ATI Real Life Student Packet
N202 Advanced Concepts of Nursing
2026

Student Name: Noelle Benson

ATI Scenario: MI complications

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: Myocardial Infarction

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

Anatomy and Physiology

Normal Structures

The heart is part of the cardiovascular system and is responsible for pumping out blood the entire body.
Structure:
The heart consists of 4 chamber (right & left atrium, right & left ventricle), 3 layers endocardium: inner lining, myocardium: muscular layer, and epicardium: outer layer. The pericardium covers the heart and consists of the inner (visceral) and outer (parietal) layer. ~10-15 mLs of fluid is present to lubricate and prevent friction. The septum divides the heart vertically. The left ventricle is 2-3 times thicker because it has to pump out the blood to the rest of the body.
Heart valves:
4 valves that keep blood flowing in one direction. The mitral and tricuspid valves control blood moving from the atrium to the ventricle, they have thin cord called chordae tendinea that prevent eversion. The pulmonic valve sits between the right ventricle and pulmonary artery. The aortic valve sits between the left ventricle and aorta.
Blood flow through the heart:
Deoxygenated blood enters the R atrium via the inferior or superior vena cava -> then in passes through the tricuspid valve and into the R ventricle -> it passes through the pulmonary valve and enters the pulmonary artery to go to the lungs for gas exchange to occur & blood to become oxygenated -> blood returns to the heart from the lungs via the pulmonary vein into the L atrium -> blood passes through the mitral valve and enters the L ventricle -> then blood is ejected through the aortic valve into the aorta and into the circulatory system.
Blood supply to the myocardium:
The myocardium has its own blood supply via coronary circulation. There are 2 major coronary arteries the left which supply blood to the left ventricle, atrium, interventricular, and part of the right ventricle. The right coronary artery supplies blood to the right atrium, ventricle, and part of the posterior wall of the left ventricle. 90% of people also get their blood supply for the AV node and bundle of His from the right coronary artery.
Conduction system:
Used to generate and move electrical impulse or action potentials. The impulse begins at the sinoatrial node (SA) node (pacemaker of heart), and travels through intraarterial pathways that depolarize the atria and result in contraction. The electrical impulse travels from the atria to the AV node via the internodal pathways then moves through the bundle of His and the left and right bundle branches. The AP moves through the walls of the ventricles via the Purkinje fibers. This triggers a synchronized R & L ventricle contraction & ejection into the pulmonary and system circulation. The last repolarization occurs when the

NCLEX IV *(7)*: Reduction of Risk

Pathophysiology of Disease

STEMI: ST elevation MI, occurs when a thrombus fully oculus the full coronary artery. Results of abrupt stoppage of blood flow through a coronary artery. Total coronary occlusion, heart muscle hypoxic w/n 10 sec -> switches to anaerobic metabolism -> buildup of lactate acid. Subendocardial is earliest ischemic tissue. Transmural if full thickness. Necrosis of full myocardium take 5-6 hours. Described based on the location on damage. Anterior (LAD): V1-V4, inferior (RCA): II, III, aVf, lateral (left circumflex): V5-V6, aVL
NSTEMI: non- ST elevation myocardial infarction. Caused by a nonocclusive thrombus. On the ECG, it may or may not show ST depression or T wave inversion. An MI occurs when there is an abrupt stoppage of blood flow to the coronary arteries, causing cell death. Deterioration of once stable plaque -> rupture -> platelet aggregation -> thrombus Most MI occur in people who already have CAD. NSTEMIs do not need emergent cardiac caths but will need one w/n 12-72 hrs.
During the initial phase of the MI ischemic heart cells release catecholamines (norepinephrine and epinephrine) which cause diaphoresis, increased HR and BP, and vasoconstriction of peripheral blood vessels. The pt will initially have an increased HR & BP then the BP will decrease due to decreased CO. They can present w/ abnormal heart sounds, JVD, crackles, n/v, & fever.
During the healing process, leukocytes infiltrate the ischemic area, enzymes of neutrophils & macrophages remove necrotic tissue, and a collagen matrix is laid down to form scar tissue. After 10-14 days the scar tissue is weak & the heart is vulnerable to stress. By 6 weeks the scar

pathways regain the resting polarized condition. Ablute refractory period: during ventricular contraction, heart muscle doesn't respond to any new stimuli. Relative refractory period: after muscle recovers in early diastole.
ECGs:
P wave; 1sr wave begins at the firing of SA node, depolarization of atria. QRS complex: depolarization from AV node through ventricles. T wave: repolarization of ventricles, U wave: repolarization of Purkinje fibers
Mechanical System
Systole: contraction of heart muscle, results in ejection of blood from ventricles. Diastole: relaxation of heart muscles, filling of ventricles. Cardiac output: amount of blood pumped by each ventricle each minute. (normal 4-8 L/min). Preload: volume of blood stretching the ventricles at the end of diastole, before the next contraction. Afterload: peripheral resistance against pumping of L ventricle. Depends on the size of the ventricle, wall tension, and arterial BP. Increasing preload, contractility, and afterload increases the workload of the heart muscle, resulting in increased O2 demand.
Blood vessels
Arteries: carry oxygenated blood away from the heart (except for the pulmonary artery).
Veins: carry deoxygenated blood towards the heart (except for the pulmonary vein)
Capillaries: have walls made of thin epithelia cells to allow for exchange of nutrients & metabolic products. The connect through arterioles and venules.
Regulation
SNS: increase speed and impulse of conduction through the AV node. Parasympathetic nervous system: slows HR by decreasing impulses from SA node.
Baroreceptors: in aortic arch and carotid sinus are sensitive to pressure within the arterial system.
Chemoreceptors: found in the aortic and carotid bodies and the medulla. They cause change to the RR and BP in response to arterial CO2.
Blood Pressure
Arterial blood pressure: force exerted by blood against walls of arterial system
Systolic blood pressure: peak pressure exerted against arteries when heart contracts
Diastolic blood pressure: residual pressure in arterial system during ventricular relaxation

tissue is replaced with necrotic tissue. there is ventricular remodeling (which can lead to hypertrophy and HF)

To Be Completed Before the Simulation

Anticipated Patient Problem: Decreased cardiac output

Goal 1: ECG will not show any signs of worsening condition (arrhythmias or increased ST elevation) during time of care

Goal 2: Will have >/= 30 mL/hr UO during time of care

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?
Monitor continuous ECGs	Correct any arrhythmias (common with MI), correction based on type and frequency of arrhythmia prn
Monitor BP, HR, RR, SpO2, temp at least q2 or continuous	Give O2, ACE inhibitors, beta blockers as ordered
Monitor cardiac biomarkers (troponin, CK-MB) q daily	Alert primary HCP to rising levels and prepared for other interventions prn
Monitor I&O & UO q4 & prn	Give fluids (hypovolemia) or diuretics (pulm edema/HF) as ordered
Assess for signs of cardiogenic shock q6 & prn	Alert HCP immediately, quick tx with O2 & inotropic agents prn
Assess electrolytes q daily	Replace any electrolytes and correct any imbalance to prevent and treat potential dysrhythmias as ordered

To Be Completed Before the Simulation

Anticipated Patient Problem: Acute pain (angina)

Goal 1: Will have relief of chest pain with use of nitroglycerin w/o an side effects (hypotension) during time of care

Goal 2: Will not have reoccurrence of chest pain after intervention (PCI or thrombolytic) during time of care

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 of pain q1 & prn	Notify provider about any increasing pain or pain in different location (jaw, back, arm) prn
Assess type of pain (crushing, pressure, stabbing) q1 & prn	Provide supplemental O2 to improve myocardial O2 demand prn
Assess relief of chest pain after any admin of nitro and morphine	Titrate Nitro drip to decrease level of chest pain or till BP is below allowed parameter prn
Assess for recurrence of chest pain after PCI or thrombolytics prn	Alert HCP for possible reoccurrence of infraction prn
Assess for factors that increase angina q2 & prn	Limit factors than increase pain till proper treatment is given (limit activity & increased cardiac workload) prn
Assess for amount of time pain has been occurring for prn	Determines window how long provider has to perform intervention which thrombolytics (30 from arrival at hospital) or PCI (STEMI – 90 from arrival) prn

To Be Completed During the Simulation:

Actual Patient Problem: Decreased CO/inadequate tissue perfusion

Clinical Reasoning: Acute MI, hypotension, tachycardia, PVCs, PCI & stent placement, cardiogenic shock, vasopressors, damage to LV

Goal: Will maintain SBP >/= 90 during time of care Met: ☐ Unmet: ☐

Goal: Will maintain UO >/= 30 mL/hr during time of care Met: ☐ Unmet: ☐

Actual Patient Problem: Deficient knowledge

Clinical Reasoning: Eats out 4x/wk, sedentary lifestyle, unaware of modifiable risk factors, new medications & side effects

Goal: Will state 2 risk factors for Mi and 2 ways to decrease those risk factors during time of care Met: ☐ Unmet: ☐

Goal: Will properly identify medications to be discharged on and why they are need and anything to monitor with them during time of care Met: ☐ Unmet: ☐

Additional Patient Problems: Acute pain, fluid & electrolyte imbalance, risk for bleeding, Ineffective Airway Clearance

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
Acute pain	12/7 1655	Outside shoveling snow "I just don't feel well" chest tightness & squeezing, visible discomfort	12/7 1655	Took nitroglycerin tab	12/7 1625	Chest pain not relived in 5 min after nitro, took 3 doses of nitro,
Acute pain	12/7 1715	Chest pain continued after 3 doses of nitro	12/7 1715	Administered 325 mg ASA, called EMS & on way to hospital	12/7 1725	Chest pain continues "feels like my chest is being squeezed", hard to breathe, dizzy, & sick to stomach, no CP relief with nitro, pain 8/10

Decreased CO/inadequate tissue perfusion	12/7 1720	HR: 104, BP: 96/56m RR: 26, SpO2: 94% 4L NC, pain 8/10	12/7 1720	1000 mL NS 300 ML bolus then 100 mL/hr, titrate O2 to maintain SpO2 > 96%	12/7 1735	HR: 104, BP: 102/68, RR: 22, SpO2: 97% 4L NC, pain 8/10
Decreased CO/inadequate tissue perfusion	12/7 1730	ECG shows ST segment elevation, dx with STEMI	12/7 1755	Going to cath lab to reopen blocked artery, PCI & placement of stent in left LAD	12/7 2100	HR: 96, BP: 112/66, RR: 14, SpO2: 98% 2L NC, CVP: 10, NSR with PVCs, pain 0/10
Risk for bleeding	12/7 2100	Vascular closure device in right femoral puncture site, no bleeding or hematoma, dressing clean & dry	12/7 2100	Admitted to ICU for frequent VS, insertion site checks, & LE neurovascular assessment, educated about need to stay flat for 2 hours, if need to cough press on dressing	12/7 2115	Dressing clean, dry and intact, HOB remains at 10 degrees
Ineffective Airway Clearance	12/7 2115	Reports itchiness in arm and chest, stated when ate shrimp tongue swelled up, Coughing and running nose, having a hard time catching breath, wheezing on auscultation, intermittent stridor, ashen, nailbeds dusky, appears anxious	12/7 2120	Alert HCP about shellfish allergy, Admin 25 mg IV diphenhydramine, applied non rebreather 15L/min, alerted HCP & RR team	12/7 2120	Still feeling SOB, HR: 116, BPL 155/98, RR: 32, SpO2: 87% non rebreather
Ineffective Airway Clearance	12/7 2120	Still feeling SOB, HR: 116, BPL 155/98, RR: 32, SpO2: 87% non rebreather	12/7 2130	Administer epinephrine IM	12/7 2200	Anaphylaxis reversed, “feel much better” no reports of dyspnea or itching, HR: 88, BP: 108/74, SpO2: 100% NC, RR: 14, CVP 10
Risk for bleeding	12/7 2215	Continues to report “nagging cough”, hematoma present a puncture site on assessment, 7.26 cm groin	12/7 2230	Remined to hold pressure on insertion site when coughing.	12/7 2300	Bleeding stopped, outlined hematoma to assess if getting bigger, Hgb: 14.8 (was 15.9 on admit)

		hematoma, dressing saturated with bright red blood		Applied pressure right groin site for at least 10 min		
Fluid & electrolyte imbalance	12/7 2230	K+ 3.2, HRL 74, NSR w/ PVCs, output 300 mL, intake 500mL	12/7 2300	Administered K+ 20 mEq PO	12/8 0400	K+ 3.4 continue PO K+ BID
Deficient knowledge	12/8 1700	Obesity, only walks at work, fast food 4x/week, stopped smoking 1 month ago	12/8 1705	Provided education on modifiable risk factors for CAD & MI, educated about eating foods high in fiber & low in sat fat, 4-6 serving of fruit & veg each day, take meds regularly	12/8 1710	Verbalized understanding of modifiable risk factors way to reduce risks
Decreased CO/inadequate tissue perfusion	12/8 1945	Not progressing, damage to LAD, manifestations of cardiogenic shock. MAP: 54, agitation and restlessness, arterial BP 88/54, skin cool & clammy, LV damaged, UO drop	12/8 1945	Dobutamine 2.5 mcg/kg/hr (16.5 mL/hr) titrate prn, 1000 mL NS at 250 mL/hr	12/8 2010	HR: 58, sinus brady, BP: 78/56, CVP: 8, SpO2: 96% 4L NC, RR: 12, UO 42 mL
Decreased CO/inadequate tissue perfusion	12/8 2010	HR: 58, sinus brady, BP: 78/56, CVP: 8, SpO2: 96% 4L NC, RR: 12, UO 42 mL	12/8 2010	Administered norepinephrine to increased BP and help improve blood flow, start at 0.5 mcg/min & titrate to maintain SBP > 100	12/9 1900	BP documented q2-3 min initially then, q5 when stabilize Less shaky and dizzy. BP: 124/72, HR: 68 NSR, RR: 12, SpO2: 98%
Deficient knowledge	12/9 1915	Eats out of lot 4x/wk, should be consuming 1500 mg sodium intake a day	12/9 1915	Educated about sodium reduction, like reading labels & provided hand out from dietitian	12/9 1915	Selected shredded wheat cereal which was lowest in sodium, verbalized understanding of reducing sodium and foods that are high in sodium
Deficient knowledge	12/9 1915	Taking clopidogrel at home, pt asked for more information	12/9 1915	Provided education of med and use, also provide education about importance of monitoring for s/sx bleeding & need to	12/9 1915	Verbalized understanding of medications need for DTAP and what to look out for

				DTAP (clopidogrel & ASA)		

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
CK: 0
Troponin T: 0.2
Troponin I: 0.06
Lactate venous: 0.6
aPTT: 34 sec
PT: 122
INR: 0.9
K+: 3.2
ECG: ST segment elevation (LAD)
CXR: no fluid or pneumothorax, aortic arch has calcification, & intact, no dilation of artery
PCI/PTCA: blocked LAD
ECG/Tele: NSR with occasional PVC
A-line
CVP: 8-10

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

Signs and Symptoms
Chest pain 8/10
Angina unrelieved with nitro and rest
Squeezing in chest
Nausea/vomiting/feeling ill
Diaphoretic
Tachycardia
Hypotension
Dizziness
SOB, dyspnea

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

Contributing Risk Factors
HTN
CAD w/ angina
Asthma
Smoking & chewing tobacco
Male
AA
Obesity
Family hx of cardiac disease
Sendatary
Poor diet

NCLEX IV *(7)*: Reduction of Risk

Therapeutic Procedures
Non-surgical
Thrombolytic therapy

Surgical
PCI/stent placement
CABG
Atherectomy
Intraarotic balloon pump (cardiogenic shock)

Prevention of Complications
(Any complications associated with the client's disease process? If not what are some complications you anticipate)
Allergic reaction to dye
Cardiogenic shock
Dysrhythmias
Acute decomp HF
Pulmonary edema
Thromboembolism
Pericarditis/Dressler's syndrome
LV free wall rupture
Ventricular aneurysm
Papillary muscle rupture

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

Medication Management
Nitroglycerin
Morphine
ASA
Clopidogrel
Lisinopril
IVF
Dobutamine (cardiogenic shock)
Norepinephrine (cardiogenic shock)
Electrolyte replacement (K+ for hypokalemia)

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

Non-Pharmacologic Care Measures
O2 therapy
Rest initially
Cardiac rehab
Diet modifications/cardiac diet
Lifestyle modifications

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

Stressors the client experienced?
Worry about getting worse
Not being home/away from loved ones
Complications
Long recovery
Never getting back to baseline

Epinephrine (allergic reaction to contrast media)		

Client/Family Education

Document 3 teaching topics specific for this client.

- 1500 mg sodium restriction per day
- Use of both ASA and clopidogrel for DAPT after MI and stent placement
- Reduction of risk factors such as obesity and poor diet

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

Multidisciplinary Team Involvement

(Which other disciplines were involved in caring for this client?)

Emergency medicine
Cardiology
Cath lab
ICU/intensivist
PT/OT/cardiac rehab
Social work
Dietitian

Patient Resources

American heart association
Support groups
Survivors have heart
Education handouts

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 from this ATI clinical was how important it is for through assessments and observation to catch and treat complications. The client in this scenario had multiple complications related to his MI & the nurse in the ICU was able to recognize these complications and promptly treat them preventing further complications and damage. It also was helpful to see how different complications can present and the time frames that they can occur within.

2. **What was something that surprised you in the care of this patient?**
   A few things about the care of this client surprised me. First was the clients lack of knowledge relating to his condition and risk factors he had. This goes to show that not all clients receive adequate teaching about their conditions and certain risk factors that they can modify. Another thing that surprised me was the amount of complication the client faced after his MI and receiving treatment. It was helpful to have this in the scenario because it reminded me that a client can have more than 1 complication at the same time or different times.

3. **What is something you would do differently with the care of this client?**
   Something I would do differently with the care of this client is I would provide more education. I thought that the ICU nurse did a good job teaching the client and his wife about diet modifications that can help improve his condition and reduce further risk. I would have also included education about the importance of regular exercise, need to take all prescribed medications, other potential complications that can occur later on & what to look out for, & when to contact a provider about new or worsening symptoms.

4. **How will this simulation experience impact your nursing practice?**
   This simulation will impact my nursing practice by reminding me of the need for good assessment on patients, especially those who are in a critical care setting. Small changes to a client can indicate a much bigger issue and when caught early the more likely it is to have a better outcome.