To Be Completed Before the Simulation

** Blue boxes should be completed using textbook information. What do you expect to find? This information should be collected before you start the ATI simulation.

Medical Diagnosis/ Disease: Myocardial infarction

NCLEX IV *(8)*: Physiological Integrity/Physiological Adaptation

NCLEX IV *(7)*: Reduction of Risk

Anatomy and Physiology
Normal Structures

Page attached for A+P

Blockage of the R coronary artery results in an inferior or posterior wall MI. Blockage of the L coronary artery results in an anterior wall MI. Damage can occur in more than one location.
Degree of collateral circulation influences the severity of the MI. Long history of CAD -> develop good collateral circulation to provide area surrounding the infarction site with adequate blood supply.

Pathophysiology of Disease

Abrupt stoppage of blood flow through a coronary artery with a thrombus caused by plaque rupture and thrombus formation. Causing irreversible myocardial cell death in the heart muscle beyond the blockage. Serum cardiac biomarkers are released into the blood. Imbalance exists between myocardial oxygen supply and demand. Most Mis occur in the setting of preexisting CAD.

STEMI: caused by an occlusive thrombus resulting in ST-elevation in the ECG. Ischemia starts transmural. Emergency. To limit the infarct size, the artery must be opened within 90 minutes of presentation to restore blood and O2 to heart muscle.
NSTEMI: nonocclusive thrombus, doesn't cause ST segment elevation on ECG. Need cath within 12-72 hours, thrombolytic therapy not indicated because the thrombus isn't occlusive. Can have hypokinesis or akinesis in the necrotic area. The earliest tissue to become ischemic is the sub-endocardium (innermost layer of tissue in heart muscle).
If ischemia persists, it takes 4-6 hours for entire thickness if the heart muscle to necrose. If thrombus not completing blocking artery, the time may be as long as 12 hours to completely necrose.
Hypoxia in 10 seconds to heart muscle. Can withstand for 20 minutes before cell death. Mostly affects the left ventricle.
Stimulates nerves to send pain messages throughout thoracic -> chest pain.

Anticipated Diagnostics
Labs

Serum cardiac biomarkers- cardiac specific troponin, CK-MB
CBC, BMP, coagulation studies
Myoglobin, ESR
ABG
Lactic acid

Additional Diagnostics

ECG
Cardiac Catheterization
Continuous cardiac monitoring
CXR
Echo

NCLEX II *(3)*: Health Promotion and Maintenance

NCLEX IV *(7)*: Reduction of Risk

Contributing Risk Factors

CAD
Tobacco use
Hypertension
Hyperlipidemia
Age: men over 45, women over 55
Obesity
Diabetes
Family history of MI
Stress
Lack of physical exercise, unhealthy diet

Signs and Symptoms

Severe chest pain not relieved by rest, position change, or nitrate admin.
Heavy, pressure, tight, burning, constricted, or crushing feeling.
Pain radiating to neck, lower jaw, arms, back. Feelings of indigestion.
Discomfort, nausea, weakness, SOB.
Women: atypical discomfort, SOB, fatigue. Diabetes- asymptomatic
Elderly: change in mentation, SOB, pulmonary edema, dizzy, dysrhythmias. Fever – 38C
Initial phase: diaphoresis, increased HR + BP, clammy and cool to touch
Later phase, BP may drop -> decreased UO. Crackles, JVD
S3 and S4, loud holosystolic murmur

Possible Therapeutic Procedures

Non-surgical

Thrombolytic therapy

Surgical

Cardiac catheterization/PCI
Bypass surgery
Atherectomy

Prevention of Complications

(What are some potential complications associated with this disease process)
Dysrhythmias
Heart failure
Cardiogenic shock
Papillary muscle dysfunction
Left ventricular aneurysm
Pericarditis
Dressler syndrome – high dose asa

NCLEX IV *(6)*: Pharmacological and Parenteral Therapies

NCLEX IV *(5)*: Basic Care and Comfort

NCLEX III *(4)*: Psychosocial/Holistic Care Needs

Anticipated Medication Management

MONA: morphine, oxygen, IV nitro, asa
Thrombolytics
Heparin
Glycoprotein IIb/IIIa inhibitors
Antidysrhythmic
Clopidogrel or ticagrelor
Atorvastatin
Beta blockers, ace inhibitors. CCB
Stool softeners

Non-Pharmacologic Care Measures

Semi/high-fowlers
2 IV catheters
Emotional support and reassurance
Rest and comfort, bedrest
Quite environment
Relaxation techniques – music therapy, guided imagery
Bed rest
Cardiac rehabilitation
Dash diet

What stressors might a patient with this diagnosis be experiencing?

Pain
Lifelong medication therapy
Scheduled surgery
Time off of work

NCLEX I *(1)*: Safe and Effective Care Environment

List 3 potential teaching topics/areas	**Multidisciplinary Team Involvement** (Which other disciplines do you expect to share in the care of this patient)
•Taking aspirin should be lifelong. •Rest is important after a MI because the body requires less work from the heart when less active. • Exercise, diet, smoking cessation for discharge teaching	PCP Cardiologist Cath lab nurses Cardiac rehab staff

Anticipated Patient Problems, Goals, & Interventions Based on Medical Diagnosis

** This worksheet should be completed before you begin the ATI simulation.

Problem #1: Decreased Cardiac Output

Patient Goals:

1. The client will have a normal blood pressure (120/80) and a normal urinary output (30ml/hour) by the end of my care.
2. The client will have no longer have ST-segment elevation by the end of my care.

Assessments:

- Assess BP, HR, RR, and O2 q15minutes. Auscultate heart sounds for normal rhythm and presence of S3, S4, and murmurs q4hours. Assess lung sounds q4hours. Assess urinary output q1hour. Assess ECG for dysrhythmias PRN. Assess pulses q4hours. Assess lung sounds q4hours. Assess cardiac biomarkers PRN. Monitor electrolytes PRN.

Interventions (In priority order):

1. Administer nitroglycerin as ordered during my time of care.
2. Assist client to the cath lab within ninety minutes of arrival during my time of care.
3. Administer thrombolytic therapy as ordered if no cath lab during my time of care.
4. Administer morphine as ordered during my time of care.
5. Administer and titrate oxygen to ensure oxygen saturation stays above 93% during my time of care.
6. Administer aspirin as ordered during my time of care.

Problem #2: Acute pain: angina

Patient Goals:

1. The client will report no chest pain (0/10) during my time of care.
2. The client will have normal vital signs (120/80 BP, 60-100HR, 12-20RR) during my time of care.

Assessments:

- Assess PQRST of pain q15minutes. Assess BP, HR, O2 and RR q15minutes. Assess for previous history of angina including family history PRN.

Interventions (In priority order):

1. Administer nitroglycerin as ordered during my time of care.

2. Administer morphine as ordered during my time of care.
3. Administer oxygen and titrate as needed to ensure oxygen remains above 93% during my time of care.
4. Provide a calm and quiet environment during my time of care.
5. Administer metoprolol as ordered during my time of care.
6. Encourage bedrest with semi-fowlers PRN during my time of care.

At this time, complete assigned ATI Real Life Simulation

Actual Patient Problems & Goals

** The following should be completed after the ATI simulation.

Problem #1: Decreased cardiac output

Patient Goals:

1. R.D. will have a normal blood pressure (120/80) and a normal urinary output (30ml/hour) by the end of my care. Met x Unmet ☐
2. R.D. will have no longer have ST-segment elevation by the end of my care. Met x Unmet ☐

Problem #2: Impaired gas exchange

Patient Goals:

1. R.D. will demonstrate adequate oxygenation as evidenced by normal oxygen saturation (>93%) after epinephrine administration during my time of care. Met x Unmet ☐
2. R.D. will have clear breath sounds and will have no shortness of breath after epinephrine administration during my time of care. Met x Unmet ☐

SOAP Notes Based on Priority Problems

Priority Patient Problem #1: Decreased cardiac output

Subjective: *This section explains the client symptoms. Include a narrative of the patient's complaints/concerns and/or information obtained from secondary sources.*	Chief Complaint: chest tightness that isn't relieved with nitroglycerin tablets PMH: HTN, CAD with angina, asthma, quit smoking 1 month ago and occasionally chews tobacco. Allergies: pcn, peanuts, sulfa Current Medications: Nitroglycerin sublingual tablets, lisinopril 10mg PO daily, albuterol inhaler 2 puffs PRN, amlodipine 5mg PO daily, aspirin 5mg PO daily, clopidogrel 75mg PO daily, morphine 2mg IV push q4hours for moderate pain. Naloxone 0.2mg IV bolus PRN q2-3minutes if RR <10/min or if oversedated.
Objective: *This section is your clinical observations. Include, pertinent vital signs, pertinent labs and diagnostics related to priority problem.*	**Vital Signs:** 12/7 1722: 37.2, 94 HR, 26 RR, 96/56, 94% on 4L NC. Pain 8/10. UO 600ML. 12/7 1735: 104HR, 102/68, 22RR, 97% on 4L NC. 12/7 2100 postop: 36, 96HR, 14RR, 112/66, 98% 2L NC. Arterial BP 114/70. CVP 10.2, no pain. 12/8 1930: arterial BP 88/54, MAP 54, 96HR 12/8 2000: 64 HR, 8RR, 64/42 BP, 92% O2 on 4L/min NC 12/8 2040: 64HR, 14RR, 96/56. 12/9 1900: 68HR, 12RR, 124/72, 98% RA, sinus rhythm **Labs:** 12/7 1745: RBC 5.2, Hgb 15.9, Hct 54%, carbon dioxide 24 Cardiac enzymes: Ck 0, troponin T 0.2, troponin I 0.06, lactic acid 0.6 12/7 2230: Hgb 14.8, potassium 3.2, aPTT 38 seconds 12/8 0600: RBC 4.8, Hgb 14, potassium 3.2. 0800 potassium 3.4 Cardiac enzymes: troponin T 2145 0.4, 0100 0.6, 0400 0.8, 0800 0.6 Troponin I 2145 0.07, 0100 0.08, 0400 0.09, 0800 0.07 **Diagnostics:** Chest x-ray: no fluid or pneumothorax. Heart has no enlarged heart shadows. Aorta and aortic arch has calcifications and appears intact with no dilation of the artery. Telemetry 12/17 2100: normal sinus rhythm with PVCs Telemetry 12/8 1900: normal sinus rhythm with occasional PVCs
Assessment: *Focused assessment on your priority problem.*	Emergency services report 12/07: chest tightness that isn't relieved with nitroglycerin tablets. GCS 15. Alert and oriented x3, responds to verbal stimuli. Skin ashen. Oxygen given at 4 L/min NC. Last oral intake was lunch at noon. Medications: nitroglycerin sublingual tablets, lisinopril, albuterol inhaler. Treatment: taken nitroglycerin 0.4mg q5minutes x3 – last dose at 1710, asa 325mg orally at 1715, oxygen started. VS enroute: 1715 100/82, 96HR, 28RR 92%, prolonged p wave, PVCs, ST elevation Response to treatment: continues to report chest tightness and pain. Arrived at 1720. Progress note 12/7 1740: Client has ST-segment elevation MI. Continues to report chest pain with pain 8/10. A+O. Urine output at least 30ml/hour post-cath. 12/7 2205: right groin puncture site has saturated dressing with bright red drainage and 3-inch hematoma. Dressing removed and pressure held above puncture site. 2230: right groin bleeding stopped and pressure dressing applied. Hematoma 6 inches. 12/8 1900: indwelling catheter intact and draining clear/yellow urine. Right groin puncture site pressure dressing clean and dry, no hematoma noted. 12/8 2040: BP maintained with norepinephrine infusion
Plan ***Based on priority problem only** *Include what your plan is for the client. What treatments or medications are needed. You can include procedures, consults, labs/diagnostics, etc. What nursing interventions are being performed?*	**Plan:** 12/7 orders: Titrate oxygen delivery to maintain O2 saturation at 96% or greater per NC or nonrebreather face mask. Telemetry monitoring continuously. BP/pulse/RR every 5 minutes. 12-lead ECG STAT. 0.9% sodium chloride 1000ml, admin 300ml bolus and then 100ml/hour. Troponin level, cardiac enzymes, lactic acid, CMP, aPTT, prothrombin, INR, CXR, MRI, UA STAT. 1735: informed consent witnessed and signed. Ready for transfer to cath lab. 1745: Administered Morphine 2mg IV q10 minutes for moderate to severe chest pain. Cath lab transfer report: Central venous cath placed. Arterial line. 2L NC. Vital signs stable. No bleeding or hematoma present. Cardiologist progress note: PTCA with stent placement in the left anterior descending coronary artery. No complications. 12/7 2100 orders post-cath: ECG STAT on arrival to ICU, troponin T now and then in 3 and 6 hours. Continuous telemetry monitoring and arterial blood pressure monitoring. Vital signs

Teaching/Resources: Dr. Patterson educated on STEMI and the treatment for it. Progress note 12/7 1740: discussed with client the need + risk for invasive treatment for left cardiac cath with possible percutaneous transluminal coronary angioplasty with balloon dilation and stent placement through right femoral artery. Educated on postop assessments after heart catheterization procedure. Educated on splinting incision when coughing and importance of staying supine post-op. Educated about his risk for heart disease (exercise, diet) Educated on how to decrease sodium in his diet.	every 15 minutes for 1 hour, every 30 minutes for 1 hour, then every hour. Elevated HOB 10 degrees for two hours and ab lib there after. Arterial line connect to transducer and continue monitoring blood pressure. Indwelling urinary catheter hourly output, notify provider if less than 0.5-1mL/kg/hr. Bed rest and keep right leg straight for 12 hours. Notify provider if having chest pains or tightness. 12/7 2300: Administered potassium 20 mEq PO. 12/8 1940: NS 1000ml at 250ml/hour, dobutamine 250ml initial rate 2.5 mcg/kg/min titrate as needed. Monitor BP, cardiac rate, and rhythm per protocol 12/8 1950: Dobutamine continuous bolus infusing on IV pump with IV fluid maintenance. 12/8 2000: arousing easily, encouraged deep breathing 12/8 2000: Norepinephrine 4mg in dextrose 5% in water 1000ml started at 0.5mcg/min to improve BP. 12/8 1200: wean and discontinue the dobutamine and epinephrine drip. Start cardiac rehab tomorrow morning.

Priority Patient Problem #2: Impaired gas exchange

Subjective: *This section explains the client symptoms. Include a narrative of the patient's complaints/concerns and/or information obtained from secondary sources.*	**Chief Complaint: client reports arms and chest are itching, an irritating cough and nasal congestion. Stated "My tongue swelled after eating shellfish."** Current medications: diphenhydramine 25mg IV bolus PRN q4hours for itching or restlessness. Epinephrine 0.3mg IM STAT. Allergies: shellfish and contrast dye added
Objective: *This section is your clinical observations. Include vital signs, pertinent labs and diagnostics related to priority problem.*	**Vital Signs:** 12/7 2115: 112HR, 32RR, 94% on 2L NC, 148/94, tachycardia with PVCs. 2120 116HR, 32RR, 87% 15L/min via non-rebreather mask, 155/98. 2135 124HR, 36RR, 92% O2 on 15L/min, 162/98, pain 9/10. 2155 98HR, 18RR, 100% O2, 128/78, normal sinus rhythm with PVCs. 2205: 88HR, 14RR, 100% 3L/min NC, 110/72 **Labs: N/A** **Diagnostics: N/A**
Assessment: *Focused assessment on your priority problem.*	12/7 2100: denies shortness of breath or pain. Reports irritating cough and nasal congestion. Showing signs of dyspnea, wheezing. Progress note: experiencing moderate systemic reaction to contrast dye used for heart catheterization. 12/7 2115: shortness of breath, wheezing, intermittent stridor, HOB remains at 10 degrees. Appears ashen and nailbeds are dusky. Replaced NC with non-rebreather mask at 15L/min 100% O2 for a drop in oxygen to 87%. Added shellfish and contrast dye to allergies. 12/7 2200: stable after epinephrine administered.
Plan ***Based on priority problem only** *Include what your plan is for the client. What treatments or medications are needed. You can include procedures, consults, labs/diagnostics, etc. What nursing interventions are being performed?*	**Plan:** Administered 0.3mg IM epinephrine STAT. Cardiologist note: R.D. experienced an anaphylactic reaction to the contrast dye administered during the heart catheterization after arriving in the ICU. One dose of epinephrine 0.3mg was administered with resolution of the crisis.

Reflection:

1. Go back to your Preconference Template:
    a. Indicate (circle, star, highlight, etc.) the components of your preconference template that you saw applied to the care of this virtual patient.

2. What was your biggest "take-away" from participating in the care of this patient? How did this impact your nursing practice?

My biggest take-away from participating in the care of this patient is how the plan of care can change very quickly depending on the patients condition. I thought it was just going to be post heart cath care but it also involved an anaphylactic reaction, bleeding from the puncture site, hypotension, and hypokalemia. It should me that we need to look at the entire picture instead of just focusing on the original problem because of how quickly things can change. This scenario impacted my nursing practice because it showed me how important it is to do a thorough assessment and history and physical. It showed me how it's important to try and connect the dots. For example, the patient mentioning that when he had shellfish before, his tongue had swelled. The nurse had to make the connection that a shellfish allergy is also a contrast dye allergy and the symptoms that he was presenting with was actually a reaction to the dye he had for his heart catheterization.

Time Allocation: 8 hours

Acute coronary syndrome: spectrum includes UA, NSTEMI, and STEMI. Caused by a decline of once stable atherosclerotic plaque leading to a thrombus. Partial occlusion by thrombus -> UA, NSTEMI. Total occlusion -> MI.

Causes: vasospasm – sudden constriction or narrowing of the coronary artery, spasms because the blood cannot flow through. Decreased oxygen supply – acute blood loss, anemia, low blood pressure. Increased demand for oxygen – rapid HR, thyrotoxicosis, ingestion of cocaine causing an increase in the demand of oxygen

After minutes, anaerobic metabolism produces lactic acid. Degree of collateral circulation influences severity of MI.

EKG: ST segment elevation -> injury. T wave inversion -> ischemia. Q wave -> necrosis (develops within a few hours to days)

Healing process:

- Body's response to cell death -> inflammatory process. Within 24 hours, leukocytes infiltrate the area. Proteolytic enzymes of the neutrophils and macrophages begin to remove necrotic tissue by the 4th day – necrotic muscle wall is thin
- Serum glucose levels are high following an MI. Catecholamine-mediated lipolysis and glycogenolysis occur which allow the increased plasma glucose and free fatty acids to be used by the O2 depleted myocardium for anaerobic metabolism.
- Within 1-2 days, the neutrophils and monocytes have cleared the necrotic debris from the injured area, the collagen matrix that will eventually form scar tissue is laid down.
- 10-14 days – new scar tissue is still weak. Heart muscle is vulnerable to increased stress. By 6 weeks, scar tissue has replaced necrotic tissue and the injured area is healed. The scarred area is less compliant.
- These changes can cause changes in the unaffected areas. The normal myocardium hypertrophies and dilates to try to compensate for the damaged muscle (ventricular remodeling). Remodeling can lead to heart failure. ACE inhibitors are given to limit this remodeling.

Unstable angina:

- Reduced blood flow in a coronary artery due to rupture of an atherosclerotic plaque but the artery is not completely occluded.
- Chest pain that is new in onset, occurs at rest, or occurs with increasing frequency. Pain usually lasts 10 minutes or more.
- Unpredictable and needs to be treated immediately.
- Previously diagnosed chronic stable angina would describe UA as: change in the pattern of angina, occurs with increasing frequency and is easily provoked my minimal exertion, during sleep, or at rest.
- Without previous diagnosis of angina would describe it as: chest pain that has progressed rapidly in the past hours, days, weeks often ending in pain at rest.
- ST depression and T wave inversion -> ischemic changes

Layers: endocardium (thin, innermost), myocardium (muscular), epicardium (outermost). Covered by a fibrotic sac -> pericardium. 2 layers of the pericardium: visceral (thin inner layer) and parietal (tough, fibrous outer layer). Pericardial fluid (10-15ml) lies between the layers to lubricate and prevent friction as heart contracts.

Left ventricle thicker than the right because it has to push through systemic pressure.

4 chambers:

- Right atrium: thin wall, low pressure, receives blood from the vena cava, outflow through the tricuspid valve
- Right ventricle: thin wall, low pressure, perceives blood from the right atrium, outflow through pulmonic valve to artery
- Left atrium: thicker wall, medium pressure, receives blood from the pulmonary veins, outflow through mitral valve
- Left ventricle: thick wall, high pressure, receives blood from left atrium, outflow through aortic valve to aorta

Atrioventricular valves -> tricuspid and bicuspid. Semilunar: pulmonic and aortic.

Papillary muscles close valves with every heart beat.

Functions: managing blood pressure, producing blood pressure, securing one-way blood flow, and transmitting blood.

Properties of heart muscle:

- Automaticity: pacemaker ability
- Conductivity: each cell has ability to conduct impulses to next cell
- Contractility: ability to contract (make each cell shorter or longer)
- Irritability: each cell has ability to contract on its own, to send out impulses to other cells without it first being stimulated from another source.

Major coronary arteries:

- Left coronary artery branches: left anterior descending + left circumflex. Supplies blood to left atrium, left ventricle, interventricular septum, and part of right ventricle.
- Right coronary artery branches: supplies blood to right atrium, right ventricle, and part of the posterior left ventricle. Includes the AV node + bundle of His.

Superior + inferior vena cava: receives deoxygenated blood from the body. Aorta returns oxygenated blood to the body.

Pulmonary arteries: carries deoxygenated blood to the lungs. Pulmonary veins: oxygenated blood from the lungs to the left atrium.

Conduction system:

- Specialized tissue creates + transports electrical impulse (action potential) -> depolarization -> heart muscle contraction
- SA node -> interatrial pathways -> atrial contraction -> AV node -> intermodal pathways -> bundle of His -> left and right bundle branches -> purkinje fibers -> ventricular contraction

Mechanical system:

- SV: amount of blood ejected with each heartbeat
- CO: amount of blood pumped by each ventricle in 1 minute. CO= SV x HR. Normally 4-8L/min
- Preload: volume of blood stretching the ventricles at the end of diastole
- Afterload: peripheral resistance against which left ventricle must pump

Autonomic nervous system:

- Sympathetic stimulation: increases HR, speed of impulse through AV node, and force of contractions
- Parasympathetic stimulation: slows HR, impulse conduction from SA to AV node, vague nerve
- Sympathetic stimulation of adrenergic receptors causes vasoconstriction

Baroreceptors (aortic arch, carotid sinus): sensitive to stretch or pressure in arterial system, stimulation sends message to vasomotor center in brainstem

Chemoreceptors (aortic + carotid bodies, medulla) increase CO_2 results in changes in RR + BP.