JoAnn Smith

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.

Past medical history:

DM II, HTN, Hyperlipidemia, CVA with no deficits, GERD, Iron deficiency Anemia

1. **What stands out as relevant to you and needs to be considered when forming the patient's plan of care?**

**See highlighted. Her symptoms are consistent with an acute myocardial infarction (AMI) and being a smoker places her at an increased risk for an AMI.

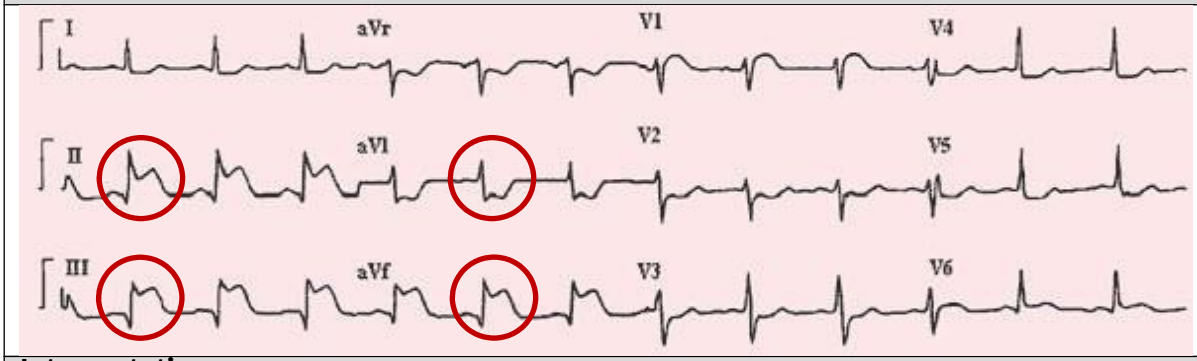
Current VS:	P-Q-R-S-T Pain Assessment (5th VS):	
T: 99.2 F/37.3 C (oral)	**P**rovoking/Palliative:	Nothing/Nothing
P: 128 **(**regular)	**Q**uality:	Ache
R: 24 (regular)	**R**egion/Radiation:	Left arm that radiates into neck
BP: 108/58	**S**everity:	5/10
O2 sat: 99% room air	**T**iming:	Intermittent-20-30"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

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

2. What findings are significant and why?

**See highlighted. Evidence of AMI persists. The coarse crackles in the lungs indicate left ventricular dysfunction – this is concerning for an emergent MI. Pulse rate is elevated, the heart is attempting to compensate for left ventricular dysfunction, which is causing a decrease in cardiac output (CO), by increasing the heart rate in increase CO. **The body will only be able to compensate this way for a short period of time.

12 Lead EKG:



Interpretation:

ST segment elevation in leads II, III and aVf with ST depression in aVl

Clinical Significance:

ST segment elevation is indicative of a STEMI, which is caused by complete occlusion of a cardiac artery, which means blood flow and oxygen are being blocked from part of the heart which will lead to necrosis of cardiac muscle. ST segment elevation in leads II, III and aVf indicate an inferior MI.

Echocardiogram Results:	Clinical Significance:
Left ventricle hypokinesis with ejection fraction (EF) of 25%	An ejection fraction less than 50% indicates poor perfusion to the body. This tells us that the heart is not pumping adequately.

Chest X-ray Results:	Clinical Significance:
Bilateral atelectasis or pulmonary edema	Atelectasis or pulmonary edema in a patient suffering from an AMI indicates left ventricular dysfunction. If the left ventricle is not pumping adequately (in this case as a result of a posterior MI causing necrosis to the left ventricle) then blood will begin to back up into the lungs since the left ventricle cannot 'receive' blood from the lungs.

Complete Blood Count (CBC):	Current:	Is result high or low? If so, why?
WBC (4.5-11.0 mm 3)	10.5	
Hgb (12-16 g/dL)	12.9	
Platelets (150-450x 103/µl)	225	
Neutrophil % (42-72)	70	
Basic Metabolic Panel (BMP):	**Current:**	**Is result high or low? If so, why?**
Sodium (135-145 mEq/L)	135	
Potassium (3.5-5.0 mEq/L)	4.1	
Glucose (70-110 mg/dL)	184	H – SNS is being stimulated which is causing an increase in glycogen
Creatinine (0.6-1.2 mg/dL)	1.5	H – decreased perfusion to the kidneys is causing acute kidney injury
Misc. Labs:		
Magnesium (1.6-2.0 mEq/L)	1.8	
Cardiac Labs:	Current:	
Troponin (<0.4 ng/mL)	1.8	H – indicates AMI
BNP (B-natriuretic Peptide) (<100 ng/L)	1150	H – indicates CHF (fluid buildup)

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

*STEMI with left ventricular dysfunction

4. **What do you expect is the underlying cause/pathophysiology of this primary problem?**

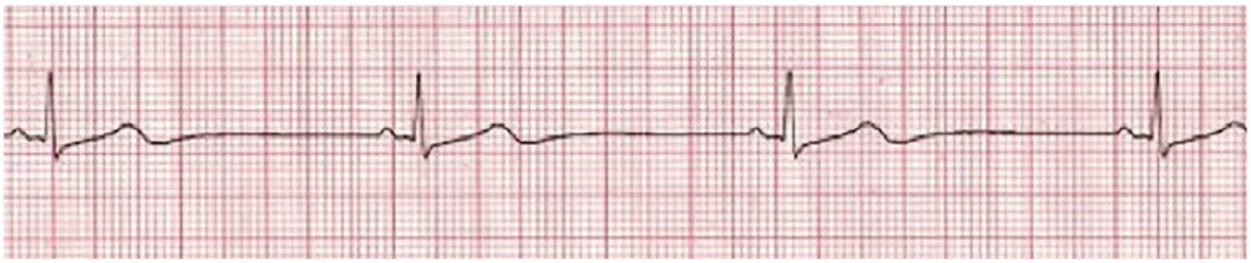
*The patient has a PMH of hyperlipidemia, HTN, DM and CVA and is a smoker. All of these problems increase the patient's risk of plaque buildup and artery narrowing. This combination places the patient at a very high risk for an AMI.

5. **What is the most crucial thing for this patient?**

*Get the patient to the cath lab so the blocked artery can be opened.

Care Provider Orders:	**Is this appropriate? Why? (Doses are fine, focus more on the drug itself, is the drug appropriate?)**
1. Establish 2 large bore peripheral IVs	1. Yes – this order is important! The health care team needs to large bore IV access for this patient, they are in an emergent situation
2. Metoprolol 5 mg IV push x1 now	2. Yes – this medication will decrease the workload of the heart; however, if the patient begins to show signs of cardiogenic shock, then we would NOT want to give this.
3. Nitroglycerin IV drip-start at 10 mcg and titrate to keep SBP >100	3. Yes – this medication will cause cardiac artery dilation and possibly allow blood flow to the area of the heart that is deficient in blood and oxygen right now
4. Clopidogrel 600 mg po x1 now	4. Yes – antiplatelet therapy is important in the patient with an AMI to decrease the chances of further clotting; the patient also needs this to prevent further clotting while the physician is working in the arteries in the cath lab; the patient has not previously received clopidogrel, so she needs the loading dose now
5. Aspirin 324 mg (81 mg tabs x4) chew x1 now	5. Yes – see above for antiplatelet therapy rationale; clopidogrel and ASA have different half-lives and work in different areas of the clotting cascade, so both medications are indicated
6. Heparin 60 units/kg IV x1 now	6. Yes – anticoagulant therapy in conjunction with antiplatelet therapy is indicated for ACS. A heparin drip is often indicated due to its short half-life and ease of reversibility; heparin works in a different spot on the clotting cascade, so is indicated in conjunction with ASA and/or clopidogrel. Heparin also works on currently circulating platelets to keep them from 'sticking together' while ASA and clopidogrel work on future platelet activation.
7. To cath lab for angiogram	7. Yes – the patient has a blocked artery that needs to be opened up

***While you are implementing orders, the patient complains of feeling dizzy. You check the monitor and see the following rhythm:**



Interpretation:
Sinus bradycardia – 40 beats per minute
Clinical Significance:
A change in heart rate from high to low would indicate in this scenario that the patient is no longer able to compensate for left ventricular dysfunction. This means that cardiac output (CO) is now too low to sustain perfusion to vital organs.

6. **What do you want to do for JoAnn? Have your previous plans changed at all?**

**We want to transport JoAnn to the cath lab STAT. Our plan of opening the blocked artery has not changed. However, since the patient is now decompensating, if we have not given the metoprolol yet we would want to ask the physician if he still wants it given since this will further decrease CO.