade C)
44. Anticipate need for hemodynamic monitoring (e.g., pulmonary artery catheter) and support (e.g., fluid resuscitation, inotropic agents). (83) (Grade C)
45. Anticipate need for medications [e.g., steroids, aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs) (may be contraindicated due to possible delayed myocardial scar healing)]. (83) (Grade C)

46. Flatten head of bed and elevate feet to optimize perfusion to vital organs. (72) (Grade C)
47. Titrate vasopressors, vasodilators and inotropic medications to optimize hemodynamic function. (6) (Grade B)
48. Prepare for emergency intubation and placement of large bore IV devices. (84) (Grade C)
49. Anticipate need for rapid administration of fluid/volume expanders and adjustment of vasoactive medications. (6; 84) (Grade B)
50. Prepare for emergent insertion of intra-aortic balloon pump (IABP) and/or cardiac surgery or pericardiocentesis (in the case of septal rupture). (6; 84) (Grade B)

Mural Thrombosis leading to Embolism AEB:
- **Mural thrombus**: asymptomatic unless portion of thrombus dislodges, leads to ischemic CVA, pulmonary/ peripheral embolism
- **Cerebral embolus**: sudden change in cognitive/sensoric/motor function; suddenly changed level of consciousness; failure to regain consciousness after general anesthetic; seizure
- **Pulmonary embolus**: sudden cardiovascular instability/respiratory distress; cough; decreased end-tidal carbon dioxide (EtCO₂); increased RR; marked decrease in O₂ saturation (hemoglobin) (SaO₂); changed arterial blood gases (ABGs); hypoxemia; thoracic/upper abdominal pain; tachycardia; petechiae; pulmonary infiltrates; positive D-dimer level; positive ventilation perfusion (VQ scan); positive pulmonary angiography or positive helical computed tomographic scan (14; 17; 73)
- **Deep vein thrombosis (DVT) leading to venous thromboembolism (VTE)**: redness, warmth, tenderness, edema of affected area; increased temperature; impaired neurovascular status; apprehension; restlessness; venous congestion; deep/local muscle pain; size asymmetry; color change; palpable cord along vein; abnormal D-dimer level; positive venous ultrasonography (17; 73)

51. Initiate strategies to prevent/treat DVT/VTE:
- Prevent venous stasis/pooling of blood in extremities [e.g., compression stockings, prevent pressure/constriction, frequent position change, avoid compression of vena cava, ankle dorsiplantar exercises, avoid crossing legs, intermittent pneumatic compression, neutral position, HOB elevated less than 30 degrees]. (4; 7; 16; 49; 65) {Grade A}
- Promote early and frequent ambulation. (17; 49) {Grade B}
- Advocate for VTE prophylactic medication. (7; 49; 50; 52) {Grade B}
- Monitor for bleeding and correlate laboratory results to dose of antithrombotic medications. (16; 49) {Grade B}
- Avoid deep/vigorous massage when suspected signs and symptoms of DVT or receiving antithrombotic therapy. (25; 31) {Grade C}
Acute Pain AEB: (19)

Patient may have nociceptive, neuropathic or a combination of both types of pain:

- **Nociceptive (somatic):** recent onset; well-localized; sharp, pin-prick, aching, stabbing or throbbing
- **Nociceptive (visceral):** poorly localized; diffuse with vague complaints such as generalized ache or pressure; may be referred to sites remote from the primary injury. Autonomic symptoms include N/V, hypotension, bradycardia, sweating
- **Neuropathic:** radiating or specific burning, electric-like, shooting, tingling or lancinating (stabbing, piercing) pain. Physical exam may reveal allodynia (pain on light touch), hypesthesia or hyperalgesia (relatively decreased or increased perception of noxious stimulus) or hyperpathia (exaggerated pain response). Symptoms usually are experienced distal to the site of injury
- **Geriatrics - additional considerations:** changes in mental status; changes in appetite; agitation; rigidity-resistance to certain movements during care; withdrawn affects; paucity of conversation; depression; allodynia; decreased activity levels; loss of function; lack of adaptation skills (9)
- **Nonverbal patient (e.g. advanced dementia, mechanically ventilated and/or sedated, unconscious):** sympathetic stimulation [e.g. facial expression (relaxed versus brow lowering, eyelid closing, grimacing), upper limb movements (no movement versus partially bent, fully bent with finger flexion, permanently retracted)], mechanical ventilation compliance (tolerating movement versus coughing but tolerating most of time, fighting ventilator, unable to control ventilation] (1; 45; 68; 71; 77) {Grade A}

52. For patients able to communicate, evaluate pain using identified tool/self-report description [e.g., onset, location, radiation frequency/duration, intensity/severity at rest and with activity, aggravating/relieving factors (medications, positioning, treatment devices), exacerbation]; behavioral indicators [e.g., moaning, rubbing a particular area, guarding; decreased attention span, agitation, inability to rest/sleep, inability to alleviate distress; facial expression (brow lowering, orbital tightening, levator contraction, eyelid closing), upper limb movement, mechanical ventilator compliance], patient’s stated perception of pain tolerance, type of procedure/treatment/disease process, expected pain progression, cultural factors that may influence pain perception and baseline assessment data. (1; 10; 18; 19; 34; 42; 45; 47; 68; 71; 77) {Grade C}

53. For patients unable to communicate (e.g., advanced dementia, mechanically ventilated and/or sedated, unconscious), evaluate pain using report of pain/behavior changes from family/caregiver; pain behavior checklist sympathetic stimulation [e.g., facial expression (relaxed versus brow lowering, eyelid closing, grimacing), upper limb movements (no movement versus partially bent, fully bent with finger flexion, permanently retracted)], mechanical ventilation compliance (tolerating movement versus coughing but tolerating most of time, fighting ventilator, unable to control ventilation]) and consider analgesic trial. If behaviors do not improve with analgesia, consider addition of sedatives and/or antipsychotic medications. (1; 45; 68; 69; 71; 77) {Grade C}

54. For patients unable to respond behaviorally to pain (e.g., pharmacologically paralyzed, flaccid) assume pain is present if patient has chronic painful condition(s), painful activity (e.g., physical therapy) or potentially painful procedures (e.g., suctioning, turning, dressing changes) and begin to treat appropriately with analgesia trial. (1; 45; 68; 69; 71; 77) {Grade C}

55. Establish with patient what level of discomfort is acceptable that will allow for maximal function with basic/instrumental activities of daily living (BADL/IADL). (19; 47) {Grade B}

56. Mutually develop plan for pharmacologic/nonpharmacologic comfort measures as appropriate [e.g., medications (appropriate to type of pain reported), procedures (neural or sympathetic blocks), adjuvants such as complement and alternative therapies (massage, touch, hypnosis, acupuncture), physical medicine/rehabilitation techniques (assistive devices, orthotics, transcutaneous electrical nerve stimulation, ultrasound, massage, therapeutic exercise), psychological interventions (behavioral therapy, biofeedback, cognitive behavioral therapy, counseling, relaxation)]. (19; 47) {Grade B}

57. Account for the possibility of acute pain in the presence of pre-existing chronic pain syndrome and adjust medications appropriately (e.g., use patient’s prehospital basal rate plus adjustment for acute pain). Collaborate with patient on what has worked best for him/her in the past. (19; 26) {Grade B}

58. Initiate pain management plan that includes scheduled around-the-clock pain medication dosing with PRN breakthrough medications as prescribed if pain is present the majority of a 24-hour time span. (10; 19; 47) {Grade B}

59. Provide individualized pain management measures prior to procedure(s)/planned activities (e.g., turning, suctioning, wound care/drain removal), (47; 77) {Grade B}

60. Anticipate need for management of drug-induced side effects. For opioids: N/V, constipation, itching, myoclonus and respiratory depression. For NSAIDs: dyspepsia, prolonged bleeding times, renal dysfunction, urinary retention, hypertension. (10; 11; 19; 47) {Grade B}

Situational Response AEB:

- expression of anxiety, fear, denial, guilt, frustration, vulnerability, disappointment, anger, shock, blame, sadness, depression, hopelessness, apprehension, uncertainty, ambiguity, grief/loss (e.g., loss of control, loss of previous family/social role, loss of independence); increase stress (e.g., financial, relationships, roles, responsibilities) (20; 58; 81)

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61. Develop trust relationship/rapport through therapeutic presence, active/empathic listening and sensitivity to nonverbal communication. (20; 29; 58; 59; 74) {Grade C}

62. Correlate patient/support system response to medical illness/injury with ability and/or readiness to comprehend information, past experiences/history (including mental health), current situation, developmental stage, medications/substance use and baseline assessment data. (20; 29; 74) {Grade C}

63. Encourage verbalization of feelings regarding current situation. (20; 29; 58; 74; 81) {Grade C}

64. Support coping by recognizing current strategies and developing new strategies (e.g., journaling, relaxation techniques, guided imagery, problem solving, prayer). (20; 29; 58; 74; 81) {Grade C}

65. Evaluate the need for anticipatory guidance (e.g., provide information on realistic expectations, education/resources) and encourage patient/support system to ask questions regarding treatment/procedures. (20; 58; 59) {Grade C}

66. Facilitate the presence of and private time with the patient support system (e.g., partner, family members, children, friend, spiritual advisor). (20; 29; 59; 74) {Grade C}

67. Address concerns, offer reassurances and provide support for patient/support system. (20; 29) {Grade C}
A. **CLINICAL DESCRIPTION:** (33)
The term acute coronary syndrome (ACS) refers to myocardial ischemia (an imbalance between supply and demand of myocardial O2) and is reflective of the clinical diseases of acute myocardial infarction and unstable angina. Heart disease is the leading cause of all deaths in people over age 65 in the U.S. (31.8 percent). (21)

1. **Classification and Symptoms of ACS:** (5; 33; 84)
   a. **Angina pectoris (chest pain):** classic symptom of ACS. Pain intensity ranges from absent (silent ischemia) to severe. The pain is usually substernal and may radiate to the epigastic region, jaw, shoulder, elbow or forearm. Chest pain is described as heaviness, tightness/pressure, constriction, indigestion or burning. **Associated symptoms include:** SOB, diaphoresis, N/V, weakness, extreme fatigue, anxiety and/or sense of impending doom.
     
     **Variants of angina include:**
     - **Chronic stable:** episodic pain lasting 5 to 15 minutes. Brought on by predictable increase in physical effort, predictable intensity and duration of symptoms. Relieved with rest or nitroglycerin.
     - **Unstable:** patient notes a change in severity or duration of symptoms of chronic stable angina. Types include new-onset exertional; angina of increased frequency or duration or refractory to nitroglycerin and angina at rest.
     - **Variant (Prinzmetal):** occurs at rest or sleep. Related primarily to coronary artery spasm. Triggered by smoking. Frequently seen in connective tissue disease, arterial inflammation and infection.

   b. **Unstable angina/non-ST-elevation myocardial infarction:** closely related conditions with similar pathogenesis and clinical symptoms. They differ primarily in whether the ischemia is severe enough to release markers of myocardial injury. If biomarkers are detected in the bloodstream, the patient is considered to have non-ST elevation myocardial infarction.
     - **Pathogenesis:** decreased myocardial perfusion commonly caused by coronary artery narrowing secondary to nonocclusive thrombus that developed on a disrupted atherosclerotic plaque.
     - **Presentations of unstable angina:**
       - Rest angina (occurs at rest and prolonged greater than 20 minutes)
       - New onset severe angina
       - Increasing angina (previously diagnosed angina that has become more frequent)
   c. **ST-segment elevation myocardial infarction or presumed new left bundle branch block (LBBB):**
     - **Pathogenesis:** prolonged myocardial ischemia that leads to irreversible injury and necrosis.
     - **Presentation:** chest pain lasting more than 30 minutes. Pain is usually severe, but may be mild or absent.

2. **Location of myocardial infarctions: associated findings, management and complications:**
   a. **Inferior myocardial infarction:** occlusion of right coronary artery (RCA) and/or left circumflex artery. Right ventricular infarctions should always be suspected as well. ECG changes: ST-segment increased II, III, AVF; reciprocal changes: V1-3, decreased ST, diagnostic Q wave in two adjacent or contiguous leads.
     - **common dysrhythmia:** bradycardia, primary heart block (HB), accelerated junctional, secondary to heart block (Type I), third degree heart block (rare) (Note: Do not give lidocaine if ventricular beats are escape beats).
     - **common findings:** N/V; cold, clammy skin; murmur or mitral valve regurgitation (MVR)/papillary muscle dysfunction.
     - **major complications:** A-V nodal blocks; complete heart block.

   b. **Anterior myocardial infarction:** occlusion of the left anterior descending coronary artery (LAD). ECG change, abnormal Q wave and ST elevation in leads V1-4; ST-segment depression in inferior leads II, III and AVF would be a more ominous feature, T inversion in V1 and V2.
     - **common dysrhythmia:** sinus tachycardia, atrial flutter (A flutter), atrial fibrillation (A fib), third degree heart block, ventricular tachycardia (V-tach), ventricular fibrillation (V-fib), BBB and junctional rhythms
     - **common findings:** decreased BP; decreased urine output; S3, S4; increased PAWP; pulmonary edema
     - **major complications:** septal rupture
   c. **Lateral myocardial infarction:** occlusion of circumflex coronary artery, ECG change, increased ST-segment in I, AVL, V5 and V6. Anterolateral occlusion of diagonal branch left anterior descending coronary artery (LAD).
     - **common dysrhythmia:** atrial dysrhythmia. SVT, ST, and junctional rhythms
     - **common findings:** decreased BP, decreased urinary output, S3, S4, increased PAWP
     - **major complications:** watch for extension to anterior inferior or posterior wall
   d. **Posterior myocardial infarction:** occlusion of right coronary artery (RCA), ECG change, increased ST-segment in V1-3. Posterior-lateral: tall R waves V1-2. Occlusion of circumflex, decreased ST-segment in V1-3, increased ST in 1, AVL, V5 and V6
     - **common dysrhythmia:** bradycardia and heart blocks
     - **common findings:** decreased HR; decreased BP
     - **major complications:** heart failure (HF) and pulmonary edema; monitor for extension to lateral or inferior wall or right ventricle
   e. **Right Ventricular myocardial infarction:** occlusion of proximal RCA, often associated with inferior wall myocardial infarction. ECG change, increased ST in V1, V2, V3R.
     - **common dysrhythmia:** AV conduction block
     - **common findings:** hypotension; increased CVP; decreased PAWP/cardiac index; JVD; clear breath sounds; decreased cardiac output
2. **Causes of ACS include:**
   
i. coronary artery dissection; especially in younger women and those who use cocaine  
h. inflammation of epicardial arteries  
g. underlying coronary artery disease, which may be unmasked by severe anemia  
f. cocaine and amphetamines, which increase myocardial O₂ demand and may cause coronary vasospasm  
e. hypoxia, as in carbon monoxide poisoning or acute pulmonary disorders  
d. embolic occlusion of the coronary arteries  
c. ventricular hypertrophy due to hypertension, valvular disease or cardiomyopathy  
b. coronary vasospasm  
a. atherosclerotic plaque, ruptured or eroded (most common cause)  

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### B. PATHOPHYSIOLOGY/Etiology/Risk Factors for Potential Problems:

1. **Ischemia leading to Infarction:** occurs when myocardial O₂ demand is greater than supply. Area surrounding necrosis is affected by a prolonged decreased O₂ supply to myocardial tissue leading to instability of cell membranes leading to dysrhythmias. Pain is an indicator of decreased O₂ supply to tissue. May be indicated by T-wave inversion on ECG. (5; 33; 84)

2. **Dysrhythmia:**
   
a. **Definition:** (27; 28) Arrhythmia: “any cardiac rhythm other than normal sinus rhythm. Such a rhythm may be either sinus or ectopic in origin and either regular or irregular. An arrhythmia may be due to a disturbance in impulse formation or conduction, or both.” Not all arrhythmias result in harm, however, they do interact with other organ systems.
   
b. **Pathophysiology/Etiology:** (27; 28) Arrhythmias either result from problems of other organ systems (e.g., renal failure) or produce dysfunction in other organ systems due to hemodynamic compromise or embolic phenomena.
   
   - **Iatrogenic:** postsurgical; or secondary to multiple etiologies (e.g., postprocedural, mechanical, ischemic, metabolic, infectious and multifactorial).
   - **Noniatrogenic:** genetic or congenital; may also be secondary to multiple etiologies [e.g., scar-related (stretch or fibrosis), metabolic, infectious or multifactorial].

   **Classification:** based on the anatomical hierarchy and the type of mechanism of the arrhythmia. (27; 28)
   
   - Atrial
   - Junctional
   - Ventricular
   - Atrioventricular

   **Subclassification includes:**
   
   - Early (also known as perioperative): occur during hospitalization or within 30 days of surgery.
   - Sustained: lasting greater than 30 seconds, or requires immediate termination due to hemodynamic compromise.
   - Frequency: paroxysmal, recurrent, chronic recurring or permanent

   c. **Risk factors:** anesthesia, hypoxia, hypotension, electrolyte imbalance (e.g., diarrhea, renal failure), invasive lines (e.g., central venous), coronary artery injury, compression of ventricle by a chest tube, hepatic failure, pain (releases catecholamines that produce tachycardia), history of smoking.

3. **Hemodynamic Instability:** an alteration in blood flow through the cardiovascular system circuit in which the circuit can no longer meet the metabolic demands due to a decrease in cardiac filling pressure. (64)
   
a. The venous side of the cardiovascular system is conceptually divided into two compartments: the peripheral compartment and the smaller intrathoracic central venous compartment.
   
b. Each segment has a different role to play in the overall circulatory operation because of inherent differences in anatomical volume, compliance and resistance to flow.
   
c. The ventricular end-diastolic volume (stroke volume and cardiac output) is very sensitive to small changes in the cardiac filling pressure (blood return to the vena cava preload).
   
d. Cardiac filling pressure is a crucial factor that determines hemodynamic stability.
   
e. The intrathoracic venous compartment responds pathologically to pressure and flow changes, hypoxemia, toxins and emboli; causing endothelial dysfunction, decreased diameter to the lumen of the resistance vessels and the pulmonary arteries leading to pulmonary hypertension. (80)
   
f. Signs and symptoms of pulmonary hypertension include hypoxia, fatigue, shortness of breath, reduced cardiac output, syncope, lower extremity edema, hepatic congestion, ascites, acidosis, renal failure, malnutrition, hypotension and confusion. (80)
   
g. Presence of systolic murmur may suggest ventricular septal rupture or acute mitral regurgitation.
   
h. **Intervention considerations for peripheral compartment dysfunction:**
   
   - Focus will be on interventions which promote blood return from the peripheral veins (preload) to fill the heart prior to each beat, as a result will increase stroke volume and cardiac output. (64; 75)
6. **Intervention considerations for intrathoracic central venous dysfunction:**
   - Acute management of dysfunction is focused on the underlying mechanisms.

j. **Changes for geriatric population:** (60; 79)
   - higher BP caused by decreased arterial elasticity
   - increasing peripheral vascular resistance and cardiac workload
   - baroreceptors in aorta and carotid arches become less sensitive to pressure changes in arterial system and cardiac muscles
   - in symptomatic hypotension, chemoreceptors respond less to beta-adrenergic stimulation
   - decreased ability of cardiovascular system to vasoconstrict; instead shunting blood from peripheral arteries to major organs
   - progressive stiffening of myocardium and heart valves; leading to decreased efficiency
   - vascular changes to heart, kidneys and pituitary gland
   - increase in vessel wall rigidity and narrowing arterial lumens necessitating greater pump force
   - cardiac output and stroke volume decrease as the result of decreased conduction and blood flow
   - **Note:** Early placement of pulmonary artery catheter and aggressive management of hemodynamic parameters improve surgical outcomes in geriatric patients.

k. **Heart Failure:** cardiac output is insufficient to meet the metabolic needs of the body, possibly related to a large area of the left ventricle being affected.
   - **Cardiogenic shock:** caused by a decreased cardiac output/cardiac index. O2 and other nutrients become unavailable to the cells of the body.
   - **Dysrhythmias:** ischemia causes electrical instability of myocardium. **Ventricular fibrillation:** most common cause of death within first hour of onset of angina regardless of location of myocardial infarction.

4. **Pericarditis leading to Pericardial Effusion:**
   a. **Pericarditis:** Inflammation of the pericardial sac (a potential space holding up to 20 mL fluid with electrolyte and protein profiles similar to plasma). Autoimmune inflammatory response to myocardial infarction may cause an increase in fluid within the sac (2 to 5 percent of patients). Occurs as early as two days postmyocardial infarction and up to 12 weeks postmyocardial infarction. Up to 80 to 120 mL of additional fluid can accumulate in the pericardium before symptoms of pericardial pressure appear. (85)
   b. **Pericardial effusion:** presence of an abnormal amount and/or character of fluid in the pericardial sac with both infectious and noninfectious origin (e.g., anterior myocardial infarction). (83)

5. **Cardiovascular Structural Defects:** related to size and area of infarction. (84)
   a. **Ventricular aneurysm:** noncontractile, thinned left ventricular wall caused by an acute transmural infarction. Most common with acute left anterior descending (LAD) occlusion with wide area of infarcted myocardium.
   b. **Ventricular septal rupture:** postinfarction rupture of the ventricular septal wall that connects the left and right ventricle. Carries a very high mortality rate. Afterload reduction is necessary via IABP and vasodilators.
   c. **Papillary muscle rupture:** muscles that support the mitral valve infarct and rupture, causing ineffective valve closure and back-pressure of blood into the left atrium during ventricular systole.

6. **Mural Thrombosis leading to Embolism:** A clot in the cardiovascular system formed during life from constituents of blood; it may be occlusive or attached to the vessel or heart wall without obstructing the lumen (mural thrombus). (66) An embolus (plural “emboli”) is a plug, composed of a detached thrombus or vegetation, mass of bacteria or other foreign body, occluding a vessel. Thromboembolic complications occur secondary to increased blood viscosity, increased fibrinogen and bedrest. Mural thrombosis occurs more frequently with large, transmural, anterior myocardial infarctions. Primary concern is systemic embolism, which can lead to CVA/brain attack, peripheral embolism and pulmonary embolism.
   a. **DVT/PE:** Immobility leads to venous stasis thereby facilitating coagulation cascade in large veins. DVT causes less inflammation than thrombophlebitis; lack of inflammation increases risk that portions of clot may break off and travel through the vascular system as VTE. (7; 12) DVT may develop as late as 7 to 10 days postoperatively in vessels of pelvis and lower extremities. VTE prophylaxis should begin for surgical cases between 24 hours prior to surgical incision and 24 hours after surgery end time. (52) For nonambulatory stroke patients, DVT prophylaxis should begin by the end of hospital day two. (50)
   b. **Cholesterol embolism:** rupture of atherosclerotic plaque releases cholesterol crystals into bloodstream; crystals lodge in arterioles producing inflammation and thrombus formation which leads to fibrosis. (46)
   c. **Risks of mural thrombosis:**
      - **VTE:** more common in Blacks than Caucasians/Asians. Risk factors include: history of past VTE, pregnancy, postpartum or current estrogen use, recent trauma/surgery, immobilization, presence of cancer, airline flight longer than eight hours, obesity, fractures of hip/pelvis/lower extremity, central venous catheter, diabetes, hyperlipidemia, hyperuricemia, cardiovascular disease (e.g., hypertension, varicose veins, atrial fibrillation, angina, myocardial infarction, valvular disease), pulmonary hypertension and history of ischemic stroke. (3; 8; 14; 17; 67)
      - **VTE prophylaxis:** indicated for vascular, urologic, cardiac, orthopaedic, intracranial, neurologic or multiple trauma surgeries. General Surgery procedures are graded (low, moderate, high or very high risk) based on patient age/risk factors/duration of surgery.
      - **Cholesterol embolism:** often triggered by vascular radiographic/surgical procedures; incidence 25 to 77 percent in high-risk populations; common sources of plaque include abdominal aorta, carotid, iliac, femoral arteries. (46)

7. **Pain:**
   a. **Acute pain:** is a symptom versus a diagnosis. Pain is subjective. Acute pain is usually related to an injury, trauma or surgery, thus is protective, acting as an early warning sign that tissue damage is about to occur or is occurring. The patient expects to recover after the injury or surgical wound heals. (19; 26; 42)
b. **Chronic or persistent pain**: pain that lasts beyond the healing process and has outlived its warning role. It becomes persistent and debilitating, lasting for more than six months. It can exist when there is no evidence of tissue damage (e.g., phantom pain). It may be intermittent or continuous, poorly differentiated and may or may not be associated with an injury or chronic disease. Diagnostic tests may not necessarily reveal a specific source of the pain. Patient may show few or no outward signs of pain as they have learned to tolerate the pain for so long. (18; 26; 42)

c. **Acute-on-chronic pain**: person suffering from chronic pain (e.g., cancer or neuropathy) who is admitted to an acute care setting for surgery or injury. Both types of pain must be treated. (18)

d. **Types of Pain (can occur independently or combined):**
   - **Nociceptive (somatic)**: results from injury of the skin/mucosa, muscle, bone, tendons, ligaments and joints, arteries (e.g., superficial lacerations/burns, intramuscular injections, IV access, otitis media, stomatitis, extensive abrasions). Responds best to acetaminophen, cold packs, corticosteroids, localized anesthetic (e.g., topical or infiltrate), NSAIDs, opioids and tactile stimulation. (18; 19; 42)
   - **Nociceptive (visceral)**: results from stimulation of nociceptors in internal organs (e.g., stomach, kidney, bladder, gallbladder, pancreas, intestines). Ischemia/necrosis, inflammation, ligamentous stretch, smooth muscle spasm, and distension of a hollow organ capsule all contribute to visceral pain (e.g., colic, sickle cell, appendicitis, kidney stone, postoperative abdominal surgery). Responds best to corticosteroids, intraspinal local anesthetic agents, NSAIDs (IV route Ketorolac for NPO patients) and opioids (via any route). (18; 19; 42)
   - **Neuropathic**: caused by damage to a nerve, either centrally or peripherally. Pressure on tissues surrounding the neural tissues and abnormal processing of impulses also causes neuropathic pain [e.g., trigeminal, avulsion neuralgia, post-traumatic neuralgia, peripheral neuropathy (diabetes, human immunodeficiency virus), limb amputation, herpetic neuralgia]. This type of pain provides no protective benefit and precipitates ongoing suffering. Responds best to anticonvulsants (e.g., gabapentin), corticosteroids, neural blockade, NSAIDs, opioids (via any route) and tricyclic antidepressants. (18; 19; 26)

e. **Pain assessment for specialty groups:**
   - **Geriatric:**
     - **Normal cognitive function**: Older patients do not necessarily use the word "pain." Instead, ask which term they relate best to [e.g., ache, hurt, discomfort; or just ask the intensity of their pain (none, some, severe)]. Ask which type of scale works best for them [e.g., visual analogue, Numeric Rating Scale (NRS), Faces Pain Scale-Revised (FPS-R, no smiles or tears), Verbal Descriptor Scale (VDS) or revised verbal descriptor – includes visual pain thermometer]. Note that decreased hearing/vision may hamper verbal/visual communication as well as visual tools. (34; 44; 56; 63)
     - **Mild dementia**: If mild dementia is suspected, patient should first receive a cognitive evaluation that includes the Mini Mental State Examination (MMSE). Appropriate scales include verbal descriptor scale, pain thermometer or FPS-R (patient may have difficulty differentiating between pain or mood). (44)
     - **Advanced dementia**: assessment traditionally has moved from self-report to clinician observed scale. The "Pain Assessment in Advanced Dementia" (PAINAD) scale measures the following: breathing independent of vocalization, negative vocalization, facial expression, body language and consolability. It is rated on a 0 to 2 scale, with a total score range of 10. (35) Use of the Verbal Rating Scale (VRS) and Faces Pain Scale (FPS) have been used with some success in patients with severe dementia. (70)
   - **Pharmacology**: renal and liver function studies, as well as serum protein levels should be measured in geriatric patients prior to starting certain medications, especially NSAIDs. (44; 76)
   - **Nonverbal**: (e.g. advanced dementia, mechanically ventilated and/or sedated, unconscious): (1; 45; 68; 71; 77)
     - More than one measure may be needed to adequately detect the pain and evaluate outcome of interventions. Recommended hierarchy of assessment steps include:
       - Self-report. May be hampered by level of consciousness, artificial airway, medication(s), delirium.
       - Search for potential causes of pain/discomfort (e.g., existing medical condition, surgical-medical procedures, traumatic injuries, blood draws and routine care).
       - Observe patient behaviors. **Note**: behavioral scales are not appropriate for pharmacologically paralyzed patients, or those who are flaccid and cannot respond behaviorally to pain. In this case, assume pain is present and treat appropriately with analgesics prior to procedures thought to be painful (including turning).
       - Surrogate (family members, caregivers) report of pain and behavior/activity changes.
       - Attempt analgesic trial. If behaviors do not improve with analgesia, consider addition of sedatives and/or antipsychotic medications.
   - **Sample pain tools for assessment in nonverbal patients include**: Assessment of Discomfort in Dementia Protocol by Kovach et al (ADD), Checklist of Nonverbal Pain Indicators by Feldt, Pain Assessment in Advanced Dementia Scale by Lane et al (PAINAD), Behavioral Pain Scale by Payen (BPS). (45)

8. **Situational Response:**
   a. **Definition**: A phase in one’s life during which normal ways of dealing with the world are suddenly interrupted due to a medical event/illness/injury. (29)
   b. **Risk factors**:
     - Adjustments/reactions to a medical event/illness/injury, under any circumstances, are necessary and normal. A history of chronic mental health issues, substance use/abuse, poor coping abilities/strategies, past experience of trauma/abuse (both personal and professional), lack of support and low self-esteem contribute to an inability to deal with a medical event/illness/injury successfully. (53)
     - Although distress related to the experience of the illness/injury abates over time, the psychological impact of the distress lingers. (58)
1. Significant laboratory values/Diagnostic testing: (6; 33; 84)
   a. ECG (electrocardiogram): most important diagnostic tool for angina. An ECG shows changes both during symptoms and in response to treatment. An ECG may also demonstrate pre-existing structural or ischemic heart disease.
   b. Laboratory Values:
      - Biomarkers ("cardiac enzymes"): released from heart muscle after myocardial injury. Necrosis causes increased permeability of cell membranes which releases enzymes into blood stream. Enzymes altered due to defibrillation, intramuscular injection, thrombolytic therapy, PTCA, arachidectomy, stent.
        - Troponin I: A protein found in striated muscle and is specific to myocardial muscle necrosis. Preferred biomarker to diagnose myocardial necrosis. Rises within 3 to 6 hours and peaks within 12 to 16 hours, remains elevated for 14 days.
        - Troponin T: release kinetics similar to Troponin I. Remains elevated for 14 days. False-positive results may occur in patients with renal failure. Minor elevations in Troponin T also identify patients at risk for subsequent cardiac events.
        - C-reactive protein (CRP): a marker of acute inflammation. Patients without biochemical evidence of myocardial necrosis but elevated CRP level are at increased risk of an adverse event.
        - Erythrocyte sedimentation rate (ESR): rises above reference range values within three days, may remain elevated for weeks.
        - Myoglobin: a nonspecific muscle protein released with any type of muscle damage. Levels rise within 1 to 3 hours and peak at 9 to 12 hours. May be useful for early detection of myocardial infarction when performed with other studies.
        - CK-MB: rises within 4 to 6 hours and peaks in 16 to 30 hours. The CK-MB isoenzyme is specific for cardiac injury.
        - LDH: rises within 24 hours, peaks 3 to 6 days; increased with myocardial infarction, liver disease, lung disease and hematologic conditions.
   c. Basic metabolic panel, including electrolytes, BUN, Cr, magnesium, CBC:
      - Hypokalemia: ECG change: increase in PVC’s, "U"-wave, depressed ST-segment, flattened T-wave; potentiates digoxin toxicity.
      - Hyperkalemia: ECG change: tented T-wave, flattened P-wave, widened QRS leading to asystole
      - Hypocalcemia: potentiates digoxin toxicity
      - Hypomagnesemia: ventricular dysrhythmias; potentiates digoxin toxicity
      - Hypermagnesemia: bradycardia; prolonged P-wave; widened QRS.
      - Creatinine levels must be considered prior to initiating ACEI (51)
      - CBC to determine if anemia was the precipitant
   d. Imaging Studies: (6; 33)
      - CXR: provides information on alternative diagnoses (e.g., pneumonia, thoracic aneurysm) or demonstrates complications of ischemia (e.g. pulmonary edema).
      - Echocardiogram: detects wall motion abnormalities due to ischemia or precipitants (e.g. ventricular hypertrophy, valvular disease).
      - Radionuclide myocardial perfusion imaging:
         - Dual-source 64-slice CT scanners: visualize both the lumen of the coronary artery and plaque within the artery. May also be used with IV contrast to determine if a stent or graft is open or closed.
         - Technetium-99m (99mTc) tetrofosmin single-photon emission computed tomography (SPECT): may be used in the Emergency Department to exclude high-risk patients among those with chest pain.
         - Resting cardiac MRI: detects high fraction of patients with ACS (including enzyme-negative unstable angina), wall thinning, scar, delayed enhancement (e.g., infarction) and wall motion abnormalities (e.g., ischemia).
      - Pharmacologic stress echocardiogram: evaluates effect of increasing myocardial O2 demand by infusing inotropic drug (e.g. dobutamine) on blood supply to myocardium. Pharmacologic stress echocardiogram takes the place of exercise stress testing for patients unable to perform vigorous exercise.
   e. Graded exercise test (GXT): evaluates the effect of physical activity (increased myocardial O2 demand) on the blood supply to myocardium. May be combined with Technetium (99mTc) or Cardiolite if resting ECG has nonspecific ST-segment/T wave changes or if patient has preexisting bundle branch block.

"What happens when my body breaks down happens not just to that body but also to my life, which is lived in that body. When the body breaks down, so does the life. Even when medicine can fix the body, that doesn’t always put the life back together again."— Arthur Frank (58)

Psychological defenses allow us to cope with our day-to-day life. Increased stress leads to less effective psychological defenses, therefore, our typically well-managed emotions (e.g., anger, sadness, anxiety, frustration) become less well-managed leading to emotional instability, interpersonal conflict and diminished social/occupational functioning in many persons. (23)

The family unit is a system based on interdependency between members and patterns that provides structure and support. Illness and/or injury in one family member may disrupt these relationships and patterns. (20)

Individual differences need to be considered regarding response/reaction to medical event/illness/injury and support provided to patient/support system. Gender and/or cultural differences may influence response/reaction as well as attitudes and beliefs about support. (59)

Healthcare practitioners may hesitate to ask questions related to a patient/support system emotional/mental status, for fear of not feeling prepared to deal with the response. Failure to address this area of patient/support system health can result in suboptimal care. For this reason, it is essential to refer to someone on the healthcare team that is knowledgeable, competent and comfortable in assisting the patient/support system in this area. (20; 81)
2. **Management of ACS:** (5; 33; 84) The primary goals of therapy for patients with ACS are to reduce the amount of myocardial necrosis, prevent major adverse cardiac effect (MACE) and treat acute life-threatening complications [e.g., ventricular fibrillation/pulseless ventricular tachycardia, symptomatic bradycardia and unstable tachycardia]. The most common interventions include anti-ischemic therapy [e.g., O2 to decrease O2 demand, nitrates (Note: Nitrates should not be administered to patients who have received a phosphodiesterase inhibitor for erectile dysfunction within the last 24 hours (48 hours for tadalafl)] (6) pain control (morphine), beta blockers, fibrinolytic therapy, anticoagulant therapy, ACEIs, angiotensin receptor blockers (ARBs), calcium antagonists, HMG CoA reductase inhibitor (statin therapy), antidysrhythmic therapy, early invasive strategy (catheterization and coronary revascularization), pacemaker, and risk factor modification. See the American Heart Association (AHA) algorithm for ACS at [http://circ.ahajournals.org/cgi/content/short/112/24_suppl/IV-89](http://circ.ahajournals.org/cgi/content/short/112/24_suppl/IV-89). Although ARBs and ACEIs are indicated for ACS, they are contraindicated in the following conditions: chronic cough (a phenomenon caused in some patients by ACE inhibitors possibly related to increased bradykinin levels), angioedema (a rare but potentially serious side effect), hypotension, hyperkalemia and worsening renal function. (51)

3. **Behavior modification/lifestyle changes:** Cardiac rehabilitation program to educate patient/S.O. on low fat/low cholesterol diet, increased physical activity, stress reduction, smoking cessation, BP and blood sugar control. Specific patient populations will also require counseling for methamphetamine addiction. (84)

4. **Women's health and cardiovascular disease:** Heart disease is the leading cause of death for women both in the United States and in most of the industrialized world. More than 9,000 US women younger than 45 years sustain a myocardial infarction each year. Among survivors of myocardial infarction, 25 percent of men versus 38 percent of women die within a year after an initial myocardial infarction. Within six years after a myocardial infarction, 18 percent of men but 35 percent of women will have a recurrent infarction. Women have a cardioprotective effect of estrogen, but at 15 years postmenopause, the incidence of angina occurs with equal frequency in both sexes. (33) Women have more symptoms of angina than do men following percutaneous coronary intervention, with resultant limitations on their activities and quality of life. Women have a mortality rate from coronary artery bypass graft (CABG) surgery that is almost double that of their male peers, with women younger than age 50 years three times more likely to die at CABG surgery than comparably aged men. An excess of bleeding complications (unexplained pathogenesis) is reported for women who undergo all interventional procedures (e.g., coronary thrombolysis, percutaneous coronary interventions, CABG surgery), likely contributing to the adverse outcomes for women. (86) Women are more likely to delay seeking treatment for heart disease. Treatment-seeking delay is defined as the period between onset of symptoms and entry into the healthcare system. While transport times and therapy times (arrival at the Emergency Department until initiation of medical treatment) have improved, women still have delayed decision times (interval from onset of acute symptoms to the decision to seek care and act on their decision). (78) Cardiovascular education targeted specifically for women may be beneficial in improving delay times. Recommendations: The most publicized recommendations revolve around hormone replacement therapy (HRT). HRT is no longer recommended for cardiac health in women and has actually been shown to be harmful in some populations. All women should be encouraged to avoid tobacco, exercise regularly, maintain a healthy weight and eat a heart-healthy diet (including omega-3 fatty acids and folic acid). BP management is key and antihypertensives should be initiated to maintain BP less than 120/80 mmHg. Blood sugars should be kept at near-normal glycosolated hemoglobin levels. All women with cardiovascular disease should be evaluated for depression and treated appropriately. For complete risk scoring, refer to the Evidence-Based Guidelines for cardiovascular health in women at [http://circ.ahajournals.org/cgi/content/full/109/5/672](http://circ.ahajournals.org/cgi/content/full/109/5/672).

D. **PATIENT/FAMILY RESOURCES:**

1. **American Heart Association:** Heart Attack, Stroke & Cardiac Arrest Warning Signs
   [http://www.americanheart.org/presenter.jhtml?identifier=3053#Heart_Attack](http://www.americanheart.org/presenter.jhtml?identifier=3053#Heart_Attack)

2. Page 46 of ICSI guideline lists all resources available to families

E. **SAFETY CONSIDERATIONS AND INITIATIVES:**

1. **Acute Myocardial Infarction:**
   - **Centers for Medicare & Medicaid Services and the Joint Commission:**
   - **Safer Healthcare Now!**
   - **AACN:**

2. **Pain:**
   - **The Joint Commission:**
     - **2012 Hospital Accreditation Standards:**
       - Provision of Care:
         - **Standard PC.01.02.07:** The hospital assesses and manages the patient’s pain.
         - **Standard PC.02.03.01:** The hospital provides patient education and training based on each patient’s needs and abilities.
       - **Rights and Responsibilities of the Individual:** **Standard RI.01.01.01:** The hospital respects, protects and promotes patient rights.
   - **The Joint Commission International:**
     - **2011 Accreditation Standards for Hospitals, 4th ed. (2010):**
• Patient and Family Rights: **Standard PFR.2.4**: The organization supports the patient’s right to appropriate assessment and management of pain.

• Assessment of Patients: **Standard AOP.1.7**: All inpatients and outpatients are screened for pain and assessed when pain is present.

• Care of Patients: **Standard COP.6**: Patients are supported in managing pain effectively.

• Patient and Family Education: **Standard PFE.4**: Patient and family education includes the following topics, related to the patient’s care: the safe use of medications, the safe use of medical equipment, potential interactions between medications and food, nutritional guidance, pain management and rehabilitation techniques.

3. **Venous Thromboembolism**:
   
   The Joint Commission:
   

   • 2012 Hospital Accreditation Standards:
     
     National Patient Safety Goal:

     NPSG 3: Improve the safety of using medications.

     NPSG.03.05.01: Reduce the likelihood of patient harm associated with the use of anticoagulant therapy.

   National Quality Forum:


   • Safe Practice 29 Anticoagulation Therapy. Organizations should implement practices to prevent patient harm due to anticoagulant therapy.

   Institute for Healthcare Improvement:

   • Improvement Map: Venous Thromboembolism (VTE) Prevention and Treatment Process. Retrieved from: [http://www.ihi.org/imap/tool/#Process=5b9bfd5a-1e17-433b-a9d4-602fafef73c8](http://www.ihi.org/imap/tool/#Process=5b9bfd5a-1e17-433b-a9d4-602fafef73c8)

   Safer Healthcare Now!:


   U.S. Department of Health and Human Services:


   AACN:


F. **Refer to Heart Failure, Cardiac Catheterization with/without Percutaneous Coronary Intervention (PCI), Cardiac Rhythm Management Device (CRMD) [Includes Pacemaker, Implantable Cardioverter Defibrillator (ICD) and Cardiac Resynchronization Therapy Device (CRT/CRT-D)] and/or Cardiac Surgery Clinical Practice Guidelines that may apply to the medical diagnosis, treatment, procedure.**
References


Safety References:


