



Answer KEY-ACS-MI Unfolding Reasoning

Med Surg Clinical (Quinsigamond Community College)

Acute Coronary Syndrome (ACS) Myocardial Infarction (MI)



JoAnn Smith, 68 years old

Answer Key

UNFOLDING Reasoning Case Study-ANSWER KEY

Acute Coronary Syndrome/Acute MI

History of Present Problem:

JoAnn Smith is a 68-year-old woman who presents to the emergency department (ED) after having three days of progressive weakness. She denies chest pain, but admits to shortness of breath (SOB) that increases with activity. She also has epigastric pain with nausea that has been intermittent for 20-30 minutes over the last three days. She reports that her epigastric pain has gotten worse and is now radiating into her neck. Her husband called 9-1-1 and she was transported to the hospital by emergency medical services (EMS).

Personal/Social History:

JoAnn is a recently retired math teacher who continues to substitute teach part-time. She is physically active and lives independently with her spouse in her own home. She has smoked 1 pack per day the past 40 years. JoAnn appears anxious and immediately asks repeatedly for her husband upon arrival.

What data from the histories are RELEVANT and have clinical significance to the nurse?

RELEVANT Data from Present Problem:	Clinical Significance:
<i>Three days of progressive weakness</i>	<i>Though weakness is a generalized complaint, it must be recognized as clinically significant by the nurse because it is NOT uncommon for women, the elderly, and those with diabetes to have atypical presentations with acute coronary syndrome (ACS) that do not always include chest pain.</i>
<i>Denies chest pain, but admits to SOB that increases with activity as well as epigastric pain that has been intermittent for 20-30 minutes over the last 3 days</i>	<i>The combination of SOB, and epigastric pain must be recognized as an atypical variant of ACS. Dyspnea is a common feature associated with MI due to pulmonary congestion from diastolic dysfunction [Epocrates].</i>
<i>Her epigastric pain has become worse and is radiating into her neck</i>	<i>When clustered with weakness and SOB, the significance of an atypical presentation of ACS must be considered by the nurse. Though referred ischemic pain is most commonly seen with chest pain, it can be the ONLY physical complaint for women, the elderly, and those with a history of diabetes. For this scenario, JoAnn has two of these three risk factors.</i>
RELEVANT Data from Social History:	Clinical Significance:
<i>She has smoked 1 pack per day the past 40 years</i>	<i>Cigarette smoking is the most significant modifiable risk factor that contributes to heart disease.</i>
<i>JoAnn appears anxious and immediately asks repeatedly for her husband upon arrival.</i>	<i>Anxiety INCREASES the workload of the heart. Therefore the nurse must do all that is possible to decrease her anxiety. Find her husband and his presence will likely decrease her anxiety!</i>

What is the RELATIONSHIP of your patient's past medical history (PMH) and current meds?

(Which medications treat which conditions? Draw lines to connect)

PMH:	Home Meds:	Pharm. Classification:	Expected Outcome:
<ul style="list-style-type: none"> • Diabetes mellitus type II • Hypertension • Hyperlipidemia • Cerebral vascular accident (CVA) with no residual deficits • Gastro-esophageal reflux disease (GERD) • Anemia-Iron deficiency 	<ol style="list-style-type: none"> 1. Iron Sulfate 325 mg daily 2. Lisinopril 5 mg daily 3. Simvastatin 20 mg daily 4. Aspirin 81 mg daily 5. Clopidogrel 75 mg daily 6. Omeprazole 20 mg daily 7. Metformin 500 mg PO bid 	<ol style="list-style-type: none"> 1. Iron supplements 2. ACE inhibitor 3. Lipid lowering agents (statins) 4. Salicylates 5. Platelet aggregation inhibitors 6. Proton pump inhibitors 7. Antidiabetics (biguanides) 	<ol style="list-style-type: none"> 1. Increase Hgb 2. Lower BP/protect kidneys in DM 3. Lower cholesterol 4. Inhibit clots 5. Inhibit clots 6. Decrease gastric acid 7. Lower blood glucose

What medications treat which conditions?

- Diabetes mellitus type II >> glyburide
- Hypertension >> lisinopril
- Hyperlipidemia >> simvastatin
- Cerebral vascular accident (CVA) with no residual deficits >> ASA/Clopidogrel
- Gastro-esophageal reflux disease (GERD) >> omeprazole
- Anemia-Iron deficiency >> iron sulfate

One disease process often influences the development of other illnesses. Based on your knowledge of pathophysiology (if applicable), which disease likely developed FIRST that created a “domino effect” in her life?

- Circle what PMH problem likely started FIRST
 - Diabetes mellitus type II...likely started the domino effect, though hypertension and hyperlipidemia are close seconds that worked together to cause the vascular complications that JoAnn also has experienced
- Underline what PMH problem(s) FOLLOWED as domino(s)
 - Hypertension
 - Hyperlipidemia
 - Once the trifecta of diabetes, hypertension, and hyperlipidemia have had time to work together, vascular complications such as CVA and PVD will manifest in just a matter of time!
 - Cerebral vascular accident (CVA) with no residual deficits

Patient Care Begins:

Current VS:	P-Q-R-S-T Pain Assessment (5th VS):	
T: 99.2 F/37.3 C (oral)	Provoking/Palliative:	Nothing/Nothing
P: 128 (regular)	Quality:	Ache
R: 24 (regular)	Region/Radiation:	Left arm that radiates into neck
BP: 108/58	Severity:	5/10
O2 sat: 99% room air	Timing:	Intermittent-20-30" at a time

What VS data are RELEVANT and must be recognized as clinically significant by the nurse?

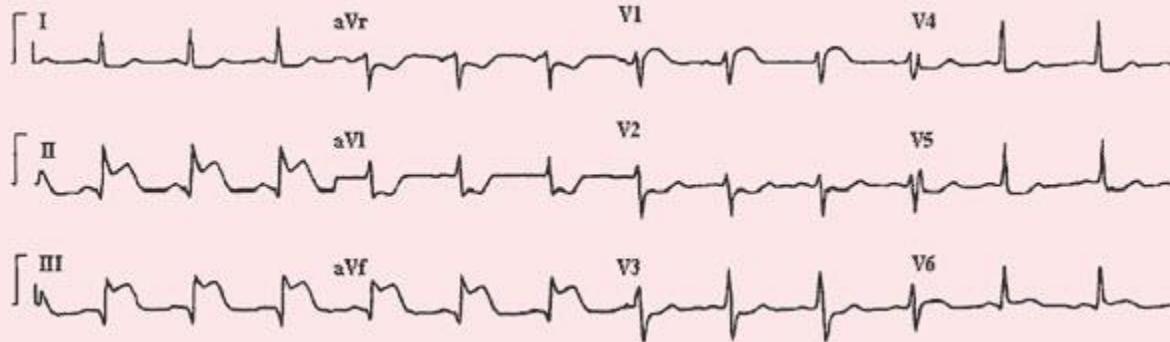
RELEVANT VS Data:	Clinical Significance:
P: 128 (regular)	<i>JoAnn is tachycardic, therefore the nurse must step back and ask WHY? Could be due to anxiety, or, knowing that she may be having an acute coronary syndrome, she could be compensating for decreasing cardiac output. Remember the relevance of this essential formula from A&P that must be applied to practice...CO=SVxHR. This tachycardia can also be due to high sympathetic output [Epocrates].</i>
R: 24	<i>Remember that with any patient with possible ACS, your goal is to DECREASE THE WORKLOAD OF THE HEART! Tachycardia in this context is a clinical RED FLAG because it increases the workload of the heart and increases O2 demands that are currently compromised.</i>
BP: 108/58	<i>Tachypnea could be due to anxiety or SOB that may be due to pulmonary edema with ACS. Recognize the need to thoroughly assess breath sounds and cluster assessment findings with tachypnea.</i> <i>In the context of ACS, this blood pressure is too low. Depending on how the physician manages the case, you will need to be cautious with any medications that can lower BP.</i> <i>In ACS or MI, afterload must be reduced. The goal in acute MI is generally a systolic BP (SBP) of around 120. This is typically done through beta blockers such as metoprolol or nitroglycerin sublingual or IV drip.</i>

Current Assessment:	
GENERAL APPEARANCE:	Anxious, appears uncomfortable, body tense
RESP:	Respirations labored, coarse crackles present in bases bilaterally anterior/posterior
CARDIAC:	Pale, diaphoretic, no edema, heart sounds regular S1S2 with no abnormal beats, pulses strong, equal with palpation at radial/pedal/post-tibial landmarks
NEURO:	Alert & oriented to person, place, time, and situation (x4)
GI:	Abdomen soft/non-tender, bowel sounds audible per auscultation in all 4 quadrants
GU:	Voiding without difficulty, urine clear/yellow
SKIN:	Skin integrity intact, skin turgor elastic, no tenting present

What assessment data is RELEVANT and must be recognized as clinically significant by the nurse?

RELEVANT Assessment Data:	Clinical Significance:
GENERAL APPEARANCE: Anxious, appears uncomfortable, body tense	<i>This could be the result of anxiety due to her current condition and/or difficulty breathing. Anxiety increases myocardial oxygen demand, elevates systolic BP, and increases the workload of the heart. Therefore it must be addressed and not allowed to continue. Emotional support and education can help decrease this immediately, but she may need medications such as lorazepam or morphine to effectively decrease her blood pressure.</i>
RESPIRATORY: Respirations labored, coarse crackles present in bases bilaterally	<i>This cluster of data confirms impairment of diffusion. The alveoli are filling with fluid (crackles) from likely pulmonary edema due to cardiogenic shock in acute MI.</i> <i>Reinforce that the first choice to more accurately assess breath sounds is POSTERIOR —there is less tissue to auscultate through compared to anterior -- especially with women and additional breast tissue. Pay close attention to the BASES as gravity will pull any secretions/fluids downward and is usually the first place that adventitious breath sounds are auscultated. Be sure to listen for full inspiration and expiration in each lung field.</i>
CARDIAC: Pale, diaphoretic, no edema, heart sounds regular S1S2 with no abnormal beats, pulses strong, equal with palpation at radial/pedal/post-tibial landmarks	<i>Pale, and being diaphoretic confirms that the sympathetic nervous system stimulation is present and is there for a reason! The nurse must step back and ask WHY? Fight or flight is now a reality with a life-threatening presentation!</i>

12 Lead EKG:



Interpretation:

The ST elevation is clearly present in the inferior leads of II, III, and AVF. This is classic diagnostic criteria for an acute myocardial infarction (MI) that is also referred to as a ST elevation myocardial infarction (STEMI) vs. a non-ST elevation myocardial infarction (non-STEMI).

A STEMI is more concerning because it involves the FULL thickness of the myocardium and more muscle is at risk of loss. This is reflected by the ST segment elevation. In contrast, a non-STEMI is PARTIAL thickness infarction of the

myocardium and is reflected by a ST segment depression on a 12-lead EKG instead.

Clinical Significance:

This must be recognized as an inferior STEMI that requires immediate intervention! Time is muscle!

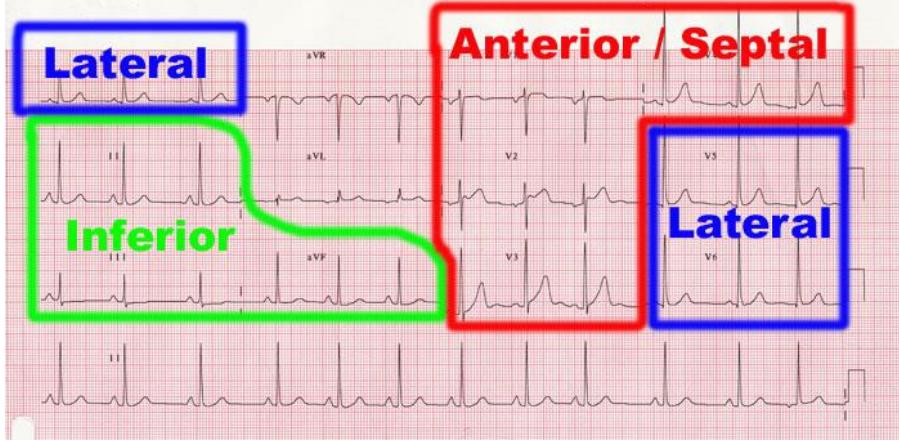
Inferior wall infarction occurs with occlusion of the right coronary artery. This infarction manifests by ECG changes in leads II, III, and aVF.

Conduction disturbances are expected with an inferior wall MI because of the anatomy of the coronary arterial supply. The right coronary artery (RCA) perfuses the sinoatrial (SA) node in slightly more than half of the population and supplies the proximal bundle of His and the atrioventricular (AV) node in more than 90 percent of individuals. Heart block and other conduction disturbances should be anticipated with an inferior MI (Urdan, 2014).

Location of ST Segment Changes (lateral/anterior/inferior):

Use the diagram below to identify the location of the infarction:

Though this content on basic 12-lead EKG interpretation may be above the scope of knowledge required for most programs, take advantage of the APPLICATION of the principle that **ischemia causes distinct EKG changes**. This is relevant when a patient on routine cardiac telemetry monitoring begins to have NEW ST-T wave changes. If the nurse understands the significance of these changes, a **RESCUE** of a patient with a change of status can begin!



Radiology Report: Chest x-ray

What diagnostic results are RELEVANT and must be recognized as clinically significant to the nurse?

RELEVANT Results:	Clinical Significance:
Scattered bilateral opacities consistent with atelectasis or pulmonary edema	The chest x-ray and echocardiogram confirm the ominous development of severely depressed left ventricle (LV) function due to inferior MI and pulmonary edema. This report confirms what you already suspect and are seeing clinically, pulmonary edema secondary to cardiogenic shock in the context of an acute MI. This should not delay implementation of reperfusion therapy. [Epocrates]

Radiology Report: Echocardiogram

What diagnostic results are RELEVANT and must be recognized as clinically significant to the nurse?

RELEVANT Results:	Clinical Significance:
Global left ventricle hypokinesis with ejection fraction of 25%	Reflects severely damaged left ventricle due to inferior MI. Remember a normal ejection fraction of 55-65 percent and what this represents. An EF of 10-15 percent is end-stage heart function because only 10-15 percent of the blood volume of the LV is being ejected into circulation. In the context of acute MI, an initial low ejection fraction is not necessarily where the patient will remain with their heart muscle, due to zones of injury with an acute MI that are similar to a target and bullseye. The bullseye is the infarcted zone that will not return to life, but there is a ring around this zone that is "stunned myocardium." With reperfusion and time (usually 3-7 days,) this stunned tissue is viable but not yet able to contract. This is similar to when your arm falls asleep when you lie on it wrong at night. It is viable, but initially does not move well. When this echo is repeated in 7 days--this is more telling of what the long-term function will be.

Lab Results:

Complete Blood Count (CBC):	Current:	High/Low/WNL?
WBC (4.5-11.0 mm 3)	10.5	WNL
Hgb (12-16 g/dL)	12.9	WNL
Platelets(150-450x 103/ μ l)	225	WNL
Neutrophil % (42-72)	70	WNL

What lab results are RELEVANT and must be recognized as clinically significant by the nurse?

RELEVANT Lab(s):	Clinical Significance:
<i>Though some of the following lab values are WNL, because they are ALWAYS RELEVANT, they must be intentionally noted by the nurse!</i>	
WBC: 10.5	<i>ALWAYS RELEVANT based on its correlation to the presence of inflammation or infection. Will usually be increased if infection present, though it may be decreased in the elderly or peds <3 months.</i>
Hgb: 12.9	<i>ALWAYS RELEVANT to determine anemia or acute/chronic blood loss.</i>
Neutrophil %: 70	<i>ALWAYS RELEVANT for same reason as WBCs. They are the most common leukocyte and their role is as a FIRST RESPONDER to any bacterial infection within several hours or when the inflammatory response is activated.</i>

Basic Metabolic Panel (BMP):	Current:	High/Low/WNL?
Sodium (135-145 mEq/L)	135	WNL
Potassium (3.5-5.0 mEq/L)	4.1	WNL
Glucose (70-110 mg/dL)	184	HIGH
Creatinine (0.6-1.2 mg/dL)	1.5	HIGH
Misc. Labs:		
Magnesium (1.6-2.0 mEq/L)	1.8	WNL

RELEVANT Lab(s):	Clinical Significance:
<i>Though some of the following lab values are WNL, because they are ALWAYS RELEVANT they must be intentionally noted by the nurse!</i>	
Sodium 135	<i>I consider Na+ the “Crystal-Light” electrolyte. Though this is simplistic, it does help to understand in principle how basic Na+ is to fluid balance. When you add one small packet of Crystal Light to your 16-ounce bottle of water, the concentration is just right. This is where a normal Na+ will be (135-145). Where free water goes, sodium will follow to a degree. Therefore, if there is a fluid volume deficit due to dehydration, Na+ will typically be elevated because it’s concentrated (less water). If there is fluid volume excess, Na+ will be diluted and will likely be low. It is the “foundational” fluid balance electrolyte!</i>
Potassium 4.1	<i>Essential to normal cardiac electrical conduction, as is Mg+. If too high or low can predispose to rhythm changes that can be life threatening! K+ tends to deplete more</i>

	<i>quickly with loop diuretic usage than Mg+.</i>
Glucose 184	<i>Though not the most important abnormal lab in this context, it reflects either poor control or stress-induced hyperglycemia due to three-day decompensation and increased cortisol secretion by the adrenals.</i>
Magnesium 1.8	<i>Essential to normal cardiac electrical conduction, as is K+. If too high or low, can predispose to rhythm changes that can be life threatening!</i>
Creatinine 1.5	<i>GOLD STANDARD for kidney function and adequacy of renal perfusion. The functioning of the renal system impacts every body system. This is very significant and the most concerning finding as it is elevated from baseline. She needs to be diuresed and will need LARGER doses of loop diuretics to have the desired response. ***Emphasize the additional relevance of creatinine as it relates to need for cath lab and angiogram. The dye load that is given in any angiogram is nephrotoxic and can exacerbate underlying chronic renal injury or acute tubular necrosis (ATN) and push this patient into full-blown renal failure after the procedure. Though an angiogram must still be done, the nurse must recognize this connection and diligently assess urine output and creatinine post procedure!</i>

Cardiac Labs:	Current:	High/Low/WNL?
Troponin (<0.4 ng/mL)	1.8	HIGH
BNP (B-natriuretic Peptide) (<100 ng/L)	1150	HIGH

What lab results are RELEVANT and must be recognized as clinically significant by the nurse?

RELEVANT Lab(s):	Clinical Significance:
Troponin 1.8	<i>Troponins are the most sensitive cardiac marker with acute tissue injury/infarction and will be elevated in acute MI. This finding with the EKG evidence confirms the MI. Review trending of Troponin; it takes 2-6 hours to elevate...peaks in 15-24 hours and will remain elevated 7-10 days. This is relevant because when a patient presents within 2 hours of onset of CP/MI, the initial troponin may be negative, but does not mean they are NOT having an MI.</i>
BNP 1150	<i>BNP is the most concerning of these cardiac enzymes as it is >500. This confirms presence of significant acute heart failure. B-Natriuretic Peptide is a hormone produced by heart muscle. When the left ventricle is stretched or stressed as in volume overload, the secretion of this hormone will increase. BNP reflects degree and progression of heart failure. BNP physiologically promotes venous and arterial vasodilation. It is the body's own attempt to compensate for heart failure through both PRELOAD and AFTERLOAD REDUCTION. BNP also promotes diuresis by increasing the glomerular filtration rate (GFR). This influences and decreases PRELOAD.</i>

Lab Planning: Creating a Plan of Care with a PRIORITY Lab:

Lab:	Normal Value:	Clinical Significance:	Nursing Assessments/Interventions Required:
Troponin Value: 1.8 ng/mL	<0.05 ng/mL Critical Value: >0.1 ng/mL <i>suggests myocardial</i>	<i>*Contractile protein found in cardiac muscle that will be released into systemic circulation with cardiac ischemia or</i>	THINK CARDIAC-MI <i>*Assess closely for recurrent or new onset of chest pain *Assess cardiac rhythm for any changes such as PVCs, VT, or atrial fibrillation *Assess HR and SBP carefully to promote decreased cardiac workload (maintain heart rate <80 and SBP <140</i>

	<p><i>injury. Troponin is a more sensitive biomarker than CK-MB and can be elevated slightly without an infarction. This is why many MDs order CK-MBs together to see if they both trend +</i></p>	<p><i>acute MI *Levels will rise 2-6 hours after injury, peak 15-24 hours, and then remain elevated for several days. *If acute onset CP, to rule out MI, they will be done every 6 hours x3 to determine pattern of abnormal elevation</i></p>	<p><i>*Assess tolerance to activity closely</i></p>
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Clinical Reasoning Begins...

1. What is the primary problem that your patient is most likely presenting with?

- Acute inferior MI with cardiogenic shock/pulmonary edema

This is an excellent scenario that illustrates the weakness of textbook learning vs. knowledge required for practice. Textbook learning is linear, with only one topic addressed at a time. Real-world clinical practice requires the nurse to situate multiple problems that have a physiologic relationship to one another. In this scenario there are three priority problems, the acute MI, that caused pulmonary edema because of cardiogenic shock.

This is another reason why students must have a DEEP and applied understanding of pathophysiology in order to make these needed clinical connections.

2. What is the underlying cause/pathophysiology of this primary problem?

Acute MI

Myocardial infarction (MI) is the irreversible myocardial necrosis (cell death) when there is an abrupt decrease or total cessation of coronary artery blood flow to the myocardium. Three words that can help explain myocardial infarction: "Clot on Plaque"

There are three distinct ways that the coronary artery can be occluded and result in the acute reduction in oxygen delivery to the myocardium:

1. Plaque rupture
2. New coronary artery thrombosis
3. Coronary artery spasm close to the ruptured plaque

Myocardial tissue can best be salvaged within the first two hours (120 minutes) after the onset of anginal symptoms. The earlier the myocardium is revascularized, the better the chances of survival. Unfortunately, many persons do not seek treatment until the acute phase has passed or delay seeking treatment because of denial of symptoms.

Complete occlusion of the coronary artery is usually due to a thrombus that has formed when a ruptured atherosclerotic plaque exposes rough edges that attract/initiate the clotting cascade that begins with platelet aggregation. This clot becomes larger, like a snowball rolling down a hill, until it completely occludes the coronary artery causing NO distal blood flow and resultant ischemia that will lead to infarction if not promptly reopened (Urdan, 2014).

As a result, 2013 American College of Cardiology Foundation/American Heart Association guideline for the management of STEMI recommends use of primary PCI for any patient with an acute STEMI who can undergo the procedure in a timely manner by persons skilled in the procedure. Timely is defined as an ideal first medical contact to PCI time of 90 minutes or less for patients transported to a PCI-capable hospital, or 120 minutes or less for patients who initially arrive at or are transported to a non-PCI capable hospital and are then taken to a PCI-capable hospital. [Uptodate]

Cardiogenic Shock

Cardiogenic shock occurs when cardiac output is insufficient to meet the metabolic demands of the body, resulting in inadequate tissue perfusion. There are four stages of cardiogenic shock: initial, compensatory, progressive, and refractory.

1. Initial stage

Diminished cardiac output without any clinical symptoms.

2. Compensatory Stage

Baroreceptors respond to decreased cardiac output by stimulating the sympathetic nervous system to release catecholamines to improve myocardial contractility and vasoconstriction, leading to increased venous return and arterial blood pressure. Impaired renal perfusion activates the renin-angiotensin system, whose end product, angiotensin II, causes sodium and water retention as well as vasoconstriction.

3. Progressive Stage

Follows the compensatory stage if there is no intervention or if the intervention fails to reverse the inadequate tissue perfusion. As tissue perfusion remains inadequate, anaerobic metabolism takes place at the cellular level, leading to metabolic acidosis (lactic acid production). Lactic acidosis causes depression of the myocardium and a decrease in the vascular responsiveness to catecholamines, further reducing cardiac output.

4. Refractory Stage

If there is no effective intervention, cardiogenic shock will progress to the refractory stage, when the chance of survival is extremely limited.

The most common cause of cardiogenic shock is acute myocardial infarction (MI) resulting in a loss of more than 40 percent of the functional myocardium. Cardiogenic shock occurs with 10 percent to 20 percent of all hospital admissions for acute MI and carries an 80 percent mortality rate (Sommers & Fannin, 2015).

Pulmonary Edema

Cardiogenic Pulmonary Edema is the accumulation of fluid in the alveoli due to increased pulmonary capillary hydrostatic pressure because of cardiac dysfunction. It usually includes left ventricular failure with elevated pulmonary venous pressure (Fraser, 2012).

Collaborative Care: Medical Management

Care Provider Orders:	Rationale:	Expected Outcome:
Establish 2 large bore peripheral IVs	<p>Will need numerous IV medications in the initial acute phase. Have a second IV ready case of IV incompatibilities with IV drips or if infiltrates. There will be no delay in administering medications needed, especially in a code! Large bore (18 g. is preferred in case she decompensates).</p>	IVs successfully established
Metoprolol 5 mg IV push x1 now	<p>Use with caution or decide to HOLD! Reassess blood pressure every 5-15 minutes to identify current trend. Contraindications include heart failure, evidence of a low output state, and high risk for cardiogenic shock, bradycardia or heart block. [Uptodate]</p> <p>Use with caution! Beta blockers will decrease the workload of the heart by decreasing heart rate.-Remember how it also impacts contractility...reassess BP and HR after each dose. Hypotension is likely in cardiogenic shock and so nitrates may be avoided. Also, JoAnn never complains of chest pain.</p>	Heart rate will decrease to 60-80 with SBP maintained >90
Nitroglycerin IV drip-start	Intravenous nitroglycerin is useful in patients with	Not likely to eliminate chest

<p>at 10 mcg and titrate to keep SBP >100</p>	<p><i>persistent chest pain after three sublingual nitroglycerin tablets, as well as in patients with hypertension or heart failure.</i></p> <p><i>However, nitrates must be used with caution or avoided in settings in which hypotension is likely or could result in serious hemodynamic decompensation, such as right ventricular infarction or severe aortic stenosis.</i></p> <p><i>[Uptodate]</i></p> <p><i>Improves circulation to the heart as a coronary artery dilator and decreases SBP through PRELOAD reduction as it causes systemic venodilation. Start at 10 mcg (3 mL) if 50 mg in 250 mL bottle and titrate 10 mcg every 5-10" until the desired blood pressure is obtained per orders. If was NOT an MI, you would titrate to relief of chest pain in unstable angina, for example.</i></p> <p><i>Remember to reinforce that since NTG comes in a glass bottle, you need to open up the air vent on the IV tubing that is typically closed by default at the top of the tubing.</i></p>	<p><i>pain, but I have seen clinically that it will decrease the level of CP. Watch BP closely to maintain >90, especially if given with beta blocker.</i></p>
<p>Clopidogrel 600 mg po x1 now</p>	<p><i>Inhibits platelet aggregation by irreversibly inhibiting the binding of ATP to platelet receptors.</i></p>	<p><i>Inhibit platelet aggregation and prevent further extension of thrombus</i></p>
<p>Aspirin 324 mg (81 mg tabs x4) chew x1 now</p>	<p><i>Since platelet aggregation is the FIRST step of the clotting cascade, ASA will inhibit platelet aggregation and prevent further extension of the thrombus.</i></p>	<p><i>See above</i></p>
<p>Heparin 60 units/kg IV x1 now</p>	<p><i>Though heparin cannot dissolve a clot once it has formed, it can prevent the extension of an existing thrombus by neutralizing thrombin, which prevents the conversion of fibrinogen to fibrin.</i></p>	<p><i>Limit size/extension of thrombus.</i></p>
<p>To cath lab as soon as team ready</p>	<p><i>This is the gold standard of cardiac care with an acute MI and will re-establish coronary artery perfusion through PTCA and stenting if able. Goal is cath lab 90 minutes or less from time of presentation to ED where a cath lab is present.</i></p>	<p><i>Able to successfully restore circulation through PTCA/stent. Though patient will still have MI, can dramatically limit the size of the infarct with cath lab intervention when done in a timely manner.</i></p>

PRIORITY Setting: Which Orders Do You Implement First and Why?

Care Provider Orders:	Order of Priority:	Rationale:
<ul style="list-style-type: none"> • Establish 2 peripheral IVs • Metoprolol 5 mg IV push x1 now • Nitroglycerin IV drip-start at 10 mcg and titrate to keep SBP >100 • Clopidogrel 600 mg po x1 now • Aspirin 324 mg (81 mg tabs x4) chew/po x1 now 	<ol style="list-style-type: none"> 1. Aspirin 324 mg (81 mg tabs x4) chew/po x1 now 2. Clopidogrel 600 mg po x1 now 3. Establish 2 peripheral IVs 4. Metoprolol (Lopressor) 5 mg IV push x1 now 5. Nitroglycerin (NTG) IV drip-start at 10 mcg and titrate to keep SBP >100 	<p><i>This is an excellent example of the relevance of the A, B, Cs to priority setting in practice. The nurse must be able to recognize and identify which interventions impact A, B or C. This is why the nurse must also understand all aspects of the physician's plan of care and interventions!</i></p> <p><i>1. C-cardiac/circulation priority that takes almost no time. Will stop the clotting snowball quickly!</i></p> <p><i>2. C-cardiac/circulation priority that takes almost no time. Will stop the clotting snowball quickly!</i></p>

<ul style="list-style-type: none"> • Heparin 60 units/kg IV x1 now • To cath lab as soon as team ready 	<ul style="list-style-type: none"> 6. Heparin 60 units/kg IV x1 now 7. To cath lab as soon as team ready 	<p>3. C-circulatory priority but takes time to implement. In real-world practice, you would likely have another RN working with you and would do these top three simultaneously.</p> <p>4. C-circulatory-DECREASE the workload of the heart by lowering HR and BP</p> <p>5. C-circulatory-decrease workload by decreasing preload</p> <p>6. C-circulatory-prevent extension of thrombus</p> <p>7. C-circulatory-definitively open coronary artery with PTCA/stent. Though this is a TOP priority, it typically takes at least 30-45 minutes to assemble the team if on-call and other priorities need to be managed and implemented.</p>
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Medication Dosage Calculation:

Medication/Dose:	Mechanism of Action:	Volume/time frame to Safely Administer:	Nursing Assessment/Considerations:
Metoprolol 5 mg IV push (5 mg/5 mL vial)	<p><i>Blocks stimulation of beta1 (myocardial)-adrenergic receptors. Does not usually affect beta2 (pulmonary, vascular, uterine)-receptor sites.</i></p> <p><i>Therapeutic Effects: Decreased BP and heart rate</i></p>	<p><i>5 mL over 1 minute</i></p> <p>IV Push: Volume every 15 sec? 1 mL every 15 seconds will push this in slightly over 1 minute</p>	<p>*Obtain BP and HR before administering-hold typically if SBP <90. HR <60</p> <p>*Change position slowly, especially with elderly, to prevent orthostatic changes.</p> <p>*Contraindicated in worsening CHF, bradycardia or heart block...use with caution in patients with diabetes, liver disease.</p>

Medication/Dose:	Mechanism of Action:	Volume/time frame to Safely Administer:	Nursing Assessment/Considerations:
Heparin 60 units/kg IV Weight: 62 kg (1000 units/mL)	<p><i>REVIEW THE CLOTTING CASCADE! Neutralizes thrombin, preventing the conversion of fibrinogen to fibrin</i></p> <p><i>Therapeutic effects: Prevent extension of thrombus in coronary artery</i></p>	<p>Administer over 1" $62 \text{ kg} \times 60 \text{ units/kg} = 3720 \text{ units (3.72 mL)}$</p> <p>IV Push: Volume every 15 sec? Simply divide 4(1/4=15 seconds) into volume of 3.7 mL. Though it is technically 0.92 mL, KEEP IT SIMPLE and administer 1 mL every 15 seconds!</p>	<p>*After infusion, assess for signs of bleeding or hemorrhage-nose bleed, unusual bruising, hematuria, or dark, tarry stools.</p> <p>*Monitor platelets every 2-3 days if on an IV drip. Can cause drastically reduced platelets due to heparin induced thrombocytopenia (HIT)</p> <p>*If patient requires prompt reversal of anticoagulant effects, protamine sulfate is the antidote.</p>

Collaborative Care: Nursing

3. What nursing priority(ies) will guide your plan of care? (if more than one-list in order of PRIORITY)

The following are NANDA -I nursing diagnostic statements that can be used to establish a plan of care in this scenario:

- Impaired Gas Exchange (B-Breathing priority)
- Decreased Cardiac Output (C-Circulatory priority)
- Acute Pain
- Knowledge deficit

From my lens of clinical practice, I have chosen to frame nursing priorities using the essence of clinical reasoning:

DECREASE WORKLOAD OF THE HEART

Though the nurse could use the NANDA diagnosis of "Impaired Gas Exchange", it does not capture the essence of what is influencing the presence of pulmonary edema --the need to decrease the workload of the heart! If the nurse recognizes the clinical relationship between decreasing the workload of the heart and progression of pulmonary edema, it will become evident that all nursing interventions that accomplish this needed objective will also improve gas exchange.

STOP/LIMIT THE SIZE of the SNOWBALL!

Limit the size of the clot before it becomes totally occlusive. JoAnn's 12-lead EKG already demonstrates that it is likely too late and that the clot is already completely occluded the RCA, causing ST segment elevation.

MONITOR CLOSELY FOR COMPLICATIONS

Ventricular tachycardia or further progression of pulmonary edema that represent cardiogenic shock in this scenario

- Beta blockers such as Metoprolol will decrease the workload of the heart by lowering HR
- See if students understand mechanism of action of beta blockers well enough to see the downside of beta blockers in this context.
 - How do beta blockers impact CARDIAC CONTRACTILITY? They DECREASE contractility, and since JoAnn is in cardiogenic shock, this could worsen her underlying failure.

PATIENT EDUCATION (Deficient Knowledge)

This may take higher priority if the Joan goes to the cath lab emergently and either she or her husband must signed an "informed consent". Emphasize the need for the nurse to always explain what they are doing at the bedside to decrease anxiety and to remember that there is a person who is receiving care. Nurses should never forget that!

4. What interventions will you initiate based on this priority?

Nursing Interventions: DECREASE WORKLOAD OF HEART	Rationale:	Expected Outcome:
Beta blockers such as metoprolol PO or IV Emotional support and patient education. Explain in a calm, matter-of-fact voice what is happening and what you are doing to ensure a good outcome. Though you may be anxious over the severity of her presentation, you must be careful to not show this or your patient will pick up on this anxiety. Her anxiety will increase the workload of the heart-something you are trying to decrease! Have the husband with JoAnn	Decreases HR as well as SBP, which lowers afterload and the overall workload of the heart Decreasing anxiety will decrease HR and therefore decrease workload of the heart! It would be helpful to have the husband with the patient.	HR <80 HR <80 Reduce anxiety, decreased heart rate
STOP THE SNOWBALL! Limit the size of the clot if it is not too late. This patient is already demonstrating that it is too late and the clot is already occlusive. <ul style="list-style-type: none">• ASA: chew ASAP if has not taken yet• Heparin IV bolus if not going to cath lab emergently	Inhibit platelet aggregation! Chewing will get the ASA into the system faster Prevent clot from getting larger. Will NOT lyse the clot like a thrombolytic	Limit size of thrombus
ASSESS FOR COMPLICATIONS Vtach or further progression of pulmonary	Determine rhythm change	Prompt identification of

<p><i>edema/cardogenic shock</i></p> <ul style="list-style-type: none"> • Continuous cardiac rhythm monitoring • Prepare for the worst possible complication... 	<p><i>In any code, you have 5-6" before irreversible brain damage. In acute care, time is still of the essence and prompt return of rhythm is still priority 1!</i></p>	<p><i>rhythm change</i></p>
<p>PATIENT EDUCATION</p> <p><i>Your goal is to naturally and concisely educate your patient about what you are doing as you are doing it. This comes with experience, but do not forget the patient in the midst of so much to do as they are infarcting. The more they know in a limited way, the less anxious they will be and this will impact their cardiac O2 requirements and benefit them directly.</i></p> <p><i>Don't forget to keep the husband informed, especially if he is not present in the room. In addition, complete initial nursing admission or delegate as able.</i></p>	<p><i>Partner with patient and empower him/her with knowledge that will likely also decrease anxiety.</i></p> <p><i>Gathering all the important information needed to make sure that the medical record is complete and accurate will assure safe patient care throughout the entire stay at the facility.</i></p>	<p><i>Understands and is informed. May decrease or even increase anxiety.</i></p>

5. What body system(s) will you most thoroughly assess based on the primary/priority concern?

- Cardiac
- Respiratory

6. What is the worst possible/most likely complication to anticipate?

- Ventricular tachycardia (VT) of ventricular fibrillation (VF) arrest
- Cardiogenic shock with pulmonary edema

7. What nursing assessments will identify this complication EARLY if it develops?

Ventricular tachycardia (VT) of ventricular fibrillation (VF) arrest

- **Ventricular tachycardia (VT) or ventricular fibrillation (VF) arrest**
 - Correctly interpret VT/VF on the monitor. Be sure to check the patient to validate unresponsiveness
 - If the patient is alert and oriented, check the patches. A loose patch can look like a lethal rhythm on the monitor!
 - Call a code, then do not PANIC! Initiate basic life support (BLS) care, which begins with vigorous chest compressions. Make sure you have a hard surface behind the patient's back to ensure that the chest is adequately compressed.
- **Cardiogenic shock with pulmonary edema**
 - Increased respiratory rate/SOB
 - Increased oxygen needs/decreasing O2 sat
 - Increased anxiety due to SOB
 - Pale and diaphoretic
 - BP decreasing from established baseline to shock levels (SBP < 90)

8. What nursing interventions will you initiate if this complication develops?

- **Ventricular tachycardia (VT) or ventricular fibrillation (VF) arrest**
 - Call a code
 - Check the patient! Assess LOC
 - Place on defibrillator
 - Initiate CPR per BLS and then ACLS protocols

- **Cardiogenic shock with pulmonary edema**
 - Increase oxygen delivery to maintain O₂ sat >92%
 - Sit upright as possible in bed
 - Contact primary care provider to facilitate medical plan of care. May need Bi-pap or even intubation if condition deteriorates.

9. What psychosocial needs will this patient and/or family likely have that will need to be addressed?

- Knowledge and education about what is taking place and care priorities for the days ahead
- Emotional support
- Spiritual support

10. How can the nurse address these psychosocial needs?

- **Emotional support**
 - BE PRESENT and AVAILABLE. See the section on caring at the end of this case study for more information.
- **Spiritual support**
 - In the ED, spiritual care/support will be limited to encouragement, providing hope and determining if they would like to have a chaplain or their spiritual leader to be notified
 - Once admitted, it would be appropriate to ask open ended questions to assess. Those that I have found natural and effective include:
 - What gives your life purpose?
 - How has this illness affected the way you view life?
 - What is the source of your strength to face the future?
 - Are you involved/connected with a faith community?
 - If answers yes to above question...How has your current health problem affected your spiritual beliefs?

Evaluation: Two Days Later...

JoAnn had an angiogram that revealed an occluded proximal right coronary artery (RCA). She received two bare metal stents with 0 percent residual stenosis. She has been in the intensive care unit (ICU) the past two days and is now transferring to the cardiac telemetry floor. She has been receiving scheduled furosemide 40 IV mg every 12 hours. Her creatinine increased from 1.7 to 2.1 today. The last dose of furosemide was given four hours ago. She has had 100 mL urine output the past four hours. She fatigues easily, but tolerates being up in the chair for short periods of time. Faint basilar crackles persist bilaterally and her O₂ is at 2 liters per n/c.

What data from this history are RELEVANT and must be recognized as clinically significant to the nurse?

RELEVANT Data from History:	Clinical Significance:
Has been receiving scheduled Lasix 40 mg every 12 hours	There is still some degree of volume overload and the nurse will need to closely monitor I&Os and urine output.
Her creatinine went from 1.7 to 2.1 today. The last dose of furosemide IV was given four hours ago and has had 100 mL out since.	This is the most concerning finding... Creatinine is trending upward. The 100 mL of urine output over four hours must be recognized as RELEVANT because it falls below the parameters of 30 mL/hr, but in the context of receiving a loop diuretic, you would expect a more aggressive response unless she was dehydrated, volume depleted, or experiencing acute kidney injury. Reinforce the time action profile of furosemide...IV it onsets in 5", peaks in 30", and lasts 1-2 hours. Students must put this data in context that after receiving furosemide, the therapeutic response is essentially complete after two hours.
Fatigues easily, but tolerates being up in the chair for short periods of time.	This is expected due to known damage to her heart post-MI. The nurse needs to closely monitor and encourage activity as JoAnn is able so she does not remain deconditioned.
Faint crackles persist in the bases	Crackles represent fluid that could be atelectic or excess volume. Need to

bilaterally. O2 at 2l per n/c.	<p>assess for signs of heart failure closely as this patient is on a balance beam and can easily tip in the wrong direction.</p> <p>This problem could also represent cardio-renal syndrome... Simply, when the heart fails, the kidneys get less blood (oxygen). The kidneys conserve sodium and fluid to increase the BP so that they can get more blood (oxygen). The increase in fluid causes an increased workload on the heart, and it fails even more.</p>
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Current VS:	Most Recent:	P-Q-R-S-T Pain Scale:	
T: 97.2 F/36.2 C (oral)	T: 97.5 F/36.4 C (oral)	Provoking/Palliative:	
P: 76 (regular/irregular)	P: 82 (regular)	Quality:	Denies pain
R: 20 (regular)	R: 20 (regular)	Region/Radiation:	
BP: 122/58	BP: 116/68	Severity:	
O2 sat: 95% room air	O2 sat: 94% room air	Timing:	

Current Assessment:	
GENERAL APPEARANCE:	Resting comfortably, appears in no acute distress
RESP:	Denies SOB, non-labored respiratory effort, breath sounds equal aeration bilaterally with faint crackles in both bases
CARDIAC:	Pink, warm & dry, 1+ pitting edema in lower extremities, heart sounds regular-S1S2, pulses strong, equal with palpation at radial/pedal/post-tibial landmarks
NEURO:	Alert & oriented to person, place, time, and situation (x4)
GI:	Abdomen soft/non-tender, bowel sounds audible per auscultation in all 4 quadrants
GU:	50 mL urine output since furosemide IV administered two hours ago, urine clear/yellow
SKIN:	Skin integrity intact, femoral puncture site soft, non-tender with no drainage, redness, or bruising

1. What clinical data are RELEVANT and must be recognized as clinically significant?

RELEVANT VS Data:	Clinical Significance:
Though all VS are vital, there are no concerning trends and are WNL. Will need to continue to assess closely. This is an excellent clinical example that even though VS are WNL, a DEEP understanding of pathophysiology and related assessment data recognizes that there is still a problem that must be recognized!	
RR:20	Though technically normal, in this scenario, this is on the high end of normal and a potential concern because she is at rest.
RELEVANT Assessment Data:	Clinical Significance:
CARDIAC: Pink, warm & dry, 1+ pitting edema in lower extremities, heart sounds regular-S1S2, pulses strong, equal with palpation at radial/pedal/post-tibial landmarks	1+ pitting edema represents small amount of fluid volume overload that should have provoked a larger urinary response. This is a clinical RED FLAG!
RESPIRATORY: Denies SOB, non-labored respiratory effort, breath sounds equal aeration bilaterally with faint crackles in both bases	If crackles are NEW, this is clinically significant. This is why it is so important to TREND all assessment data in the clinical setting from the most recent to current.

GU: 50 mL urine output the past 2 hours after furosemide IV given, urine clear/yellow	50 mL of urine output must be recognized as RELEVANT because it falls below the parameters of 30 mL/hr. In the context of receiving a loop diuretic, the nurse would expect a larger response unless the patient is dehydrated or volume depleted. This is also a clinical RED FLAG for possible acute kidney injury.
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2. Has the status improved or not as expected to this point?

JoAnn is sliding in the wrong direction. The combination of crackles, edema, decreased urine output, elevated creatinine, and easy fatigue indicates that a storm is brewing. If the nurse does not take action, JoAnn will have end organ damage to both the heart and the kidneys.

3. Does your nursing priority or plan of care need to be modified in any way after this evaluation assessment?

Nursing priority/priorities, ironically, will remain the same as they were upon admission:

- **DECREASE WORKLOAD OF THE HEART**
 - Small amount of fluid volume excess present, and 1+ edema is minimal, but must be noted by the nurse.
- **MONITOR CLOSELY FOR COMPLICATIONS**
 - The nurse must continue to remain VIGILANT with possible cardiac irritability/rhythm changes as well as the possibility of acute renal failure secondary to angiogram dye load.
- **PATIENT EDUCATION**
 - As JoAnn approaches discharge and is clinically stable, education and addressing any knowledge deficits becomes a higher priority.

Cardiac Telemetry Strip:



Interpretation:

Normal sinus rhythm (NSR) with frequent uni-focal premature ventricular contractions (PVCs)

Clinical Significance:

PVCs represent cardiac irritability. But unless the patient has sustained runs of ventricular tachycardia, continue to monitor and assess. PVCs also occur when there is oxygen deprivation in the cardiac tissue. Reinforce the need for the nurse to always check out K+ and Mg+ levels and to be sure these are WNL. These two electrolytes are the foundation for normal cardiac electrical conduction and if depleted will increase cardiac irritability and dysrhythmias

Two hours later...

JoAnn is resting quietly in bed. Foley catheter assessment reveals no new urine in bag from previous assessment two hours ago. Bladder scan reveals no residual urine. Review of labs reveal increased creatinine. The primary nurse gives the following SBAR to the on-call cardiologist:

Situation:

Name/age:

JoAnn Smith is a 68-year-old woman

BRIEF summary of primary problem:

No urine output the past 2 hours

Last creatinine was trending upward from 1.7 to 2.1

Day of admission/post-op #:

Day 2

Background:

Primary problem/diagnosis:

Acute MI

- Angiogram two days ago revealed an occluded proximal right coronary artery (RCA).
- She received two bare metal stents with 0 percent residual stenosis.
- She has been in the intensive care unit (ICU) the past two days and transferred out to the cardiac telemetry floor earlier today.
- She has been receiving scheduled furosemide 40 IV mg every 12 hours and had only 50 mL urine output two hours after her last dose of furosemide.

RELEVANT past medical history:

- Diabetes mellitus type II
- Hypertension
- Hyperlipidemia
- Cerebral vascular accident (CVA) with no residual

Asessment:

Vital signs:

T: 97.2 (oral)

P: 76 (regular/irregular)

R: 20 (regular)

BP: 122/58

O2 sat: 95% room air

RELEVANT body system nursing assessment data:

CARDIAC: Pink, warm & dry, 1+ pitting edema in lower extremities, heart sounds regular-S1S2, pulses strong, equal with palpation at radial/pedal/post-tibial landmarks

RESPIRATORY: Denies SOB, non-labored respiratory effort, breath sounds equal aeration bilaterally with faint crackles in both bases

GU: 50 mL urine output the past 2 hours after furosemide IV given, urine clear/yellow

TREND of any abnormal clinical data (stable-increasing/decreasing):

Last creatinine was trending upward from 1.7 to 2.1

INTERPRETATION of current clinical status (stable/unstable/worsening):

Condition is worsening

Recommendation:

Suggestions to advance plan of care:

Repeat basic metabolic panel (BMP)to recheck creatinine

Repeat furosemide IV or increase scheduled dose

The physician addresses your concern and orders a repeat basic metabolic panel (BMP) and repeat x1 furosemide (Lasix) 40 mg IV push. You obtain the following results one hour later:

Basic Metabolic Panel (BMP):	Current:	High/Low/WNL?	Most Recent:
Sodium (135-145 mEq/L)	135	WNL	132
Potassium (3.5-5.0 mEq/L)	5.9	HIGH	4.1
Glucose (70-110 mg/dL)	152	HIGH	184
Creatinine (0.6-1.2 mg/dL)	2.9	HIGH	2.1

RELEVANT Lab(s):	Clinical Significance:	TREND: Improve/Worsening/Stable:
Potassium: 5.9 BUN: 58 Creatinine: 2.9	<i>The RELATIONSHIP between the rising K+ and rising BUN/creatinine must be recognized by the nurse. As the kidneys fail, K+ is not able to be excreted and serum levels begin to rise. This also explains the rising BUN, which will also elevate in renal failure due to the inability to excrete the waste products of urea and nitrogen.</i>	HIGH...worsening HIGH...worsening HIGH...worsening
Glucose: 152	<i>Though elevated, in the context of the actual clinical problem that is clearly present, it needs to be noted but is not highly relevant.</i>	HIGH—slight improvement

Current Assessment:	
GU:	One hour post furosemide administration IV, continues to have no urine output.

1. Has the status improved or not as expected to this point?

JoAnn has experienced a declining change in status and is currently in acute renal failure with no urine output.

2. Does your nursing priority or plan of care need to be modified in any way after this evaluation assessment?

Absolutely. If this trend of minimal to no urine output continues, there will be a need to consider Continuous Renal Replacement Therapy (CRRT) or hemodialysis on a short-term basis as a bridge to recover from the injury to the nephrons due to the IV contrast dye from the angiogram. If she is dehydrated, the cardiologist may want to consider a gentle fluid bolus of 250-500 mL to see if this improves urine output. The kidneys could also be injured by a pre-renal cause—heart failure. Introduce or identify the types of acute renal failure: pre-renal, renal, and post-renal.

3. Based on your current evaluation, what are your nursing priorities and plan of care?

Your priorities are twofold—the obvious acute renal failure and developing fluid volume excess that will continue to compound with this development and its impact on respiratory and cardiac status, and the implications of the elevated K+ on cardiac conduction and rhythm stability. You will need to make the following essential nursing assessments from this point on:

- Assess response to diuretics (I&O) that will need to continue to be given. Joann will need larger doses and may even start a furosemide continuous gtt. At this time it is very important to get nephrology on the case ASAP! Do a fluid challenge to see if giving IV fluids increases urine output. May also need an inotropic agent or another class of diuretics.
- Assess respiratory status closely—especially breath sounds and O2 sat from current baseline
- Assess rhythm for tented T waves that are seen in hyperkalemia as well as increased ventricular ectopy such as PVCs or runs of VT.

Caring and the “Art” of Nursing

1. What is the patient likely experiencing/feeling right now in this situation?

In the context of this scenario, the nurse must fully involve the husband in the care of his wife and be available to her and present as much as possible. Caring for JoAnn will mean caring for him as well and being sensitive to his needs. The husband will also likely be anxious and has fears that he may or may not verbalize.

The nurse needs to put her/himself in the patient’s place to identify what is being experienced in this situation. The patient is likely aware of the seriousness of the current change in status and may be fearful and anxious. Support both the patient and family by giving them as much information about her current status and explain the plan of care from both a nursing and medical perspective. KNOWLEDGE is POWER from a patient’s perspective. When the nurse provides this information, it will DECREASE anxiety and fear and make a real difference in her well-being.

Even in the context of a patient who is critically ill, when you simply and matter-of-factly share what you are doing and why, it demonstrates the caring and support that is needed.

2. What can you do to engage yourself with this patient’s experience and show that she matters to you as a person?

Regardless of the clinical setting, remember the importance of touch and your presence as you provide care. If you are using Swanson’s Caring framework (which I encourage you to do—see my “Teaching Caring” tab on KeithRN.com), the following practical caring interventions can be “tools” in your caring toolbox to use depending on the circumstance and the patient’s needs (Swanson, 1991).

- **Comforting**
 - Little things to comfort—whatever it may be—are needed and appreciated! i.e. hand or foot massage for pain control
- **Anticipating their needs**
 - Staying one step ahead and not behind, especially in a crisis is essential! Is everything where the patient can reach it before you leave the room?
- **Performing competently/skillfully**
 - Remember that when a nurse or student nurse does their job well and competently, this demonstrates caring to the patient!
- **Preserving dignity**
 - Maintaining privacy at all times is essential and is all too easily forgotten due to pressing physical needs. Pulling the curtain as well as covering exposed genitalia is all that is required. They are little things, but so important to preserve human dignity. Accomplishing bodily functions which are disrupted with someone else present is significant. Be respectful of privacy issues.
- **Informing/explaining—patient education**
 - Even in a crisis, simply explain all that you are doing. If your patient is not able to respond but if family are present, do not forget to explain to them all that you are doing and why. This is truly the “art” of nursing and makes such a difference when done in practice!

Use Reflection to THINK Like a Nurse

Reflection-IN-action (Tanner, 2006) is the nurse’s ability to accurately interpret the patient’s response to an intervention in the moment as the events are unfolding to make a correct clinical judgment.

1. What did I learn from this scenario?

Have students share and reflect

2. How can I use what has been learned from this scenario to improve patient care in the future?

Have students share and reflect

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