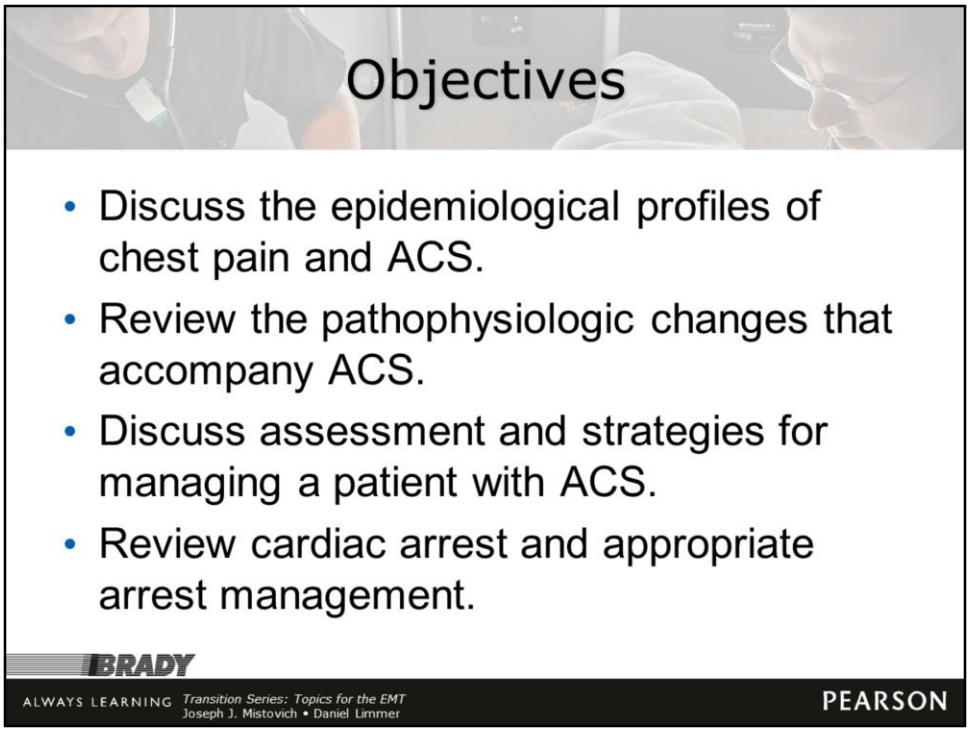
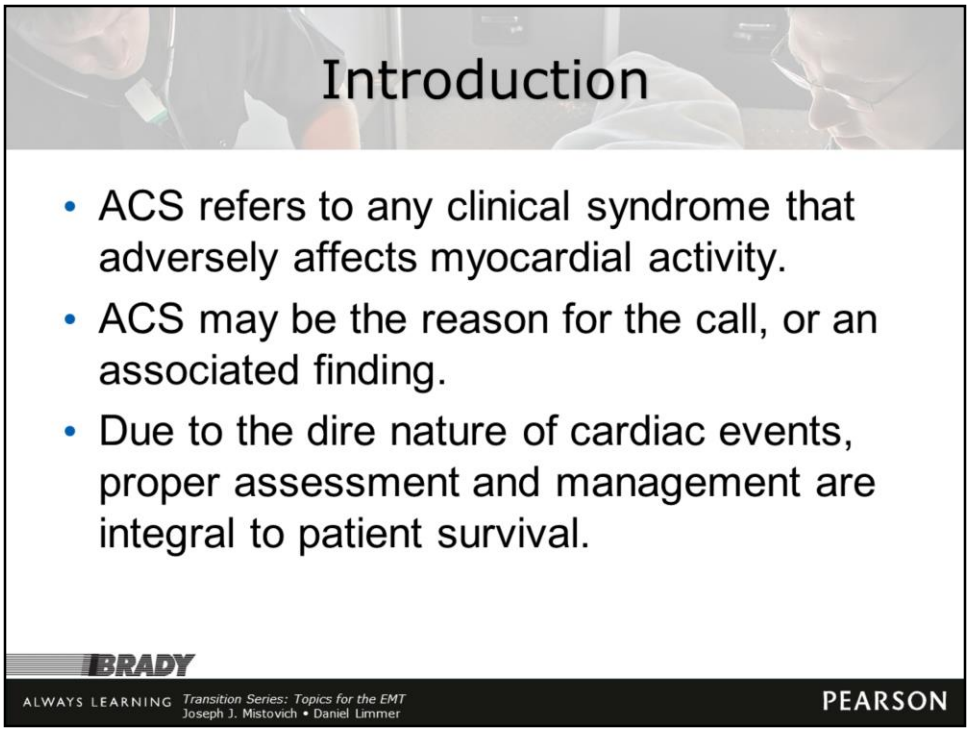

BRADY
TRANSITION SERIES
TOPICS FOR THE EMT
TOPIC 24
Cardiovascular Emergencies: Chest Pain and Acute Coronary Syndrome
ALWAYS LEARNING
PEARSON



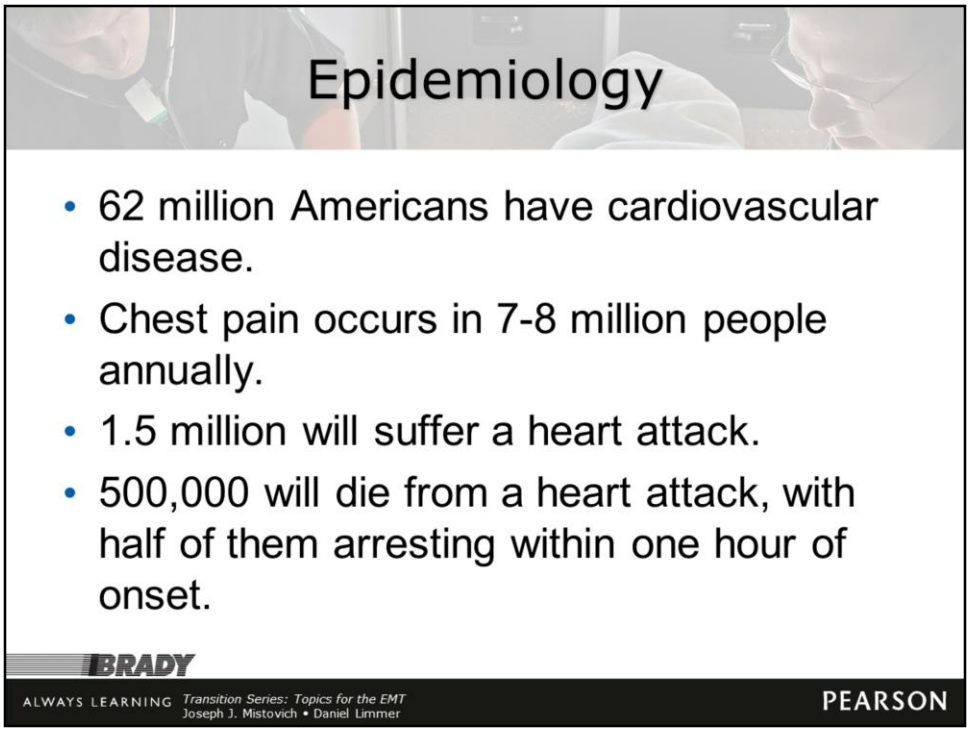
Discuss objectives.



Discuss how ACS is an umbrella term for most anything that adversely affects the heart.

It may be the precipitating cause, or an associated finding due to failure of another body system.

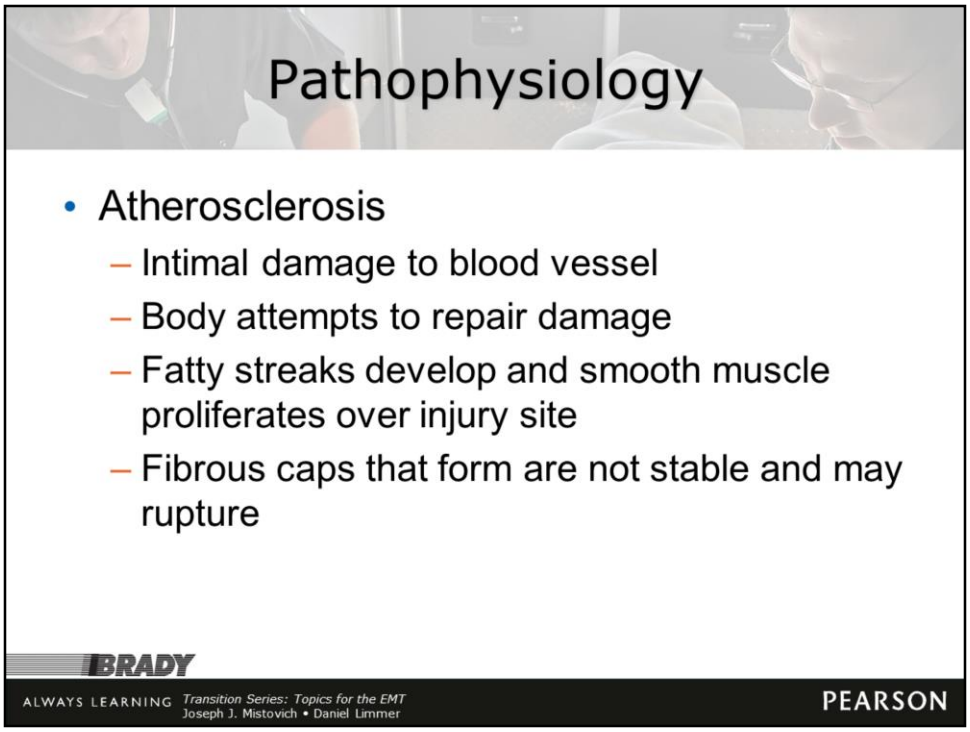
It's better to prevent arrest from occurring, rather than trying to stop a failed heart.



Review statistics.

In a more sobering light:
•About every 25 seconds an American will suffer a coronary event
•Every 34 seconds a heart attack will occur
•About once every minute someone will die from sudden cardiac arrest

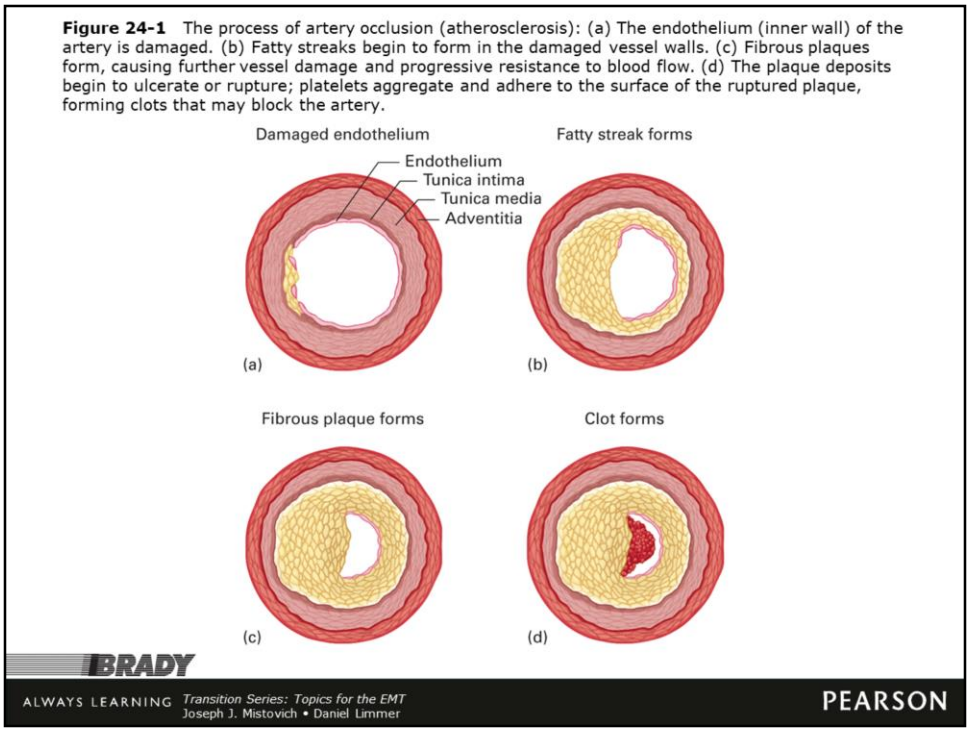
These statistics are definite indications of the significance of cardiac-related emergencies and underscore the importance of having a thorough knowledge base regarding cardiac emergencies.



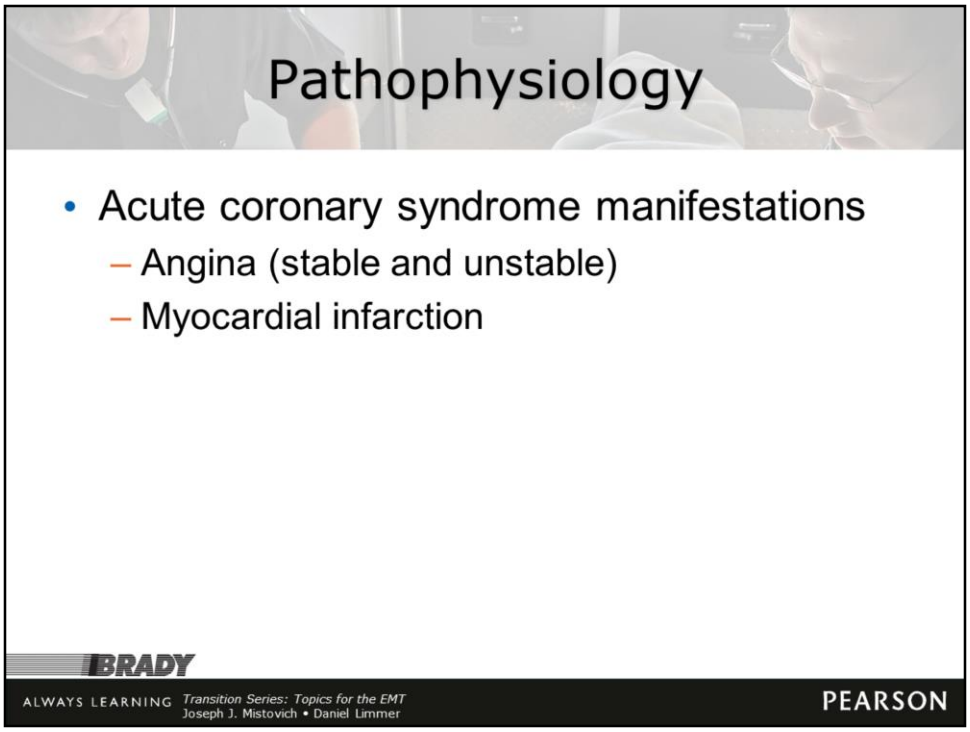
Discuss the atherosclerotic changes to a blood vessel that results in lumen deterioration and closing.

Eventually fibrous cap breaks, allowing the clotting mechanism to form a clot there which occludes the blood vessel.

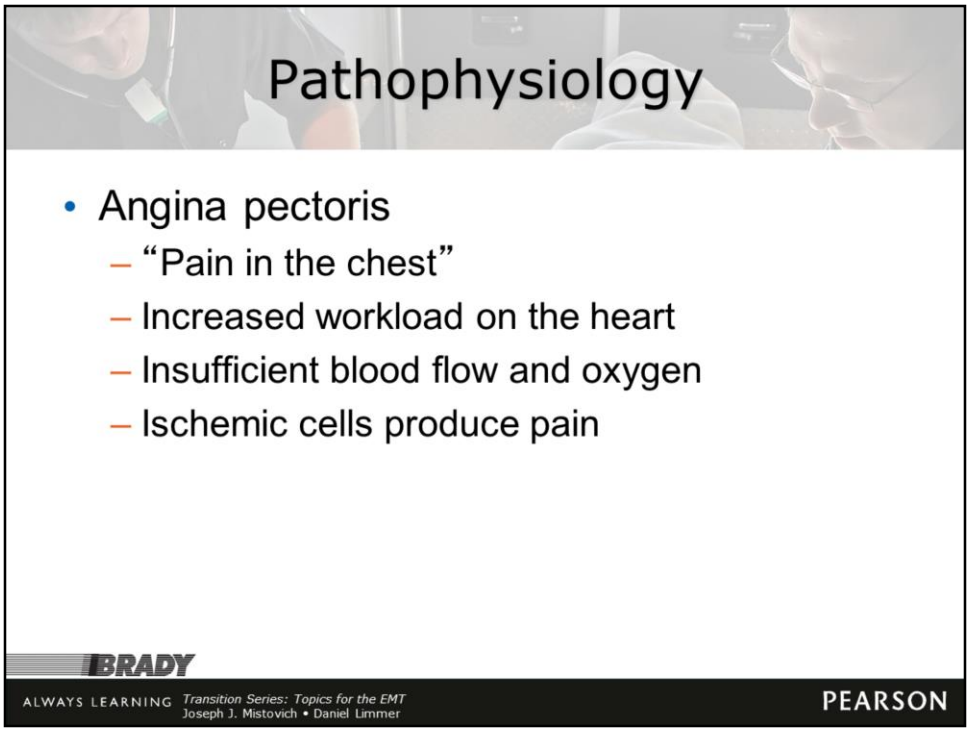
This can happen anywhere in the body, when it occurs to the coronary arteries, it creates an ischemic or infarction of myocardial muscle.



Discuss as needed.

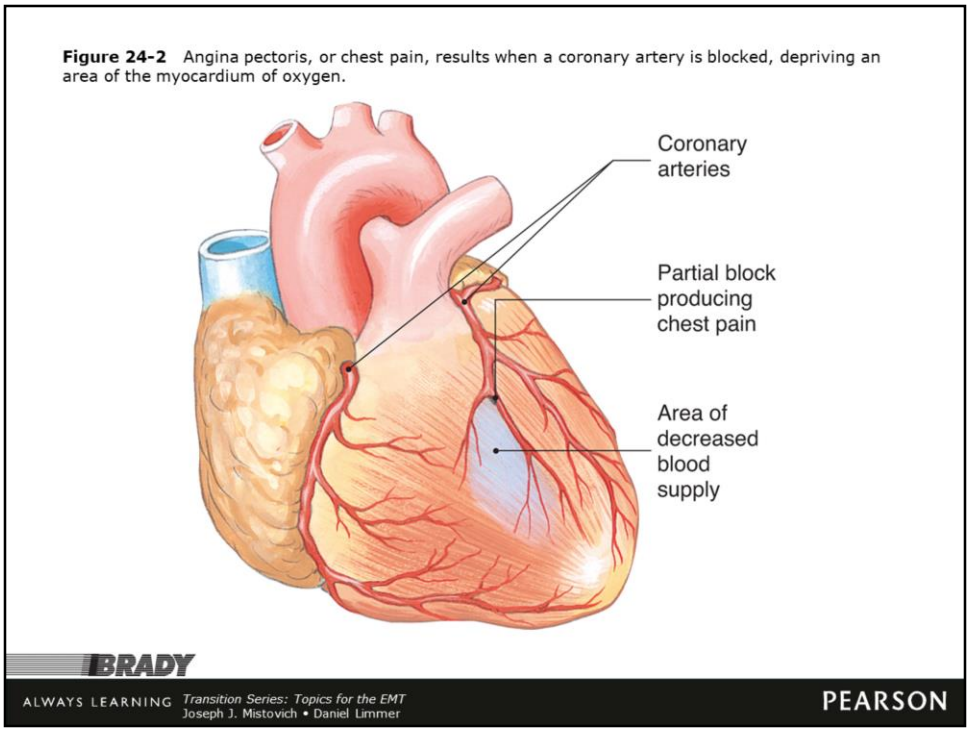


Review ACS as it relates to the two common cardiac emergencies:
•Ischemia
•Infarction



Discuss the pathophysiology of oxygen supply and demand mismatch causing chest pain.

If stress on heart is not reduced, or blood flow increased, the ischemic cells may start to die.



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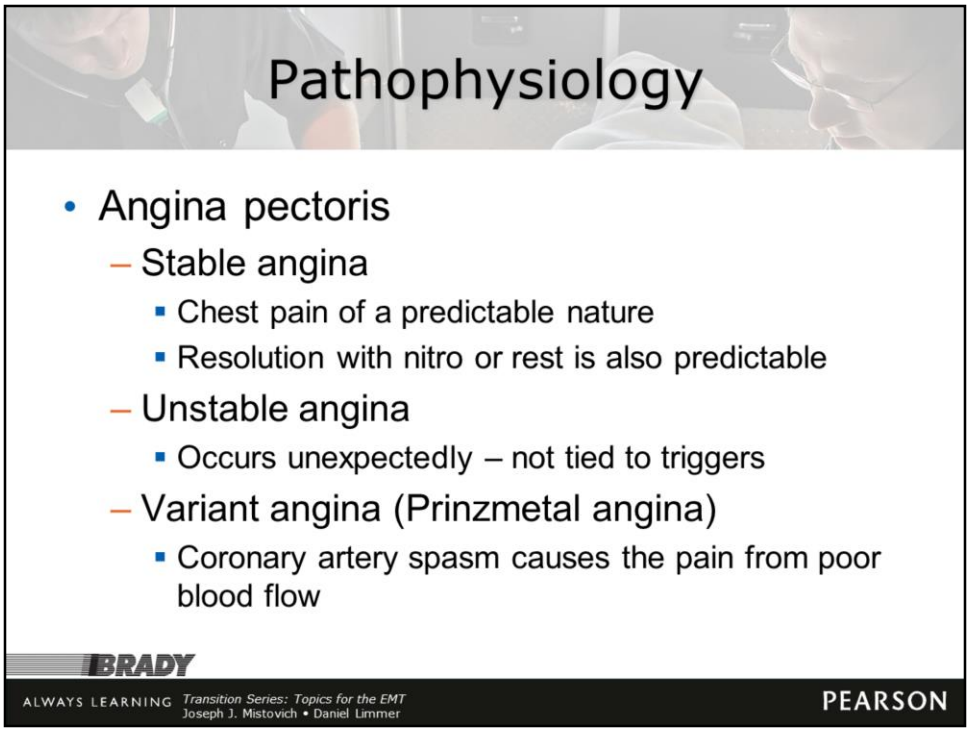
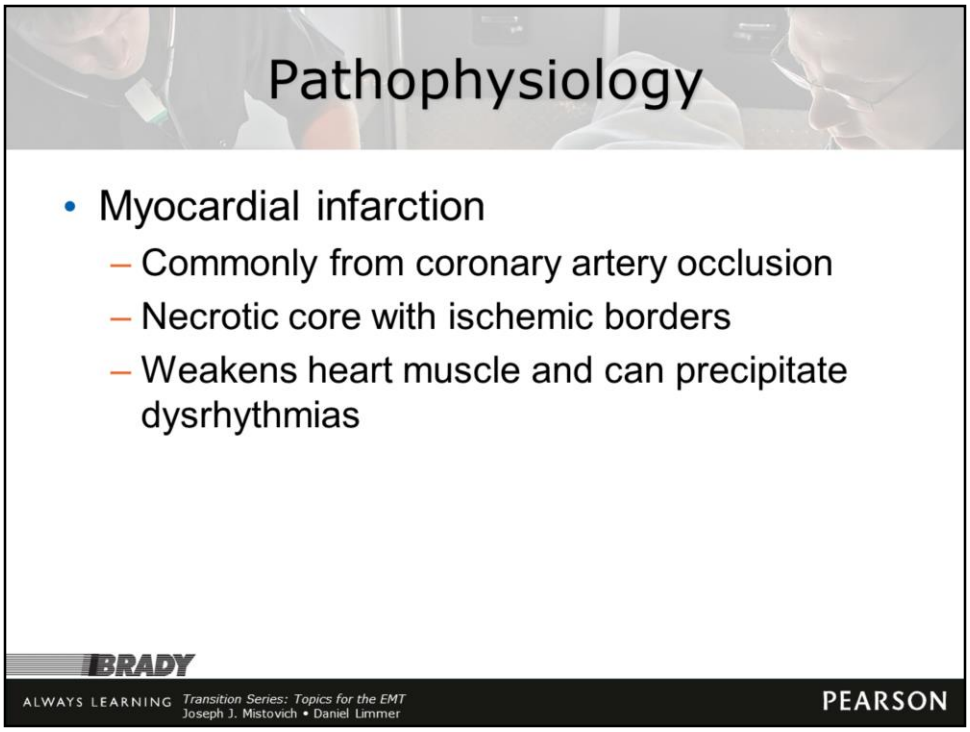


Overview and explain as needed the variants of angina:

- Stable—has predictable pattern and resolution
- Unstable—no predictable pattern, harder to resolve
- Variant angina—highly correlated with lethal dysrhythmias, MI, and sudden cardiac death



Overview and explain as needed the pathophys behind and MI:

- Cellular hypoxia
- Cellular death
- Diminished pumping action of the heart, conductiond defects, dysrhythmia formation

Figure 24-3 (a) Cross section of a myocardial infarction.

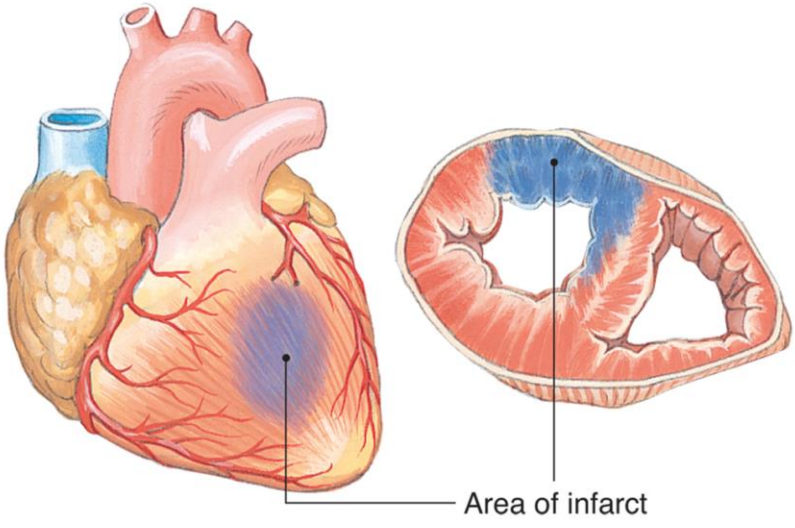
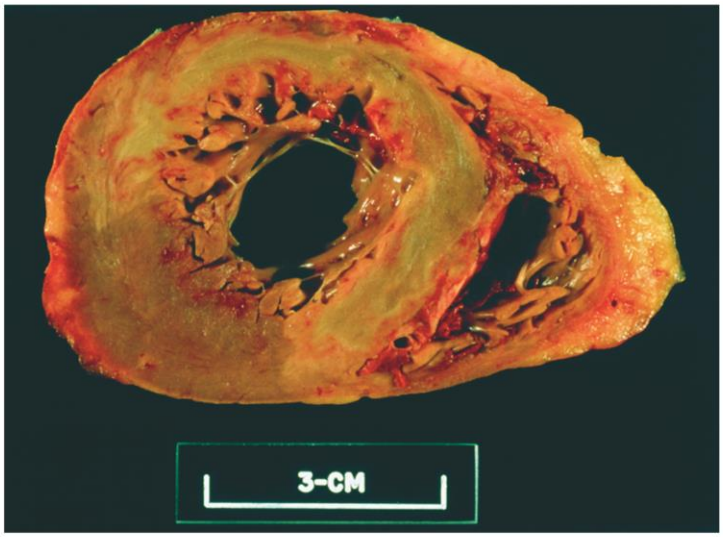
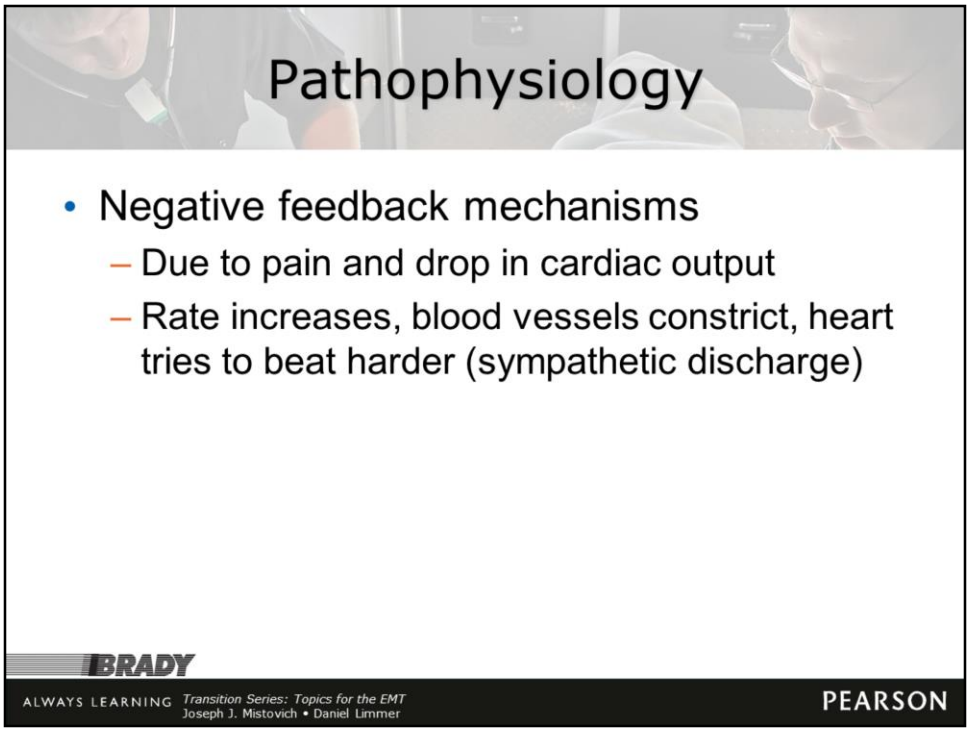


Figure 24-3 (b) A heart with normal and infarcted tissue.





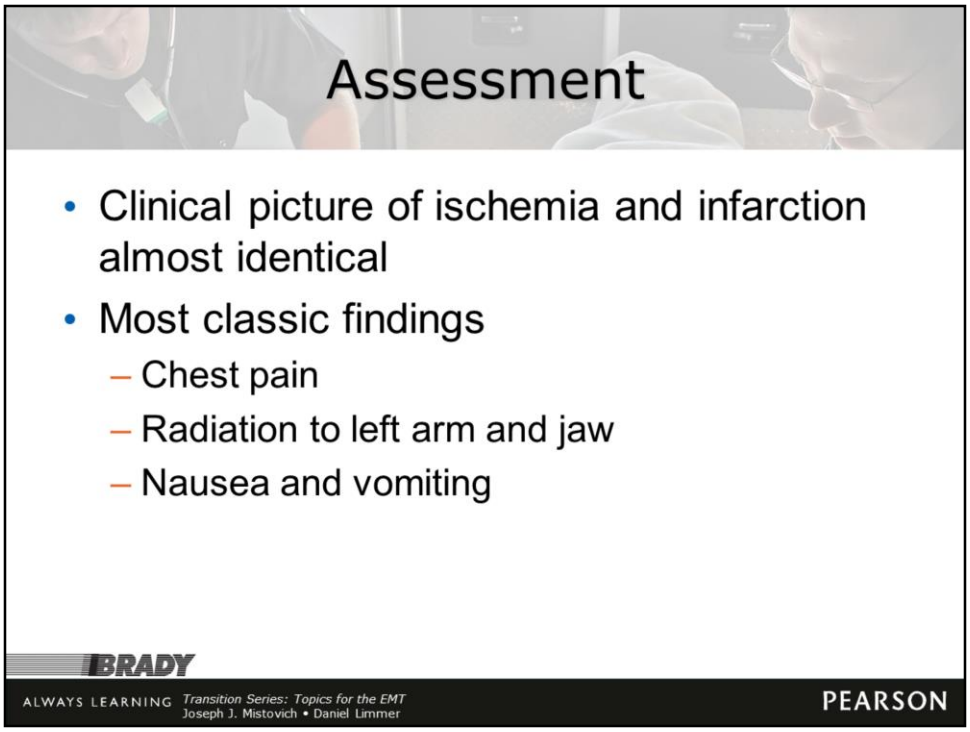
These negative feedback mechanisms cause the heart to work harder, thus increasing cardiac output.

The retention of fluid by the kidneys drives up pressure by placing more fluid into the vascular system (explaining the elevated blood pressure and tachycardia seen in many MI patients).

The pain and/or anxiety associated with a myocardial infarction further stimulates the sympathetic nervous system and results in ongoing peripheral systemic vasoconstriction and cardiac stimulation as catecholamines (such as adrenaline, norepinephrine, and dopamine) are released from the adrenal medulla.

Table 24-1 Effects of Myocardial Infarction on Body Systems

Neurohumoral effects	• Enhanced sympathetic tone • Increased circulating catecholamines • Higher levels of angiotensin II and aldosterone • Increased arginine vasopressin
Cardiopulmonary effects	• Tachycardia • Dysrhythmias • Diminished stroke volume • Increased oxygen requirements • Pulmonary vascular congestion
Peripheral vascular effects	• Increased vascular resistance (vasoconstriction) • Elevations in blood volume • Possible systemic edema



Gathering a thorough history and physical exam is the foundation of assessment in patients with suspected myocardial ischemia or infarction.

It is also important for the EMT to note that in some patients, in the absence of diagnostic tests performed by ALS providers or in the emergency department, the clinical picture of ischemia versus infarction may be nearly identical.

As such, always assume the worst and treat the patient as if the acute coronary event with chest pain is actually an infarction.

Figure 24-4 Both myocardial infarction and less serious angina can present symptoms of severe chest pain. Treat all cases of chest pain as cardiac emergencies.

DISTINGUISHING ANGINA PECTORIS FROM MYOCARDIAL INFARCTION

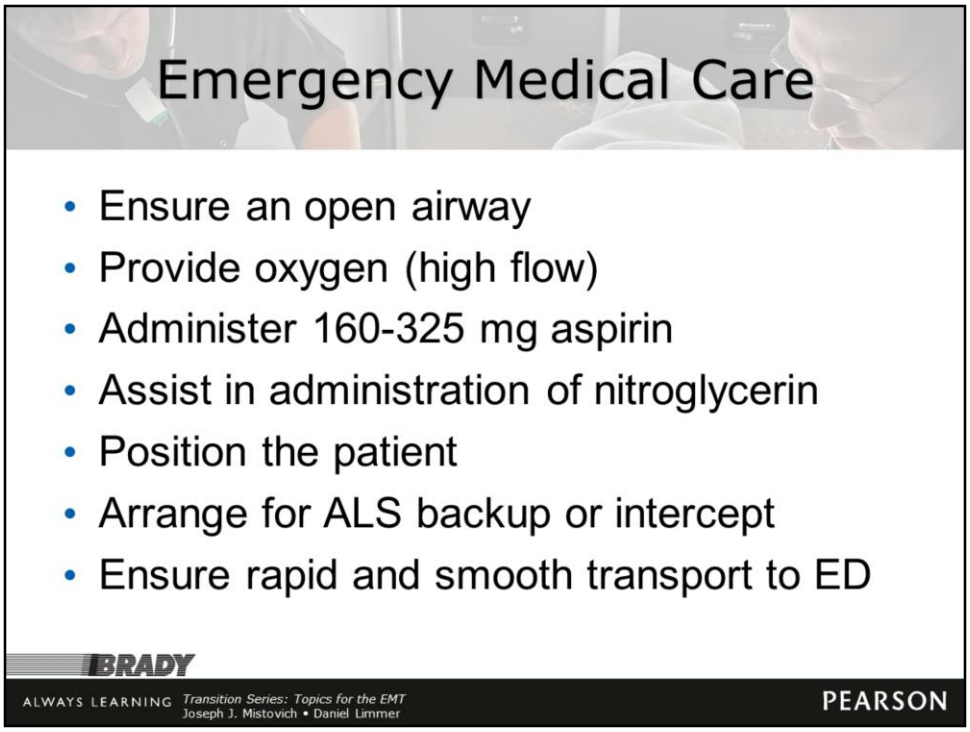
	Angina Pectoris	Myocardial Infarction
Location of Discomfort	Substernal or across chest	Same
Radiation of Discomfort	Neck, jaw, arms, back, shoulders	Same
Nature of Discomfort	Dull or heavy discomfort with a pressure or squeezing sensation	Same, but maybe more intense
Duration	Usually 2 to 15 minutes, subsides after activity stops	Lasts longer than 10 minutes
Other symptoms	Usually none	Perspiration, pale gray color, nausea, weakness, dizziness, lightheadedness
Precipitating Factors	Extremes in weather, exertion, stress, meals	Often none
Factors Giving Relief	Stopping physical activity, reducing stress, nitroglycerin	Nitroglycerin may give incomplete or no relief

TABLE 24-2 Symptoms in Women with Cardiac Ischemia or Infarction

- "Classical" findings (not necessarily *common* findings)
  - Dull substernal chest pain or discomfort
  - Dyspnea or respiratory distress
  - Nausea, vomiting
  - Diaphoresis
- "Nonclassical" or "atypical" findings (not necessarily *uncommon* findings)
  - Neck ache
  - Pressure in the chest
  - Pains in the back, breast, or upper abdomen
  - Tingling of the fingers
  - Unexplained fatigue or weight gain (water weight gain)
  - Insomnia

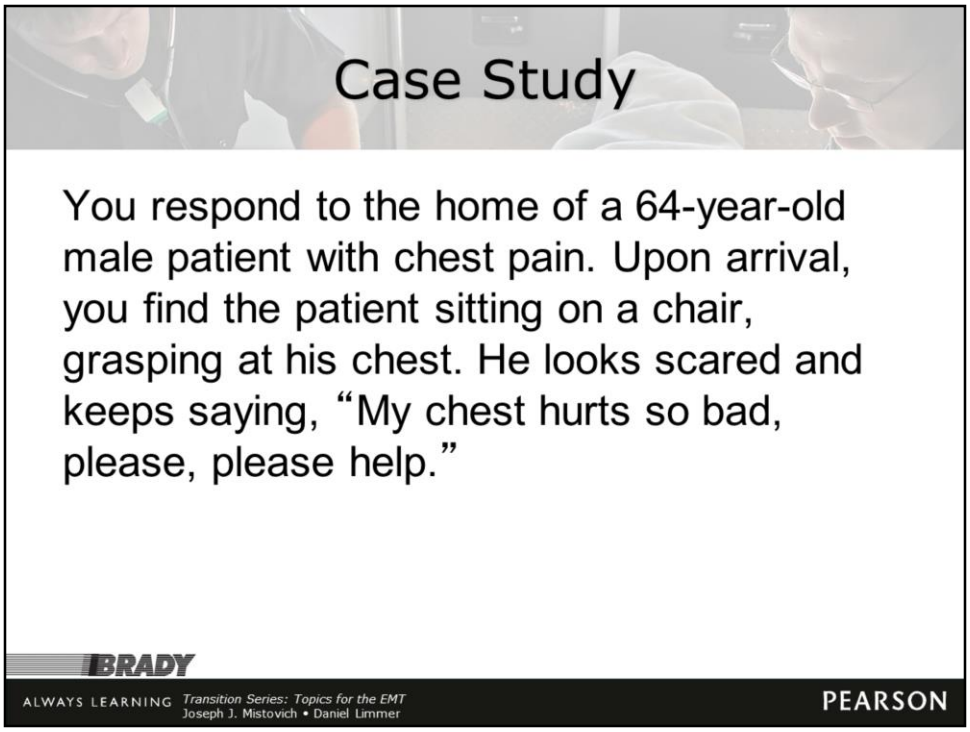
Table 24-3 Special Considerations in Geriatric Cardiac Events

History of diabetes mellitus	A geriatric patient with diabetes has long-term damage to the nerve endings in the body. This causes the typical pain from an MI to be perceived poorly, if at all, by the diabetic patient. Therefore, the diabetic patient experiencing an MI may complain only of respiratory distress or dizziness when standing, or even excessive weakness and dyspnea on exertion. It is important for the EMT to identify the patient with diabetes as potentially having an acute coronary event and to treat him appropriately. Contact ALS early, follow your local protocol, and ascertain whether additional or alternative therapies are desired by medical direction.
History of trauma	If the geriatric patient is a trauma patient, there must be a high index of suspicion for cardiac involvement as well. Geriatric patients who are traumatized can slip quickly into cardiac arrest and do not respond well to typical interventions. Geriatric patients with head trauma, chest trauma, abdominal trauma, or extremity trauma with severe bleeding are especially susceptible to cardiac arrest.
History of asthma	If a patient with a history of asthma goes into cardiac arrest, the cause may be acute bronchoconstriction that led to hypoxemia, acidosis, and cardiac arrest. Until the bronchoconstriction is reversed, the patient will not regain a pulse or start to breathe again. Early intercept or backup by an ALS unit will allow the administration of medications that may help reverse this condition.
History of COPD	Elderly patients commonly have some form of COPD (emphysema or chronic bronchitis). The arrest may have been caused by an exacerbation of the COPD, which led to hypoxemia, acidosis, and then arrest. ALS backup is needed during the resuscitation of this patient. Remember also that COPD disorders can weaken the lung tissue and cause the development of a pneumothorax and collapse of the lung. (This too may precipitate a cardiac arrest.) Be alert for the presence or the development of a pneumothorax during positive pressure ventilation, which may cause a bleb on the lung tissue to rupture.

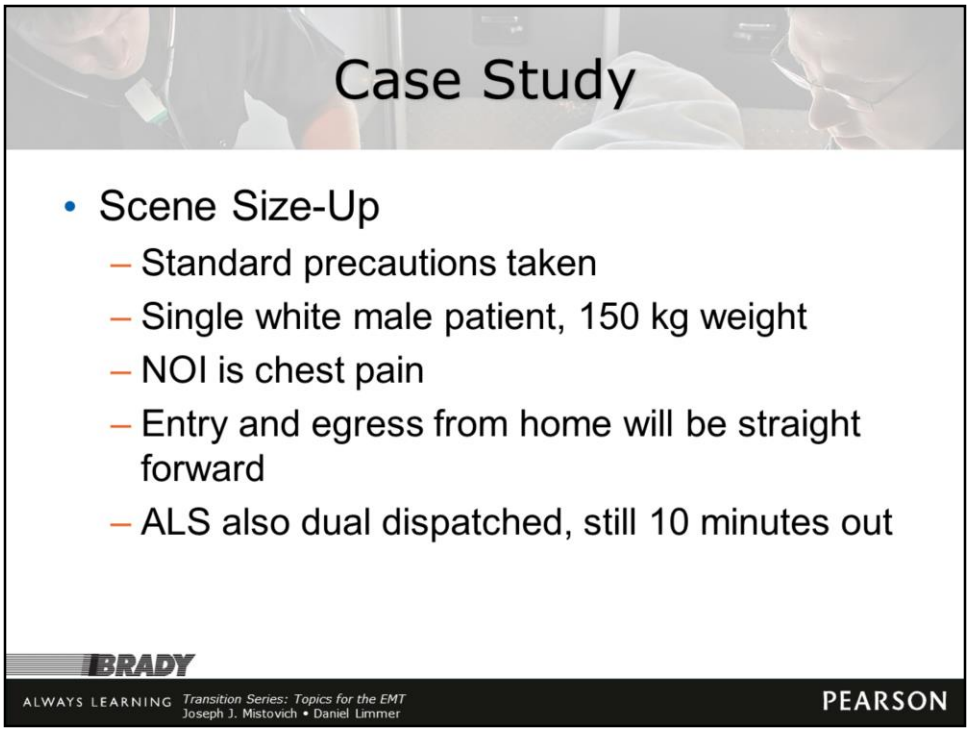


The treatment for the patient with angina pectoris or MI is geared toward:
- Ensuring patient comfort
- Improving oxygenation to the myocardium
- Diminishing enlargement of the injured area
- Preserving normotension

Remember also that ischemia and infarction may at times be impossible for the EMT to delineate in the field.



Discuss presentation.



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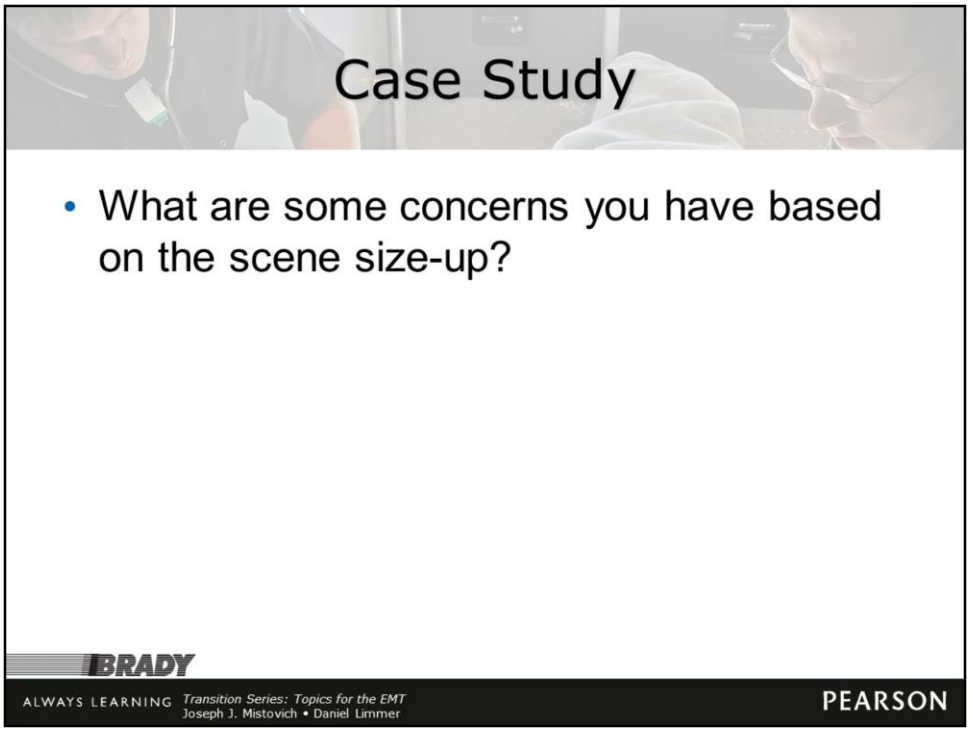
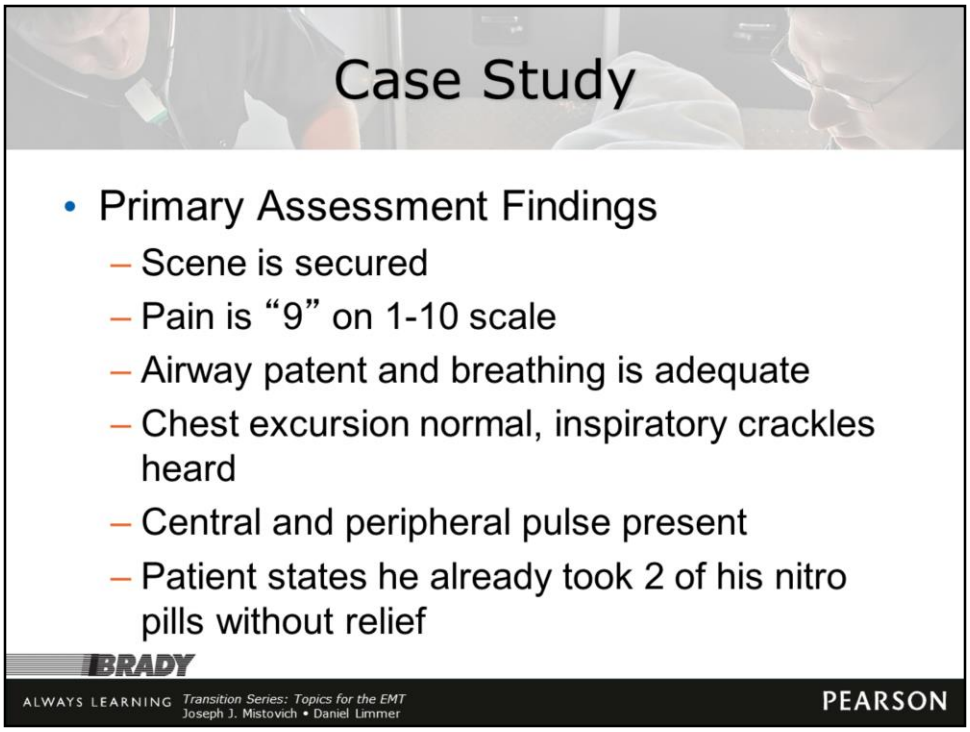


From the limited information thus far, the patient seems to have an intact airway, and is breathing adequately enough to support speech.

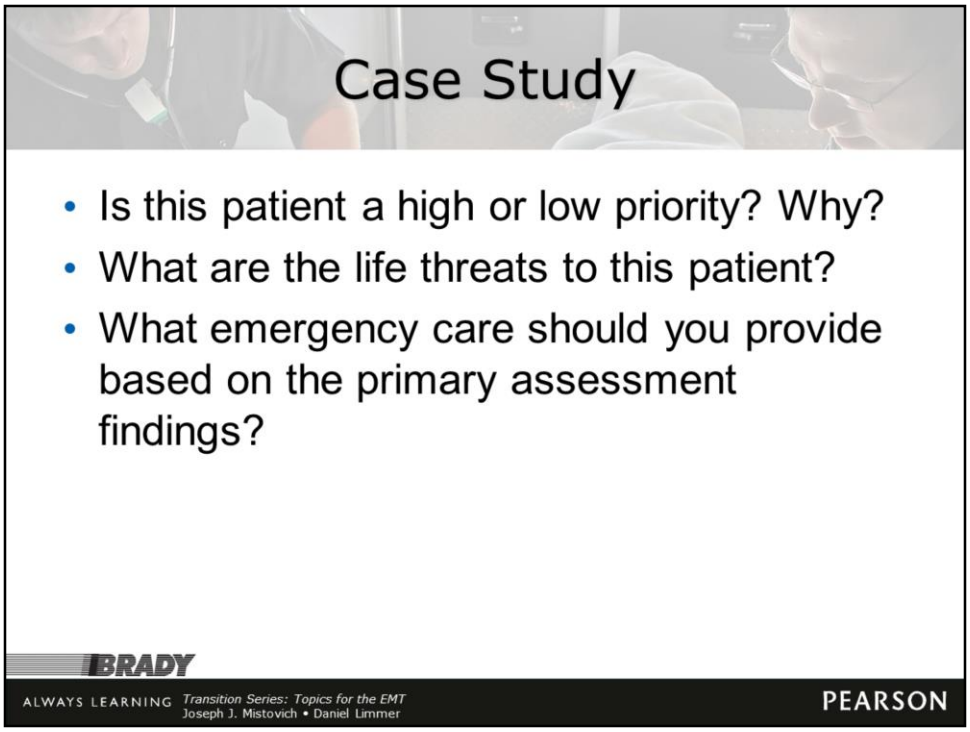
The patient must have a pulse and blood pressure as if they are conscious.

The greatest concern regards what type of event the patient is experiencing that is causing chest pain.

Although it could be an MI or ischemia, it could also be:
•A spontaneous pneumo
•Pleuritis
•A lung pathology
•An aortic dissection
•Even a GI disturbance



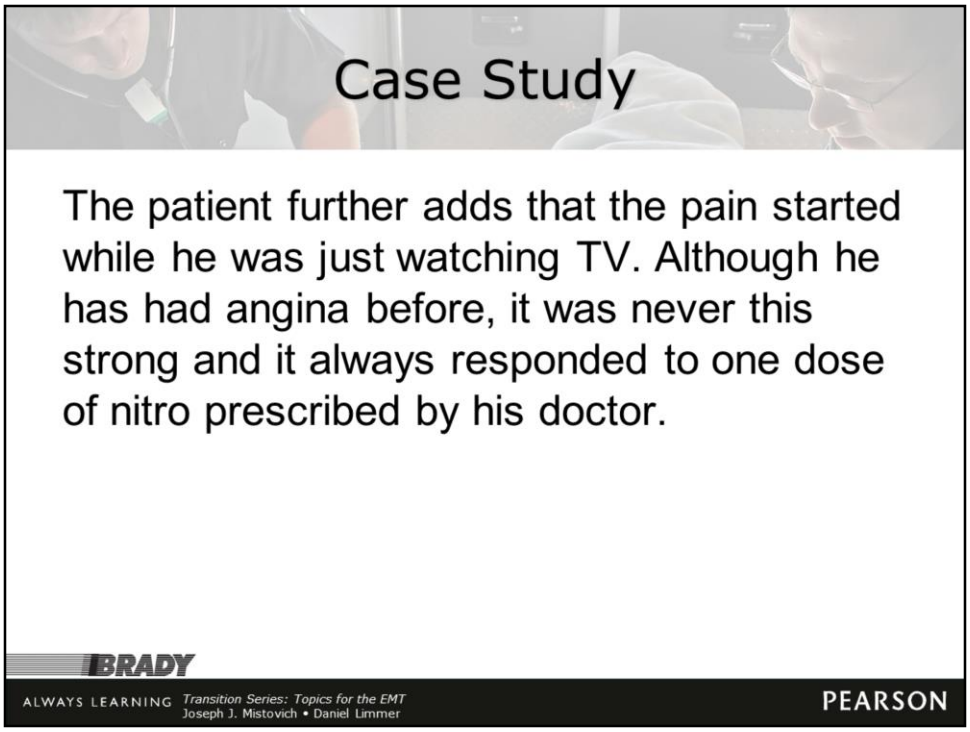
Discuss as needed.



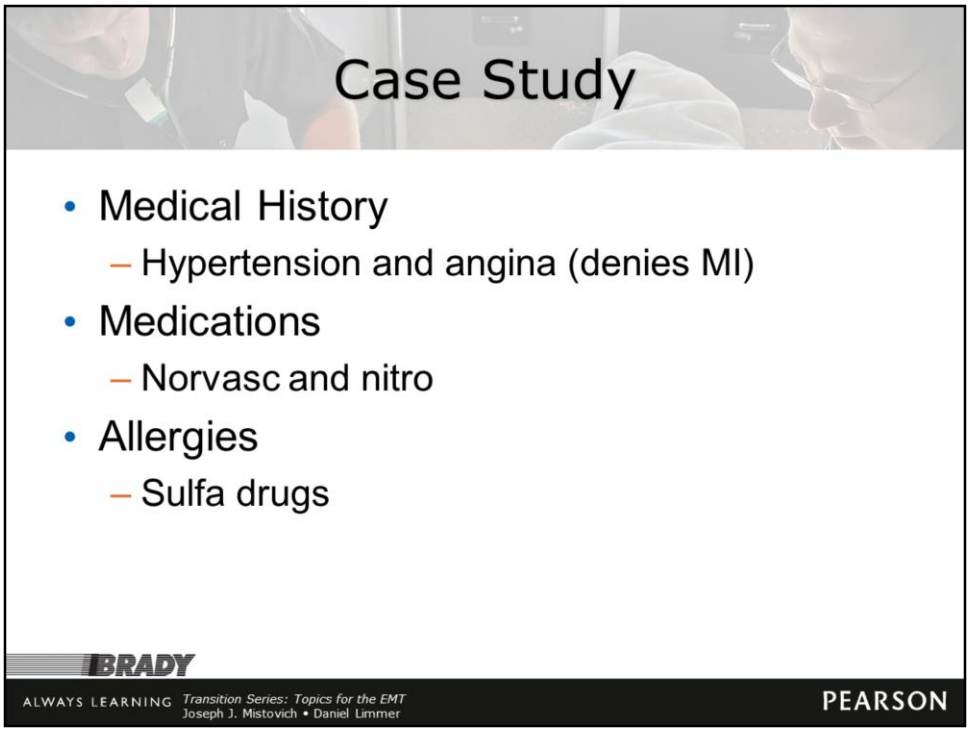
Although this patient has an intact airway, breathing, and circulatory components—they should still be categorized as potentially unstable due to the possibility of sudden cardiac arrest.

The life threat to this patient immediately is possible cardiac arrest.

As of right now, the EMT could administer high-flow oxygen and prepare the aspirin for administration after the SAMPLE history is complete (to avoid duplication of meds and to ensure the patient is not allergic to the med).



Discuss case progression.



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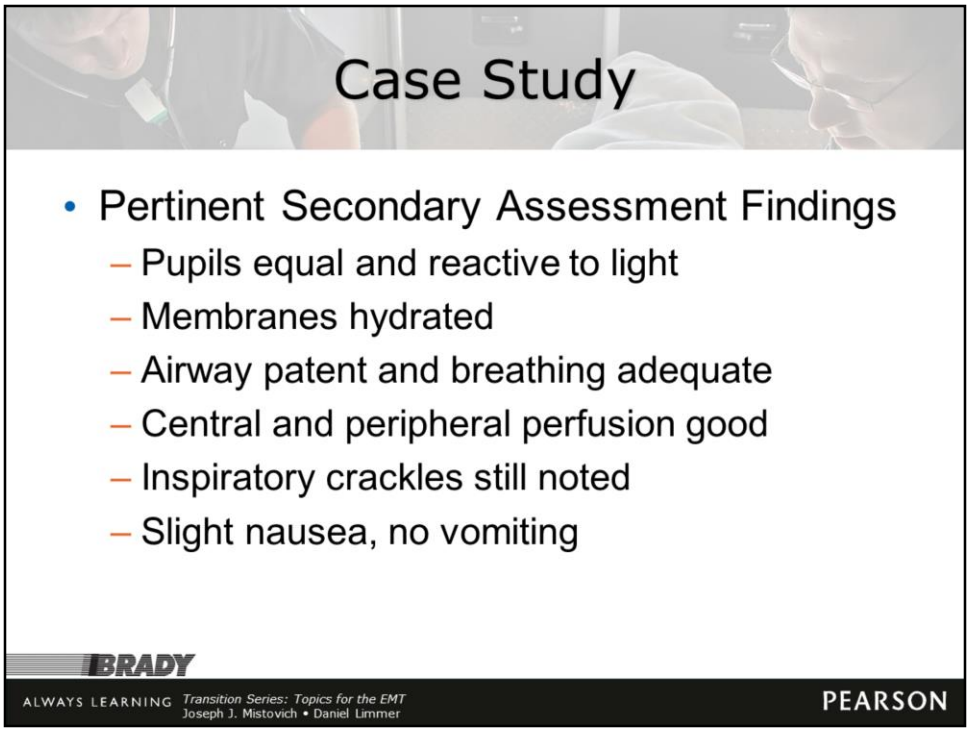

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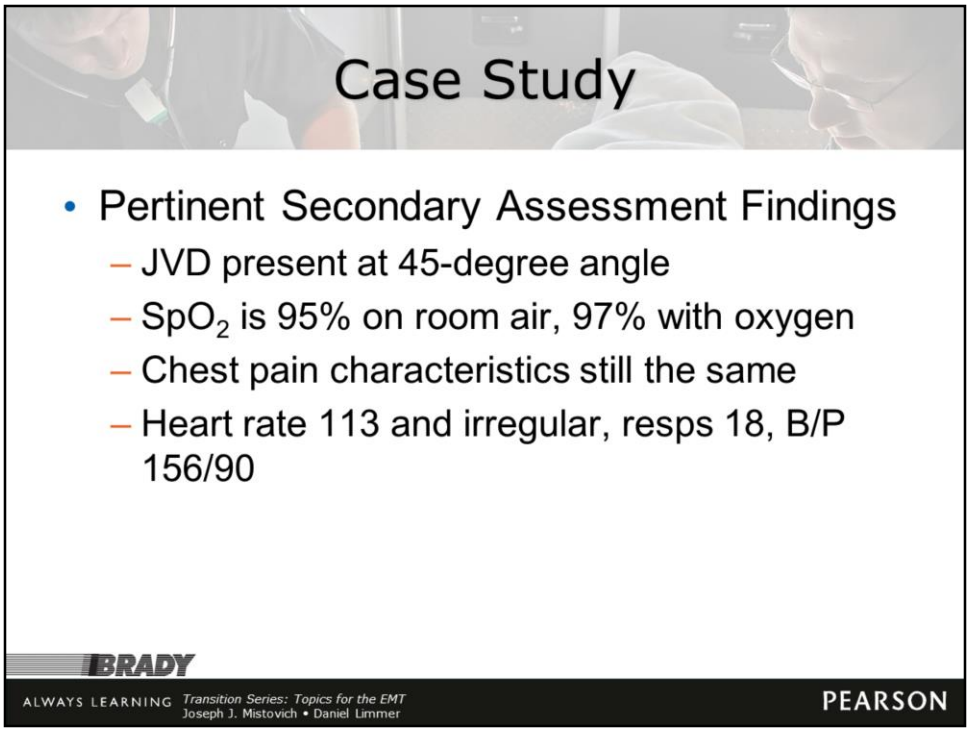

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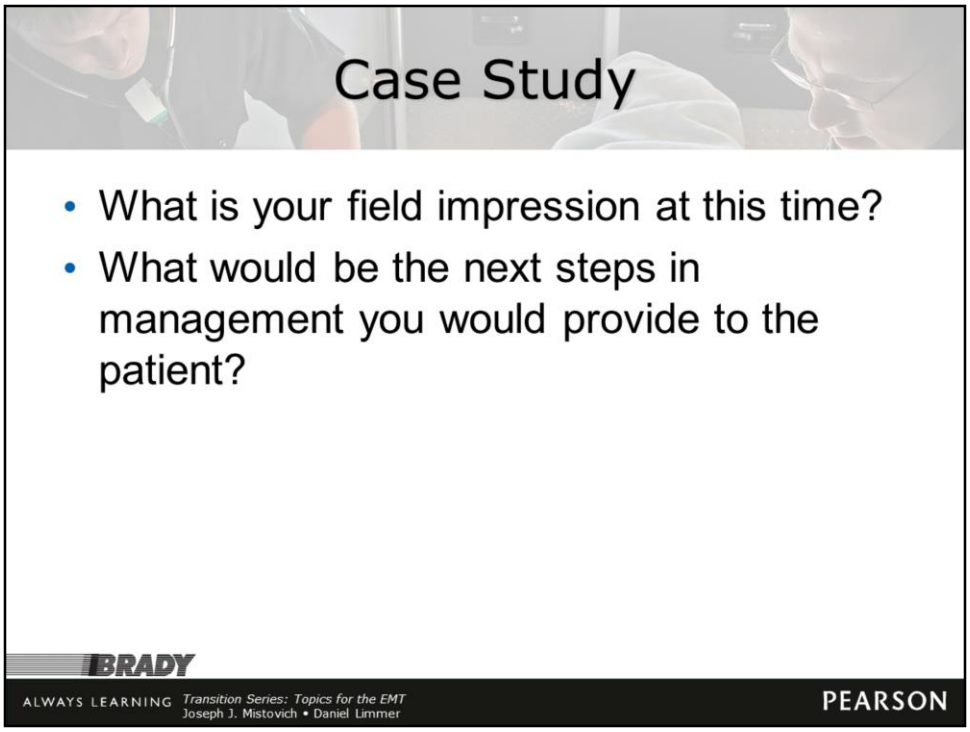
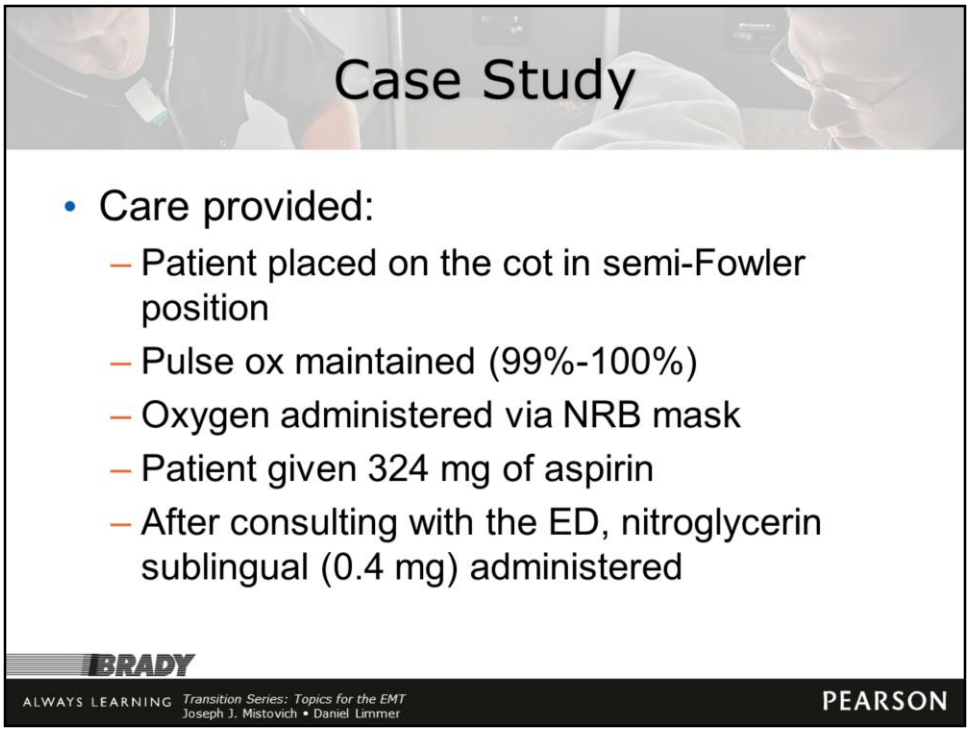


Given the presentation, the EMT should lean towards a patient having an MI rather than ischemic episode since the pain is worse than what is characteristic, and that the nitro did not have any effect.

The next step for management would include the ongoing administration of oxygen.

The patient should also receive baby aspirin (four 80mg chewable tablets), and after receiving proper medical control, the EMT can help with the administration of the patient's nitro.



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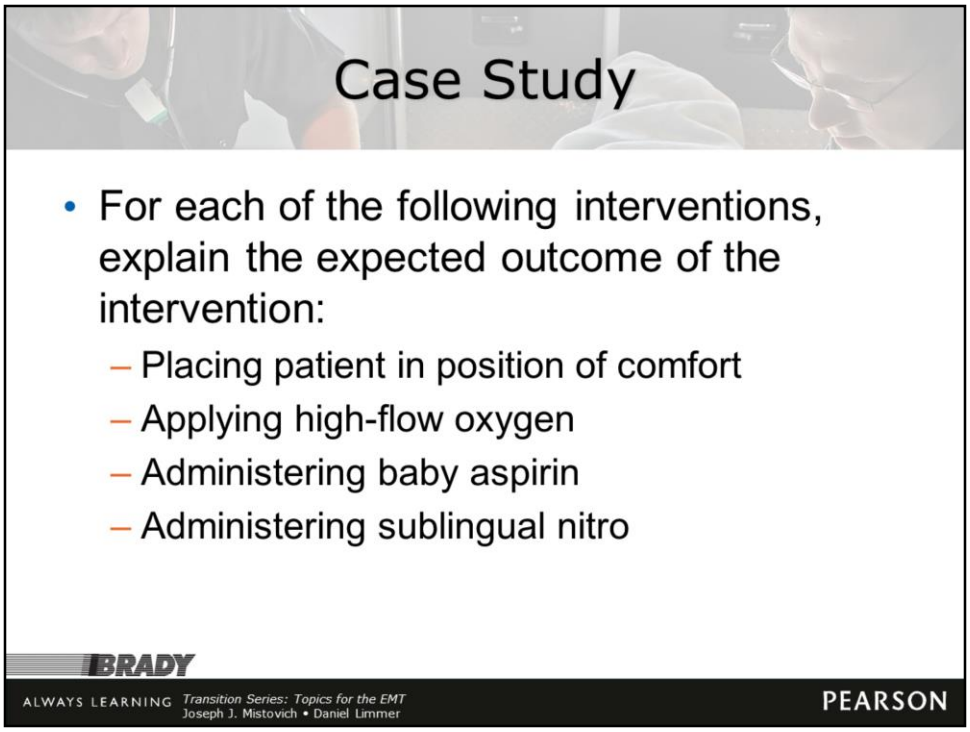
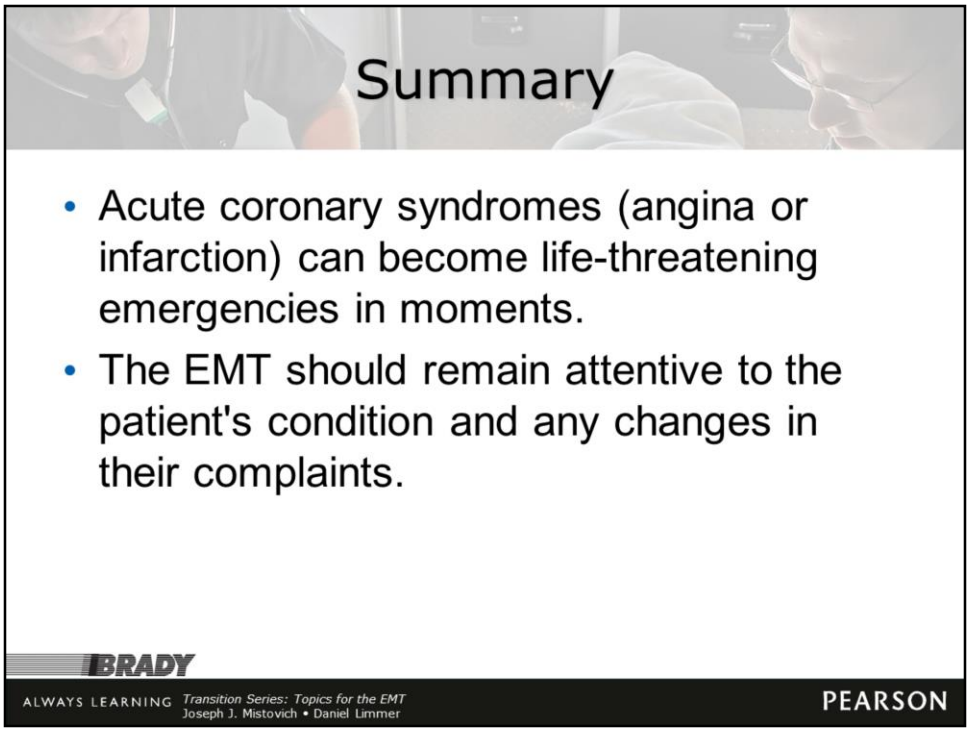


Placing the patient in a position of comfort will at least make them more comfortable, which hopefully will contribute to a drop in the sympathetic discharge. Plus, it makes it easier to breathe.

High-flow oxygen is designed to increase the on loading of RBC with oxygen to help assure the greatest amount of oxygen is reaching the ischemic or infarcting tissues of the myocardium. This will hopefully lessen the size of the injury.

Baby aspirin is used to help thin the blood and prevent it from clotting so readily. This is also hoped to help limit the amount of coronary occlusion and afford better myocardial tissue perfusion.

Nitroglycerin is used for coronary artery vasodilation effects (although it also vasodilates the periphery). With increased blood flow the myocardial, coupled with higher oxygen saturation, the outcome is hoped to be less myocardial damage.



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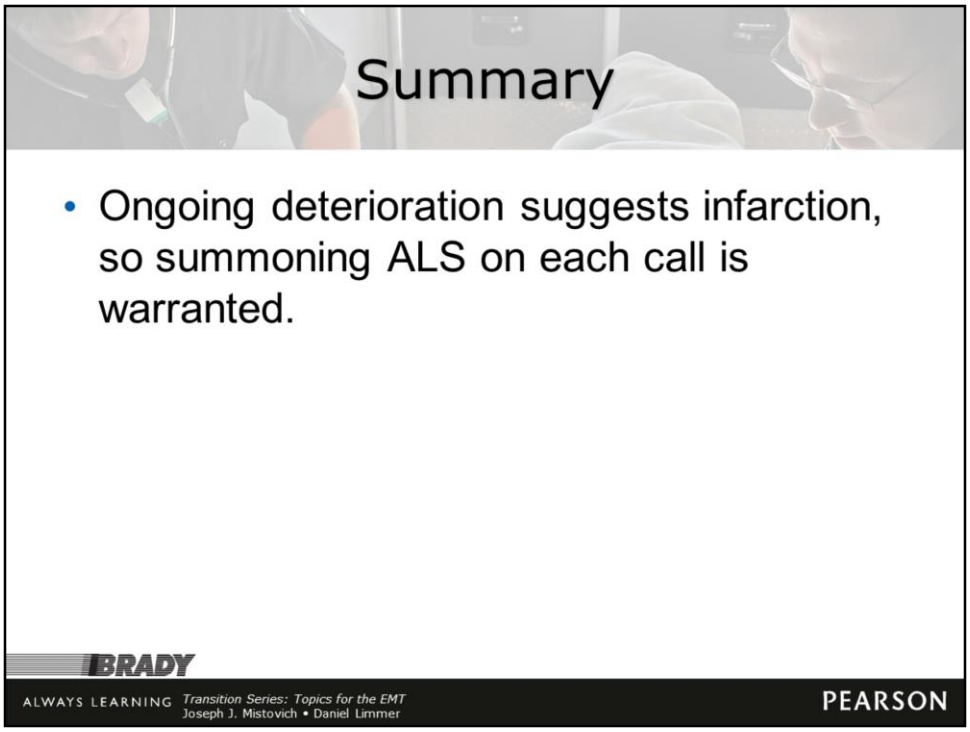

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