ATI Real Life Student Packet
N202 Advanced Concepts of Nursing
2024

Student Name: ___Catharine Cardellino_____

ATI Scenario: _____MI_____

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: ___MI_____

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

Anatomy and Physiology
Normal Structures

Blood flows into the right atrium moves into the right ventricle and is pushed to the lungs through the pulmonary arteries, after picking up oxygen the blood travels back through the pulmonary veins into the left atrium then the left ventricle and out of the aorta to the rest of the body. Blood flow into the two major coronary arteries occurs during diastole. The left coronary artery arises from the aorta → divides into left anterior descending artery and left circumflex artery → supply the left atrium, left ventricle, interventricular septum, part of right ventricle. The right coronary artery arises from the aorta, → supplies the right atrium, right ventricle, part of posterior wall of left ventricle. Most people the AV node and bundle of His receive blood supply from the right coronary artery.

The intraarterial septum creates the left/right atrium, the interventricular septum creates the ventricles. The mitral and tricuspid valves prevent the eversion of the leaflets into the atria during ventricular contraction, the pulmonic and aortic valves prevent blood from regurgitating in to the ventricles at the end of each ventricular contraction. There are also three layers 1. Endocardium (thin inner layer) 2. Myocardium (heart muscle) 3. Epicardium (outer layer.) The pericardium is a sac of a visceral layer and parietal layer. 10-15 ml of pericardial fluid lubricates the space between/prevents friction.

The conduction system creates/transports action potential. This impulse starts depolarization of heart cells → muscle contraction. Impulses

NCLEX IV *(7)*: Reduction of Risk

Pathophysiology of Disease

A myocardial infarction occurs due to an abrupt stoppage of blood flow through a coronary artery with a thrombus cause by platelet aggregation → irreversible cell death (necrosis) in the heart muscle beyond the blockage. Serum cardiac biomarkers are then released into the blood.

STEMI caused by an occlusive thrombus that results in an ST elevation in the ECG reading. Emergency! Artery must be opened within 90 minutes to restore blood and O2 to the heart muscle and limit the infarct size.

NSTEMI caused by nonocclusive thrombus and does not cause an ST elevation on the ECG.

The acute MI process evolves over time (hours to a few days.) The earliest tissue to become ischemic is the subendocardium (innermost layer). If ischemia persists → entire thickness of the heart muscle necrosis (4-6 hours). If the thrombus is nonocclusive, the time may be as long as 12 hours. Most MIs impact the LV (location is usually dependent on which coronary artery is blocked.

Manifestations include:
Pain in chest that may radiate to other body parts along the same nerve branch is not relieved by nitroglycerin.
During the initial phase, the ischemic heart cells release catecholamines (epi/norepi) which results in diaphoresis, increased HR/BP, peripheral vasoconstriction. Later the BP may drop due to decreased CO, which may lead to decreased renal perfusion (decrease UO). Crackles = LV

usually begins in the sinoatrial node → travel through interatrial paths to depolarize the atria (contraction.) The impulse travels from the atria to the AV node (internodal pathways) → the bundle of His→ left and right bundle branches. Action potential moves through walls of both ventricles via *Purkinje fibers.* This impulse travels within .12 seconds and triggers a synchronized right and left ventricular contraction → ejection of blood into the pulmonary and systemic circulations. Repolarization occurs when the contractile + conduction pathway cells regain their resting polarized condition. During ventricular contraction, there is absolute refractory period (heart muscle can't respond to any stimuli) then relative refractory period at early diastole (heart muscle may respond, but not good) Cardiac output: amount of blood pumped by each ventricle in one minute, SV x HR = CO. Stroke volume: amount of blood ejected from the ventricle with each heartbeat Normal CO is 4-8L/min Preload: volume of blood stretching the ventricles at the end of diastole Afterload: peripheral resistance against which the left ventricle must pump.		dysfunction. JVD, hepatic engorgement, peripheral edema = RV dysfunction. Nausea and vomiting may occur as reflex due to pain, or vasovagal reflexes initiated in the area of the infarcted heart. Fever can occur due to systemic inflammatory process caused by death of myocardial cells.

To Be Completed Before the Simulation

Anticipated Patient Problem: Decreased Cardiac Output

Goal 1: Patient will demonstrate adequate cardiac output as demonstrated by a blood pressure (around 120/70) and heart rate (60-100) within normal limits before the end of my shift

Relevant Assessments (Prework) What assessments pertain to your patient's problem? Include timeframes	**Multidisciplinary Team Intervention** (Prework) What will you do if your assessment is abnormal?
Assess for JVD upon arrival and after treatment during every interaction with patient	Report any JVD to the provider immediately
Assess the lungs for any crackle sounds or any shortness of breath upon arrival and one hour after interventions	Elevate the HOB to position the patient in a way that promotes breathing and comfort upon arrival and as needed
Assess the ECG for any changes in ST segment continuously	If ECG demonstrates STEMI, prepare patient for PCI immediately or fibrinolytic therapy if PCI is not an option
Assess vital signs upon arrival and maintain continuous monitoring	Administer nitroglycerin as ordered to decrease preload and afterload and increase myocardial O2 supply
Assess for edema upon arrival and an hour after every intervention	Apply compression stockings, SCDs, and/or abdominal binders upon arrival to decrease venous pooling as soon as possible, but after priority interventions have been completed
Assess peripheral pulses upon arrival and after every intervention	Apply oxygen upon arrival and maintain, titrating down as long as O2 sats maintain at or above 95 percent

Goal 2: Patient will demonstrate adequate perfusion as evidenced by a SpO2 of 95% or above on no more than 2L before the end of my shift

To Be Completed Before the Simulation

Anticipated Patient Problem: Acute Pain

Goal 1: Patient will report no chest pain (0/10) after treatment of MI with PCI or fibrinolytic therapy

Relevant Assessments (Prework) What assessments pertain to your patient's problem? Include timeframes	**Multidisciplinary Team Intervention** (Prework) What will you do if your assessment is abnormal?
Assess location and radiation of pain upon arrival (does it stay isolated to the chest? Does it travel to the jaw or arms? Is it located in the trapezius?)	Start IV line to prepare for administration of IV medications upon arrival
Assess if the patient took nitroglycerin or has a history of angina upon arrival	Administer nitroglycerin and titrate per policy/order
Assess if position changes such as sitting forward relieves the pain upon arrival to help rule out pericarditis	Position patient into fowlers upon arrival and maintain position as necessary
Assess rating of pain from 1-10 upon arrival and after every intervention	Administer IV morphine as ordered and reassess pain within 30 minutes
Assess what precipitating events may have triggered the angina upon arrival	Administer continuous nasal cannula oxygen upon arrival
Assess descriptive factors of pain such as sharp, crushing, burning, aching, stabbing etc upon arrival and after interventions	Maintain bedrest during MI and the days following the incident with a gradual increase in activity after treatment

Goal 2: Patient will feel a reduction in pain (at least 5/10) after receiving nitro and morphine

To Be Completed During the Simulation:

Actual Patient Problem: Decreased Cardiac Output

Clinical Reasoning: The infarction in the left anterior descending coronary artery prevented blood from being able to be adequately pumped out to the rest of the body

Goal: RD will demonstrate adequate cardiac output as demonstrated by a blood pressure (around 120/70) and heart rate (60-100) within normal limits before the end of my shift **Met?** At certain points he did meet this goal, during his state of shock though there were significant changes.

Goal: Patient will demonstrate adequate perfusion as evidenced by a SpO2 of 95% or above on no more than 2L before the end of my shift **Met**, his sats did drop during his state of shock, but were fixed with treatment and prior to transfer

Actual Patient Problem: **Acute Pain**

Clinical Reasoning: The infarction prevented oxygenation/perfusion to heart tissue causing ischemia resulting in acute chest pain

Goal: RD will have a decrease in pain from 8/10 to 6/10 after receiving nitroglycerin, morphine, and oxygen till he goes to the cath lab **Unmet**

Goal: RD will report and maintain a 0/10 pain level after his PCI procedure and all during my time of care **Met**

Additional Patient Problems:

Risk for bleeding

Deficient knowledge

Below will be your notes, add more lines as needed. **Relevant Assessments:** Indicate pertinent assessment findings. **Multidisciplinary Team Intervention:** What interventions were done in response to your abnormal assessments? **Reassessment/Evaluation:** What was your patient's response to the intervention?

Patient Problem	Time	Relevant Assessments	Time	Multidisciplinary Team Intervention	Time	Reassessment/ Evaluation
1, 2	12/06 1715	ED receives a call, EMT reports that RD, 54 year old male, started feeling chest pain at 1655 and took three doses of 0.4 mg nitroglycerin, 325 mg aspirin at 1715, peripheral IV access, RD appears uncomfortable as noted by facial grimacing and difficulty speaking in full sentences, reports feeling "sick to his	1720	Attached RD to 4L NC O2 and telemetry leads	1722	RD shares he was shoveling his driveway. Pain is 8/10, squeeze over heart. T 37.2, HR 104, RR 26, BP 96/56, O2 94% 4L NC

		stomach," has a history of CAD,				
1	1722	A&O x3, history of htn, cad, asthma, smoking (quit 1 month ago, chews tobacco	1722	Care team performed ECG	1725	ECG reveals an ST elevation, as well as a prolonged P wave, and PVCs
1,2	1730	RD remains uncomfortable, complaining of squeezing pain. Troponin T 0.2 ng/mL, Lactic Acid venous 0.6 mmol/L	1730	Provider provides education to RD and his wife about PCI treatment in the cath lab to treat his STEMI.	1730	RD and wife agree to treatment
1,2	1755	Cath lab gives report to ICU nurse about percutaneous transluminal coronary angioplasty in left ascending descending coronary artery. Central venous catheter, arterial line, indwelling urinary catheter, IV fluids IV pump, VS stable, receiving 2L NC, vascular closure devise used after the lines removed right femoral puncture site. RD laying supine, appears comfortable as evidenced by no facial grimacing	1930	ICU nurse provides education on importance of remaining flat with leg in straight position, education to press on puncture site dressing when needing to cough	1930	RD reports that his chest pain has resolved
1	2100	RD reports that he feels itchy over his arm and chest, that he "once ate shrimp and his tongue swelled" T 36, HR 96, RR 14, BP 112/66, O2 98% 2L, CVP 10, right femoral pressure dressing clean/dry,	2100	Nurse Carl contacted the provider and obtained an order for Diphenhydramine	2115	RD shares that while Nurse Carl was gone he started coughing, his nose feels stuffy, having difficulty breathing. HR 98, R 20, O2 96%, arterial BP 118/72, CVP 10, telemetry indicates RSR with PVCs,

		telemetry indicates regular sinus with PVCs,				
1	2115	Auscultated lungs, wheezing. HR 112, RR 32, arterial BP 148/94, tachycardia with PVCs,	2115	Administered 25 mg diphenhydramine IV bolus,	2120	87% on non rebreather face mask 15L/min, skin ashen, nail beds dusky, stridor, distressed. HR 116, RR 32, arterial BP 155/98, tachycardia with PVCs
1	2120	HR 116, RR 32, arterial BP 155/98, tachycardia with PVCs	2120	Rapid response team called, Epinephrine administered	2130	Anaphylaxis reversed
1	2145	RD reports feeling “much better,” HOB 15 degrees, reports not itching anymore, o2 saturation 100%	2145	Nonrebreather removed, nasal cannula placed instead	2145	RD reports he still has a cough
1,3	2205	Assessed dressing, hematoma developing that is 7.62 cm, gauze is saturated with bright red blood, RD reports he “feels like he is sitting on something wet.” BP 112/74, HR 88, RR 14, O2 100% 3L NC	2215	Applied pressure to site for ten minutes to create hemostasis. Applied pressure dressing.		Bleeding has stopped, P 78, BP 112/74, RR 12,
1	2230	Potassium 3.2 (previously 3.6),	2235	Reported Potassium level (low) to provider who then prescribed oral potassium, administered oral potassium. Provide education on modifiable risk factors	2240	RD shares he walks at work, doesn’t have time to go to the gym, eats fast food 4x a week “so busy it is easier to grab,” likes to eat steak, bread. Stopped smoking one month ago. Potassium increased to 3.4
1	12/08 1900	Damage to portion of left ventricular myocardium, left anterior descending	1900	Communicated with charge nurse concerns about manifestations of	1910	MAP 54, arterial BP 88/54, agitated, restless. Skin feels cold and clammy to

		coronary artery occluded and stented		cardiogenic shock. Increased o2 to 3L		the touch.
1	1915	Urinary output 48ml/per hour. BP 99/56	1920	250 ml/hr normal saline, dobutamine drip infusing	1930	RD remains unstable, systolic BP readings remain below 90
1	1940	BP 80/52, HR 96, RR 12, 96% 4L NC	2000	Norepinephrine IV 0.5 mcg/min, titrated based on blood pressure maintained at systolic over 100, administered through central venous catheter	2040	RD reports feeling “better, less dizzy and shaky” BP 96/56, HR 64, RR 14, O2 96% 2L NC
1,2,3,4	12/09 1900	Looks better as evidence by no shortness of breath when speaking, skin color appears normal not ashen, reports feeling better. T 36.8, HR 68, RR 12, BP 124/72, O2 98% RA, right femoral pressure dressing clean and dry, sinus rhythm	1905	Provided education on diet modifications to reduce sodium, read labels on food items, replace salt with spices. Provided education on clopidogrel, look for unusual bruising or blood in stools, notify md incase. don’t abruptly stop taking medications, take aspirin every day.	1920	RD is ready for discharge to the stepdown unit, understands his treatment plan. CVP catheter intact with opaque dressing, scant red drainage.

To Be Completed After the Simulation

The orange boxes should be filled out with your simulation patient's actual results, assessments, medications, and recommendations

NCLEX IV *(7)*: Reduction of Risk

Actual Labs/ Diagnostics

ECG demonstrated elevated ST segment
Cardiac enzymes, notable troponin T increasing from 0.2 to 0.8, troponin I from 0.06 to 0.09
CBC, UA, ABGs, CMP, Coag studies, CXR all done
Potassium notable result with a trend going from 3.6 to 3.2, then trending back up after oral administration
Lactic acid 6

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

Signs and Symptoms
MI:
Chest pain unrelieved by nitro that lasted longer than ten minutes
Crushing pain
Difficulty breathing, tachycardic
Cardiogenic shock:
Itching, cold/clammy, hypotension, increased heart rate, light headed

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

Contributing Risk Factors
Hypertension
Coronary Artery Disease
High fat/sodium diet
Lack of exercise
History of smoking/tobacco use
Gender (male)
weight 242 lb

Therapeutic Procedures
Non-surgical
Oxygen
Bedrest

Surgical
Percutaneous transluminal balloon angioplasty w/ stent

NCLEX IV *(7)*: Reduction of Risk

Prevention of Complications
(Any complications associated with the client's disease process? If not what are some complications you anticipate)

Complications include cardiogenic shock due to damage to portion of left ventricular myocardium, left anterior descending coronary artery occluded and stented
Other potential complications include bleeding, infection, electrolyte imbalance, DVT

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

Medication Management
Morphine
Nitroglycerine
Aspirin
Lisinopril
Clopidogrel
He received the following after allergic reaction and the start of cardiogenic shock:
Dobutamine
Epinephrine
Norepinephrine
Diphenhydramine

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

Non-Pharmacologic Care Measures

Patient education on healthier lifestyle choices
HOB 10-15 degrees
Maintaining straight leg after PCI

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

Stressors the client experienced?

Pain and dyspnea from MI
Hematoma at the puncture site
Allergic reaction from contrast
Cardiogenic Shock
Lifestyle changes

Client/Family Education

Document 3 teaching topics specific for this client.

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

Multidisciplinary Team Involvement
(Which other disciplines were involved in caring for this client?)
ED nurse, cath lab team, physicians

• Taught about platelet therapy and monitoring brusing/blood in stools • Diet modifications, limiting sodium and red meat •Importance of exercise	

Patient Resources

Cardiac Rehab
Nutritionist
Resources for local gyms
Resources for appropriate recipes in diet restriction

Reflection Questions

Directions: Write reflection including the following:

1. What was your biggest "take away" from participating in the care of this client?
   My biggest takeaway was how quickly the patient deteriorated as well as how many different things went wrong, including an allergic reaction, shock, and hematoma. This demonstrated to me how important frequent and specific assessments are.

2. What was something that surprised you in the care of this patient?
   I was expecting the scenario to be more based on the immediate and after care of an MI, so I was surprised when the care was focused on other complications. It highlighted how there really is never "just one problem." It is important to be aware of every complication and notice early signs of deterioration.

3. What is something you would do differently with the care of this client?
   I don't think I would have done anything any different from the nurse. He coordinated with the care team and wasn't afraid to ask the charge nurse questions and he was intentional about researching medication prior to administration. He also was helpful with providing education at the end.

4. How will this simulation experience impact your nursing practice?
   This nurse recognized very early on the complications that this particular patient may/did face, so he was able to appropriately respond. Going into the specialty that I am, I am acutely aware that many of the medical risks related to psych are related to adverse effects from medication. I will continue to educate myself on the adverse effects of the medications my patients will likely be on (Ie serotonin syndrome from SSRIs, Lithium toxicity, etc) so I can quickly identify even small changes in physical health and mentation.