

**Northwest Community EMS System**  
**Continuing Education Program – April 2018**  
**ACS, ECG/12 Lead Interpretation, Dysrhythmia Management**  
**PARTICIPANT handout**  
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**References:**

**SOPs:** ACS, Bradycardia with a Pulse; Narrow QRS Complex Tachycardia; Wide Complex Tachycardia with a Pulse.

**OBJECTIVES:**

After reading the class handout, select sections of the SOPs, and referenced policies and completing the class, each participant will independently do the following with at least an 80% accuracy and no critical errors:

1. Evaluate select cases and discuss clinical findings pointing to an impression of ACS or cardiac dysrhythmias.
2. Sequence a plan of care for patients experiencing ACS, guided by assessment findings and the ACS SOP
3. Correctly identify cardiac dysrhythmias including narrow and wide QRS brady/tachyarrhythmias and AV blocks
4. Correlate the above dysrhythmias w/ assessment findings to formulate a plan of care according to NWC EMSS SOPs
5. Describe and sequence the updated vagal maneuver procedure using a 10 mL syringe
6. Explain and defend the rationale for high quality EMS 12 Lead ECG acquisition, interpretation, and transmission
7. Explain the rationale and need for serial EMS 12L ECGs in select patients.
8. Describe and adopt into consistent practice, methods to avoid or eliminate ECG artifact.
9. Accurately describe and find elements on the 12 Lead ECG that suggest acute or old injury, ischemia, or infarct:
  10. T-P segment baseline
  11. Q waves and QRS progression
  12. J point and ST segment elevation or depression
  13. T waves
  14. Reciprocal changes
15. Accurately identify acute changes on 12 Lead ECG without the aid of computer interpretation.
16. Identify ECG findings that warrant a V4R Lead configuration and repeat tracing.
17. Describe ECG findings on a V4R 12L tracing that indicate inferior wall involvement of the right ventricle.

**Introduction**

Current literature, expert opinion, and national guidelines establish benchmarks for high performing EMS and hospital practice that identify EMS assessments, interventions and reporting plus advocate for rapid activation of cardiac alerts to minimize EMS to perfusion time as pivotal in achieving optimal patient outcomes with the goal of limiting cardiac damage and preserving function and quality of life. This includes identifying a person who is experiencing ACS and accurately interpreting cardiac rhythms and 12 Lead ECGs for evidence of ischemia or infarction. Optimal care also includes rapid deployment to the cath lab with none or minimal time in the ED if there is evidence of STEMI from the EMS 12 L tracing.

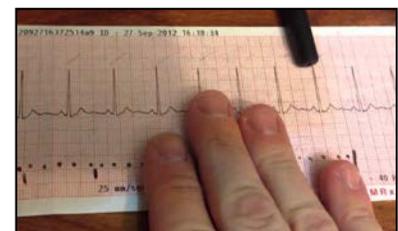
Discussion with system members, review of patient records, and NWC EMSS PBPI data reveal that we have opportunity with respect to recognizing anginal equivalents, obtaining diagnostic quality 12 L tracings without artifact, reliably transmitting those tracings to appropriate persons prior to EMS arrival at the hospital and having the hospitals act on the cardiac alert in a timely manner to consistently meet EMS to perfusion targets; understanding and executing the appropriate use of NTG and dysrhythmia management; accurately

**Reading the 12 Lead ECG for Acute Changes:**

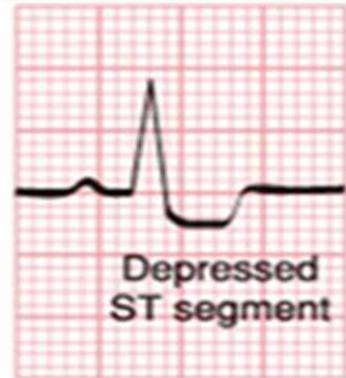
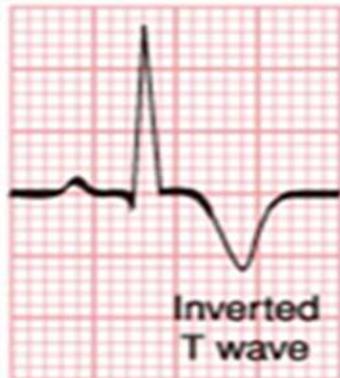
The 12 Lead gives you 2 types of interpretive information: 1) ischemia/injury/infarction and 2) rhythm. Rhythm interpretation is not reliable on a computerized 12 Lead interpretation. To detect acute changes on the 12 Lead, begin by looking at two aspects of the 12 Lead:

1. ST segments: is there elevation equal to or greater than 1mm (1 small box)?
2. All leads: is there ST elevation in two or more anatomically contiguous leads?

Following is a guide to Components of 12 Lead Interpretation.



**Ischemia: Inadequate blood supply; damage is reversible**



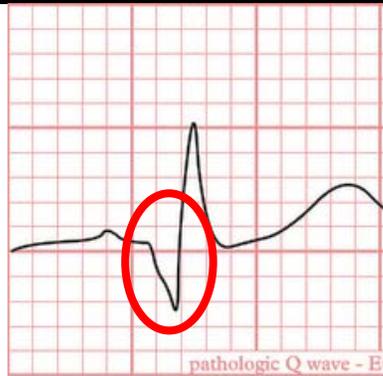
**Injury - ST Elevation**



Prolonged ischemia; reversible if flow restored quickly.  
 Diagnostic for AMI if > 1mm elevation in at least 2 contiguous

leads.

**Infarct: Q Wave**

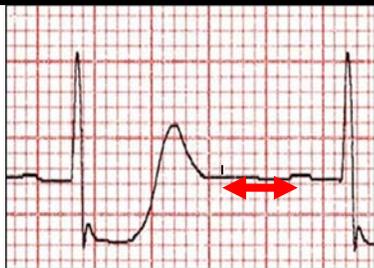


Myocardial cell death. Irreversible.

Diagnostic for MI of indeterminate age.

Measures > 1 small box wide &/or 1/3 amplitude of QRS.

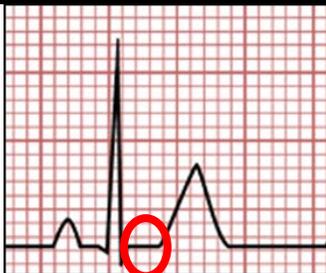
**T-P Segment**



Portion of the ECG from the end of the T wave to the beginning of the P wave.

Used as a reference to determine whether the ST segment is elevated or depressed.

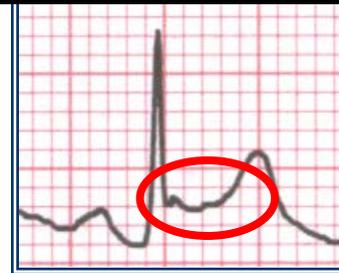
**S-T Segment**



End of S wave to beginning of T wave



ST Depression  
 Pathological in V1, V2. Represents ischemia or reciprocal changes.



ST Elevation  
 1-2 small boxes above isoelectric line. Usually occurs in first 1-2 hrs after onset of myocardial hypoxia.

### J Point



QRS complex ends and ST segment begins. Often a “range” if ST is sloped, slurred, etc.

Difficult to identify if HR is very fast (complexes close together).

Measure for ST depr / elev **ONE SMALL BOX TO THE RIGHT** of the J point!



### Determining Infarct Age: Q Waves

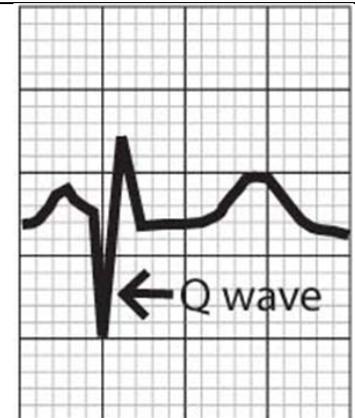
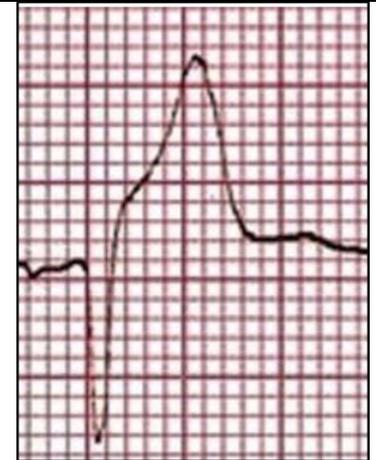
Myocardial infarction occurs in a continuum, beginning with myocardial ischemia, ending in myocardial cell death. Once the infarct is complete, a chronic or old infarct ECG pattern develops. The ST segment returns to baseline, the T wave becomes upright again, and if there was a Q wave, it may or may not remain permanent. This process may take several weeks.

The term **“age undetermined”** occurs frequently on 12 Lead ECG’s. This term means that the timing of the MI is not known, but there is no acute process occurring presently (it is not an “acute MI”). **When a Q wave is present on a 12 Lead, with no other ECG changes to indicate acute ischemia/injury/infarct, you will see the notation “age indeterminate”** because the changes both on ECG and in the myocardium that would allow you to time/date the damage have already occurred.

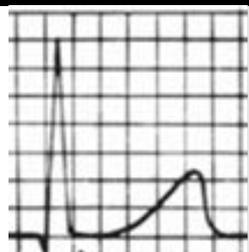
Q waves are classified as either) physiologic (normal) or pathologic (abnormal).

**Physiologic Q waves:** A physiologic Q wave is merely a representation of the first vector of ventricular polarization. It is small and thin. Normal Q wave size is < one small box/0.04 seconds duration and < 1/3 amplitude of the R wave in limb leads. A physiologic Q wave may actually simply be a QS wave, if there is no intervening R wave (positive deflection) to break up the 2. If isolated to only lead V1, the Q wave is benign.

**Pathologic Q waves:** This Q wave represents depolarization of the injured myocardium overlying infarcted tissue via slow, cell – to – cell impulse transmission. In the presence of a pathologic Q wave, the age of an MI is considered “indeterminate” unless there is accompanying evidence for an MI (ST elevation and T wave inversion) or symptoms suggesting ACS/AMI. In limb leads, this Q wave measures > one small box/0.04 seconds duration and at least 1/3 amplitude of the R wave. Both criteria won’t always be present. Width is more specific for serious pathology. Not all MI’s have Q waves (Non-Q wave MI). Why? The infarct may not have been transmural (affecting all layers of the myocardium), or it may have occurred in a “silent” area of the heart (no electrical activity = ECG cannot record). Of those infarctions that do develop Q waves, a significant number do not persist. A pathologic Q wave will not appear until the infarct has resolved.



### T Waves



The normal T is deflected in the same direction as the QRS.

Should be slightly asymmetrical, slightly rounded, the peak is closer to its end than to its beginning, and the first half has a more gradual slope.

“Flipped”, tall and symmetrical T waves are often associated with ischemia. If the T wave is more than 2/3 the height of the R wave, it is abnormal

## High Quality ECGs and Artifact

Steps to obtaining a high quality ECG:

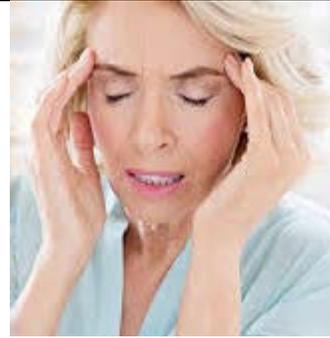
- Proper skin prep: rub briskly with a dry towel to remove old, dry skin cells
- Excess hair can be clipped, or removed by firmly applying a pair of therapy pads and briskly removing them
- Use a fresh set of pads
- Dry diaphoretic skin
- Store electrodes in the foil packs, unopened, so gel is fresh and pliable
- Electrode gel must actually penetrate the skin. Press around the entire edge of each electrode
- Leads must be properly placed. Improper chest lead placement is the most common cause of variability in an ECG
- Limb leads should be placed on limbs, at least 10 cm from the heart
- Place electrodes on flat, fleshy parts of arms & legs; avoid bony areas and major muscles
- Position patient supine if possible (If not, this should be documented on the ECG)
- Support limbs if necessary to avoid muscle tension / tremors (artifact)
- **Artifact:** Poor skin prep is the most common cause of artifact. Artifact can produce a poor quality, "unclear", ECG. A diagnosis based on an inaccurate ECG could lead to inappropriate, unnecessary, and perhaps detrimental treatment. **Steps to eliminate artifact:**
  - (1) change the lead (for monitoring)
  - (2) monitor away from other equipment / do not cross lead wires over equipment or ventilator tubing
  - (3) warm a shivering patient
  - (4) position the patient comfortably.

## Overview of Infarcts

Location of Infarct	Arterial Supply	Indicative Changes	Reciprocal Changes
<b>Anterior</b>	LAD	V1-V4	II, III, aVF
<b>Inferior</b>	RCA	II, III, aVF	I, aVL
<b>Lateral</b>	Circumflex	I, aVL, V5, V6	V1
<b>Posterior</b>	Posterior Descending (RCA)	None	V1, V2
<b>Septal</b>	Septal Perforating (LAD) Posterior Descending (RCA)	Loss of R wave in V1, V2, or V3	None

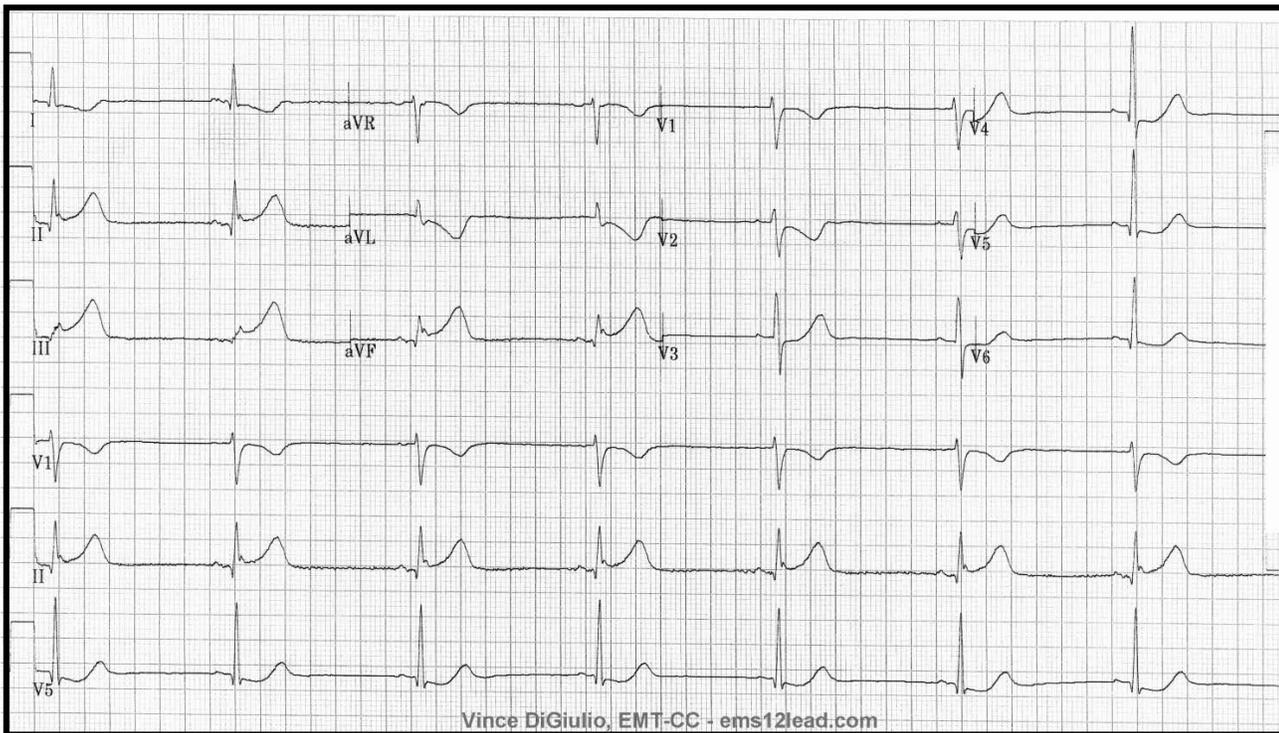
Scenario #1

It is a sunny, 66° day. You respond for a patient with weakness and lightheadedness. You find your 68 y/o female patient on the sofa in the golf course locker room, eyes closed, leaning her head against the back of the sofa. Her friends tell you they drove her in from the 2nd tee when she began to feel like she was going to faint. She did not fall or lose consciousness. The patient is pale, cool and moist. She is awake and answers your questions appropriately, but keeps her eyes closed when not asked to open them. Her breathing is unlabored. She denies chest pain, SOB, or nausea. She denies allergies. Meds: ASA 81 mg, losartan. PMH: HTN. Her radial pulse is very weak and very slow. Lungs are clear. Your partner gets VS while you attach the monitor.



What is this patient's rhythm? \_\_\_\_\_

VS: BP 88/60, HR 40, RR 18, SpO2 on RA 95%, ETCO2 is 32. 12 Lead as follows:



What do you see on this 12 Lead? \_\_\_\_\_

How will you treat this patient?

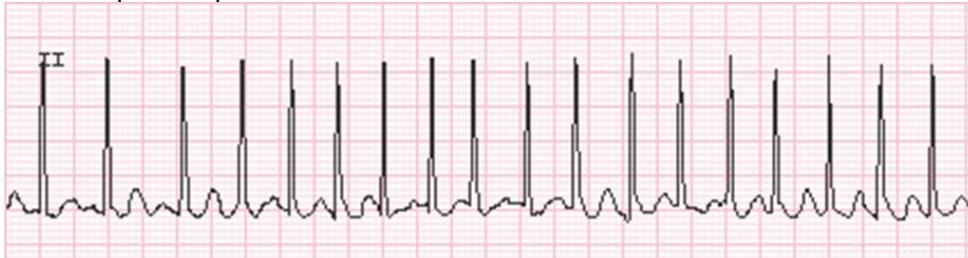
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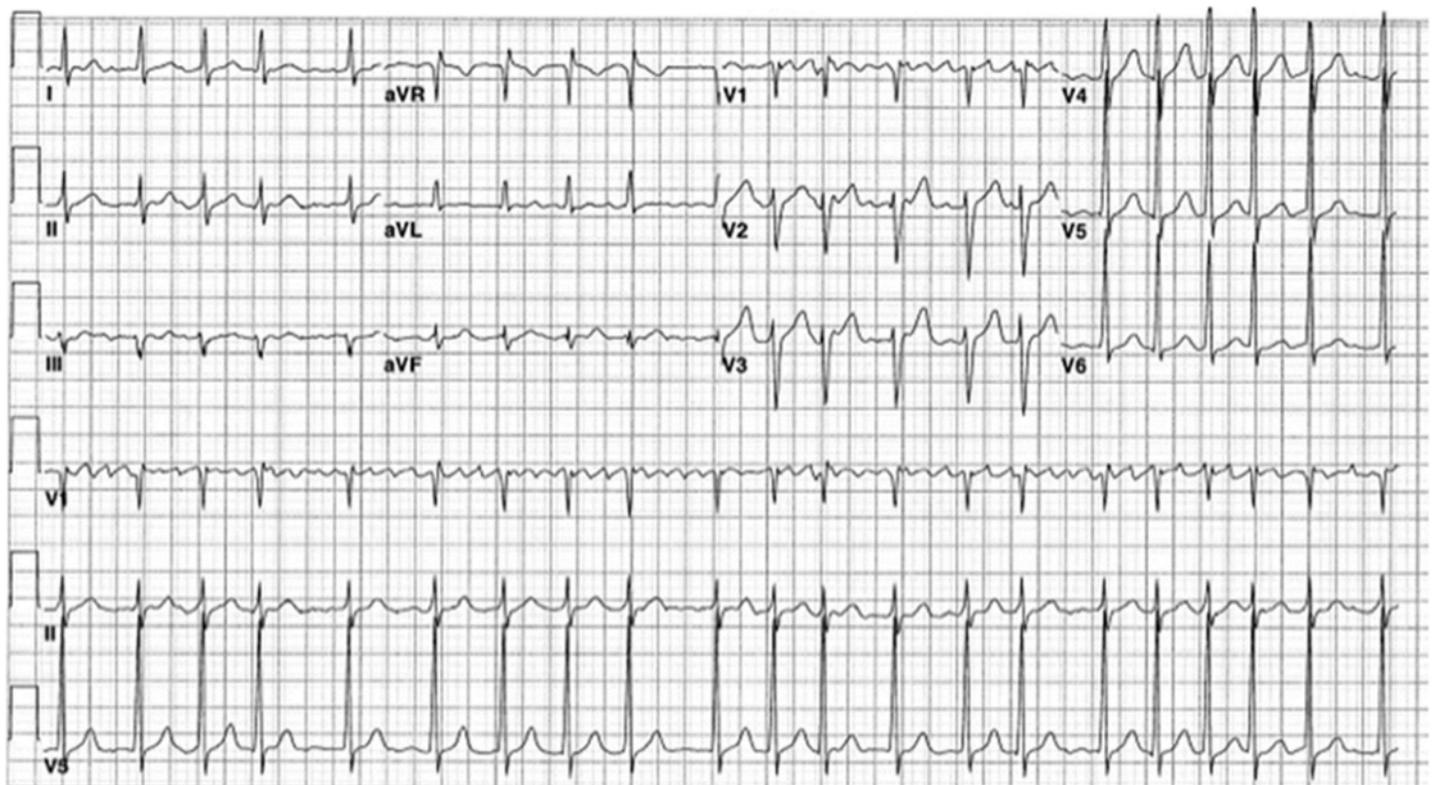
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Scenario #2: You are dispatched for a 48 y/o man with pounding in his chest and chest heaviness. You find him lying on the bed in a hotel room. His wife says they just returned to their room when he suddenly said he didn't feel good and needed to lie down. The patient is alert and answers questions appropriately. He speaks in full sentences and his breathing appears unlabored. His skin is diaphoretic but warm. You attempt to count his radial pulse but it is too fast. Lungs are clear. Your coworker puts the pt on the monitor:



What is your rhythm interpretation for this patient? \_\_\_\_\_

VS: BP 100/66, HR 166, RR 18, SpO2 96%, ETCO2 34. He rates his discomfort as 6/10. His wife states the pt has no PMH, no allergies, is a non-smoker, and takes no meds except Viagra and some new diet pill. His 12 Lead follows:



What do you see on this 12 Lead? : \_\_\_\_\_

How will you treat this patient? \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

When approx. half the verapamil is given, the pt becomes slow to respond. Skin is cool and clammy. VS: BP 88/60, HR 172, RR 12, SpO2 93%, ETCO2 32, square. What action is indicated?

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The pt's rhythm changes to

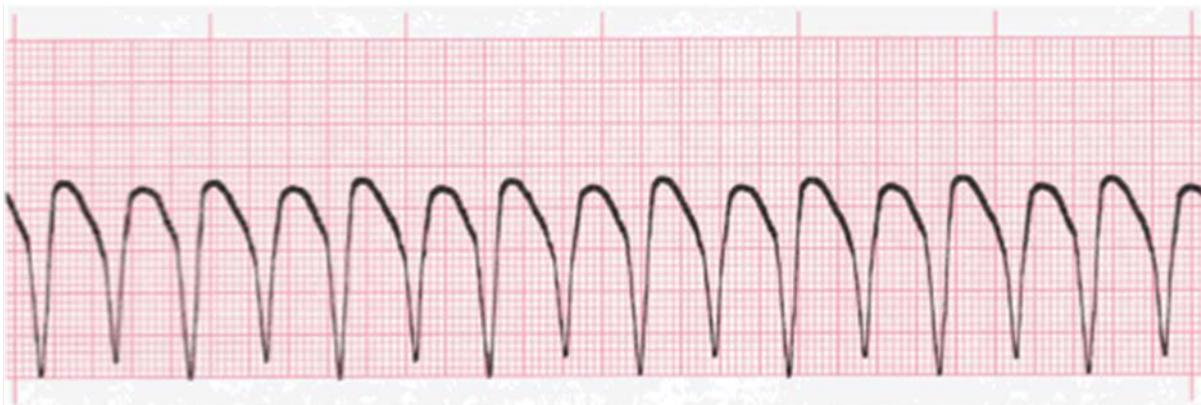


He becomes more alert and says he feels much better – the chest heaviness is gone and his heart is no longer pounding. He denies difficulty breathing, lightheadedness, nausea, dizziness. VS: BP 136/80, HR 76, RR 16, SpO2 97%, ETCO2 37.

What should be done before transport?

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**Scenario #3:** You respond for a 55 y/o patient with chest pain. You arrive at the pt's residence and find the pt sitting at the kitchen table. He is grimacing, and leaning forward w/ his hand on his chest. He says he has terrible chest pain and can't catch his breath. His wife says he walked into the house from the garage where he was sanding boards for a project, and he was gasping and leaning against the wall as he came into the house. She tried to give him some cold water but he won't drink it. As you approach him he says he might pass out. You move him to the floor, supine, and notice that he is very diaphoretic and pale. You touch his wrist to check a radial pulse and note that it is very fast and weak. He is placed on the monitor and you see this in Lead II:



What is your rhythm interpretation? \_\_\_\_\_

History is as follows: Allergies: none. Meds: Norvasc, Flomax. PMH: HTN, palpitations, BPH. Last oral intake: 3 hrs ago. Events: standing in the garage using electric sander. O: Rapid onset; P: sitting down (better); Q: squeezing, constant; R: stays in mid chest, no radiation, has never happened before; S: 10/10. T: 10 min ago.

How will you treat this patient and what SOP will you follow?

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Here is the 12 Lead. What do you see? \_\_\_\_\_



How will you treat this patient?

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His rhythm now is as below: What is your interpretation? \_\_\_\_\_



The pt opens his eyes and when asked, says his chest discomfort is gone and his breathing is OK now, but he still feels a little light headed. Skin is pale but warm and dry now. Radial pulses are irregular but stronger compared to that on arrival. Lungs are still clear.

VS: BP 102/84, HR 86, RR 18, SpO2 97% on O2, ETCO2 38, square. Discomfort rated 0/10 now. His repeat 12 Lead is shown below. What is your interpretation of this 12Lead?

