

HISTORY

41-year-old man.

CHIEF COMPLAINT: Chest pain of one hour duration.

PRESENT ILLNESS: While playing racquetball, the patient developed nausea and epigastric discomfort that increased over 30 minutes. His distress became an intense substernal ache that radiated into the neck. Fire rescue was called, and he was immediately transferred to a nearby emergency department.

Question: *Based on the history, what is your diagnosis?*

Answer: Acute myocardial infarction is the most likely diagnosis.

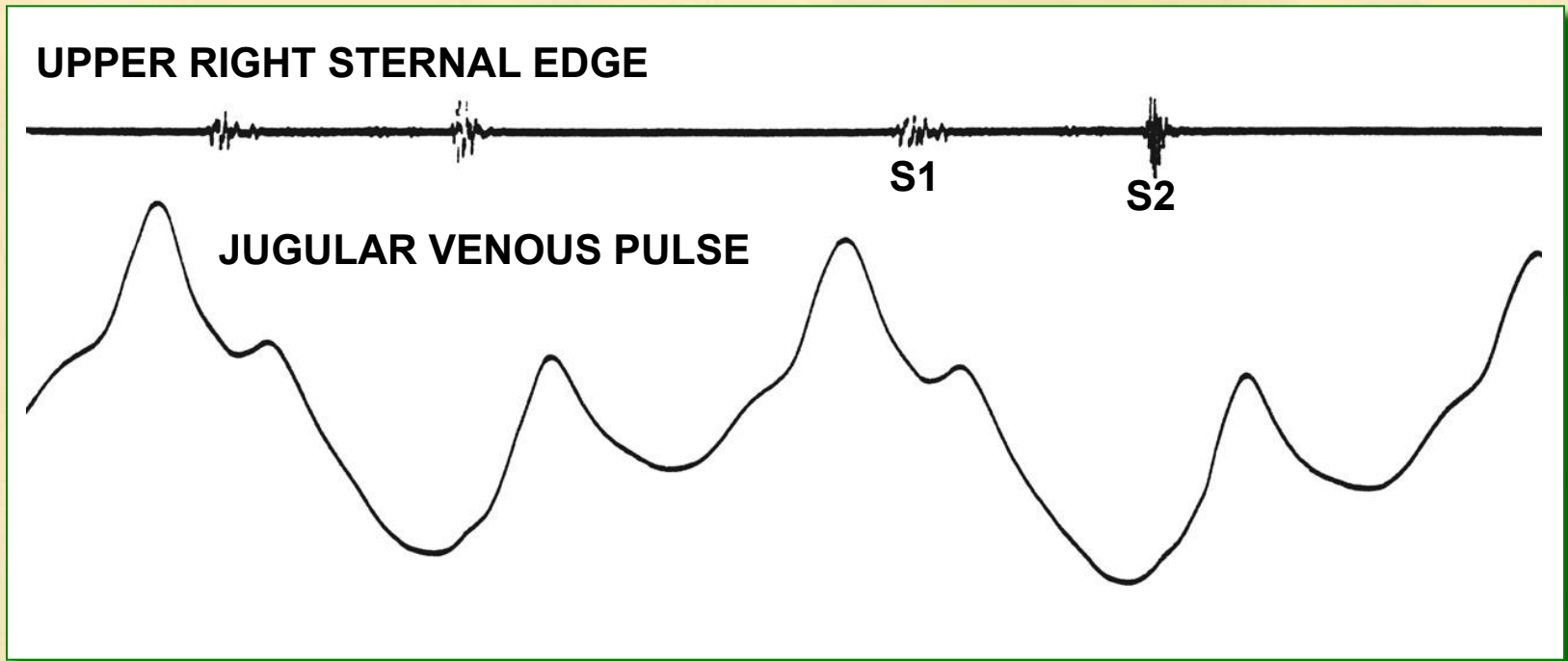
Based on this history alone, electrocardiographic monitoring was initiated immediately upon his admission to the emergency department. In addition, pertinent laboratory studies were ordered stat, including an ECG, chest X ray and blood for cardiac biomarkers.

Other causes of severe, prolonged chest pain that must be considered include pericarditis, aortic dissection, pulmonary embolism, musculoskeletal diseases and gastrointestinal lesions, e.g. esophagitis, cholecystitis and pancreatitis.

Proceed

PHYSICAL SIGNS

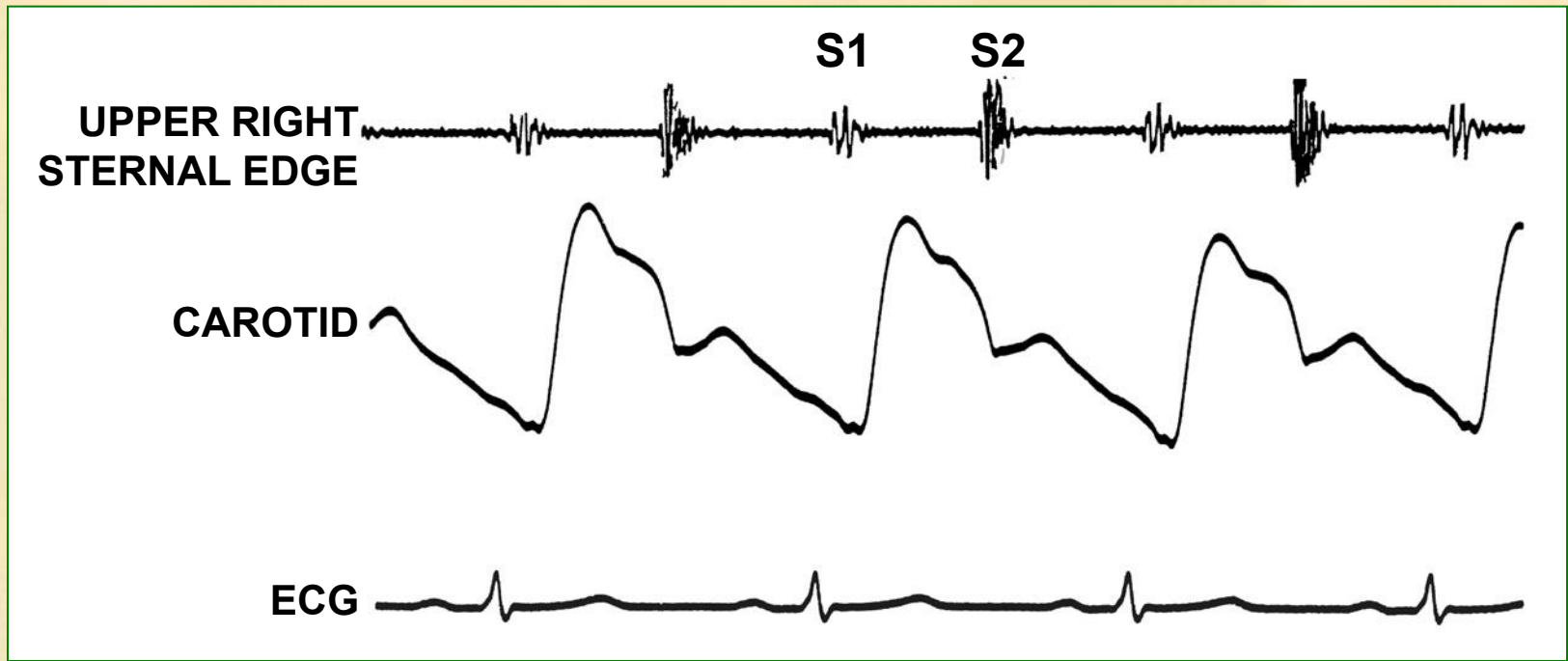
- a. **GENERAL APPEARANCE** - Anxious, pale, diaphoretic man.
- b. **VENOUS PULSE** - The CVP is estimated to be 3 cm of H₂O.



Question: *What is your interpretation of the venous pulse?*

Answer: The venous pulse is normal in mean pressure and wave form.

c. ARTERIAL PULSE - (BP = 90/60 mm Hg)



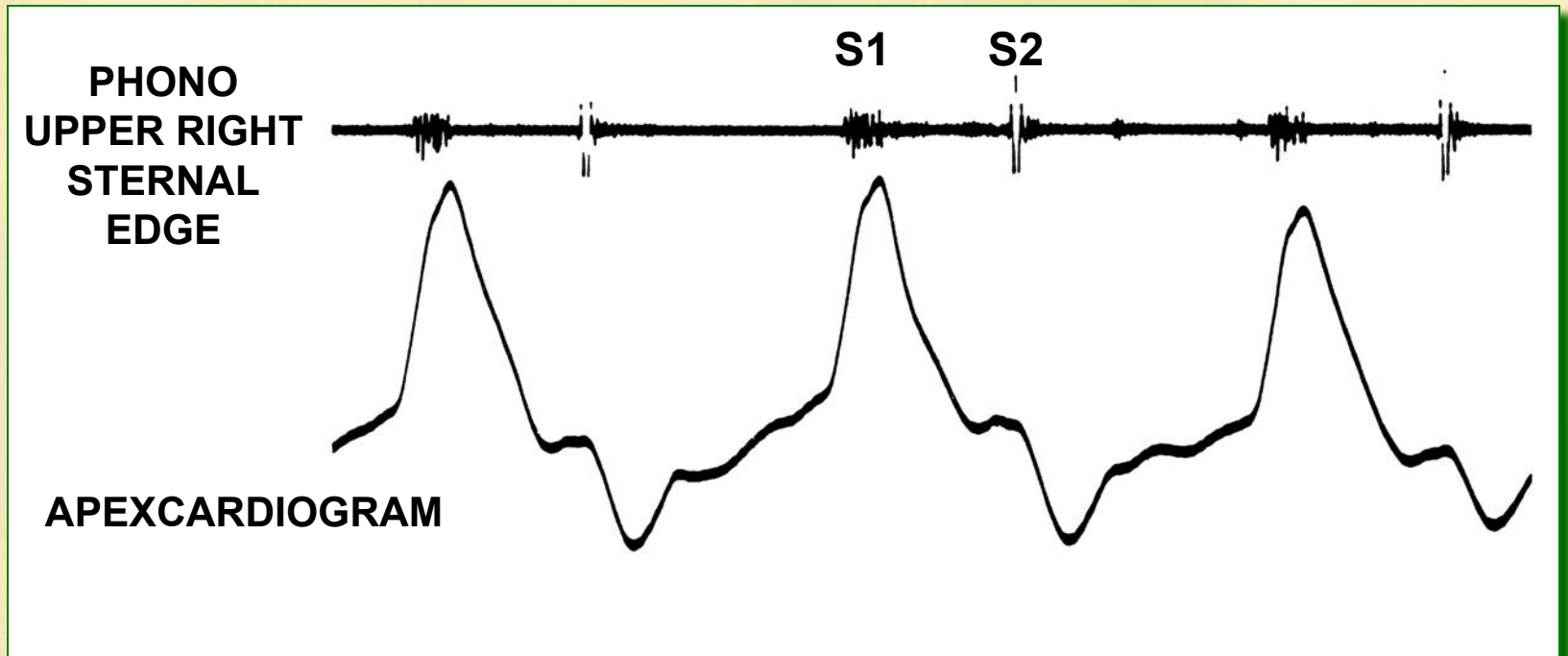
Question: *What is your interpretation of the blood pressure and arterial pulse?*

Answer: The blood pressure is mildly decreased, while the arterial pulse contour is normal.

Parasympathetic overactivity may cause hypotension in this clinical setting, but hypovolemia, arrhythmia, drug therapy and cardiac structural damage (e.g., left ventricular dysfunction, right ventricular infarction, rupture of a papillary muscle, septum or free wall) must also be considered.

Proceed

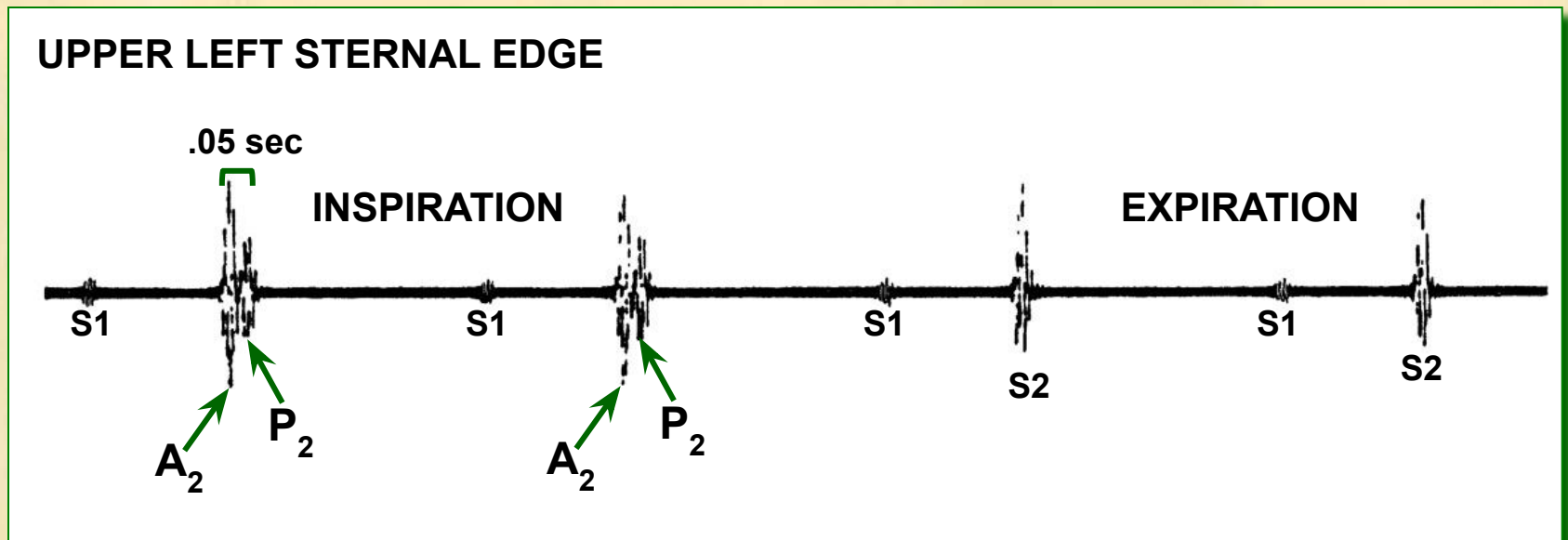
d. PRECORDIAL MOVEMENT



Question: How do you interpret the apical pulse?

Answer: The apical impulse is normal

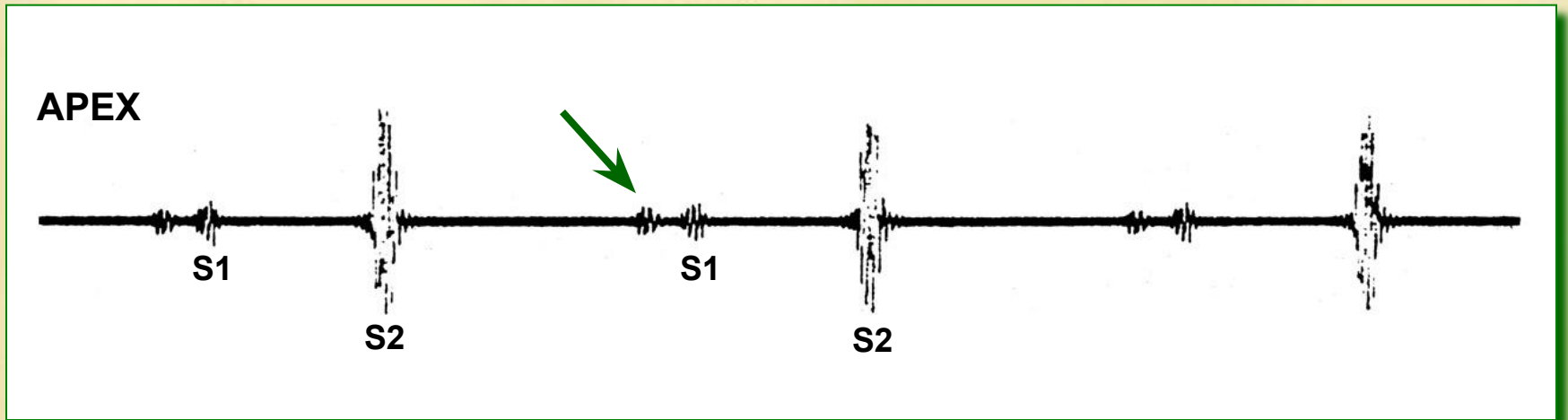
e. CARDIAC AUSCULTATION



Question: How do you interpret the acoustic events at the upper left sternal edge?

Answer: There is normal inspiratory splitting of the second sound.

e. CARDIAC AUSCULTATION (*continued*)



Question: How do you interpret the acoustic events at the apex?

Answer: The first heart sound at the apex is diminished in intensity. In this clinical setting, factors such as reduced left ventricular contractility or early closure of the mitral valve, as occurs with a prolonged PR interval, may diminish the first sound.

The arrow points to the fourth heart sound heard at the apex. This reflects left atrial contraction against a left ventricle with reduced compliance.

f. PULMONARY AUSCULTATION

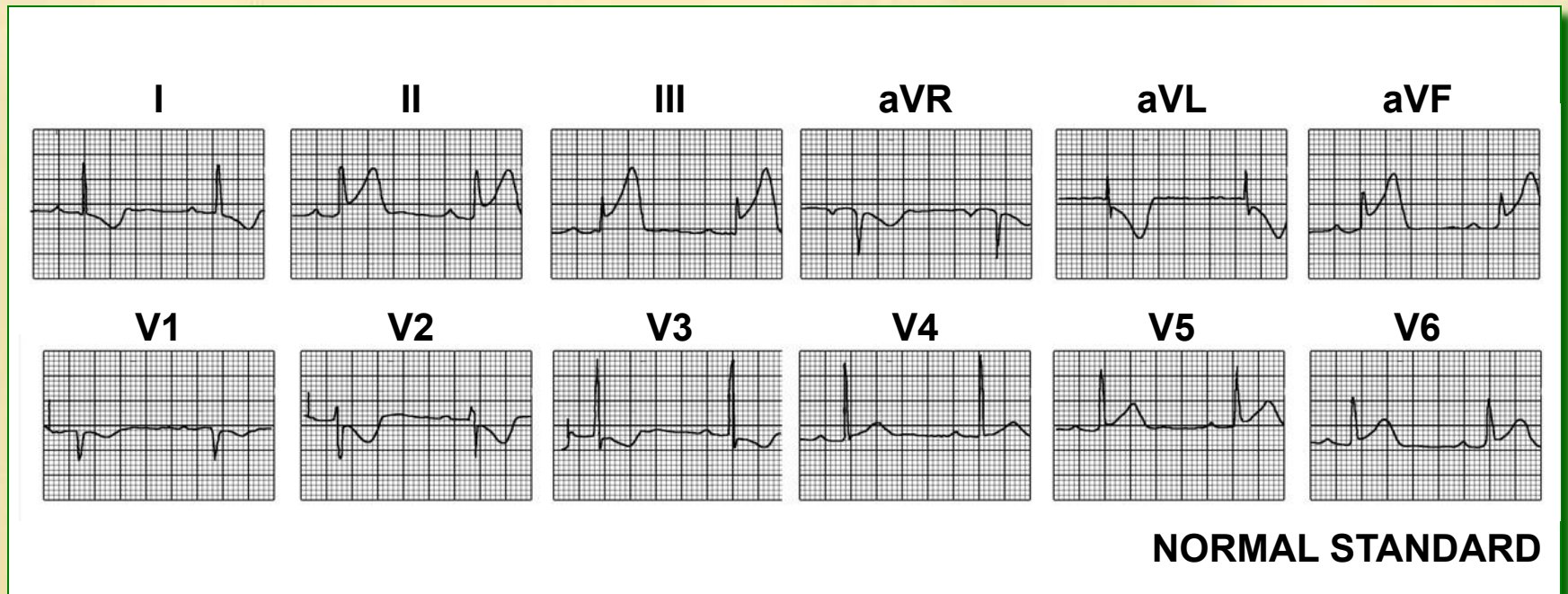
Question: *How do you interpret the acoustic events in the pulmonary lung fields?*

Proceed

Answer:

In all lung fields, there are normal vesicular breath sounds.

ELECTROCARDIOGRAM



Question: How do you interpret this ECG?

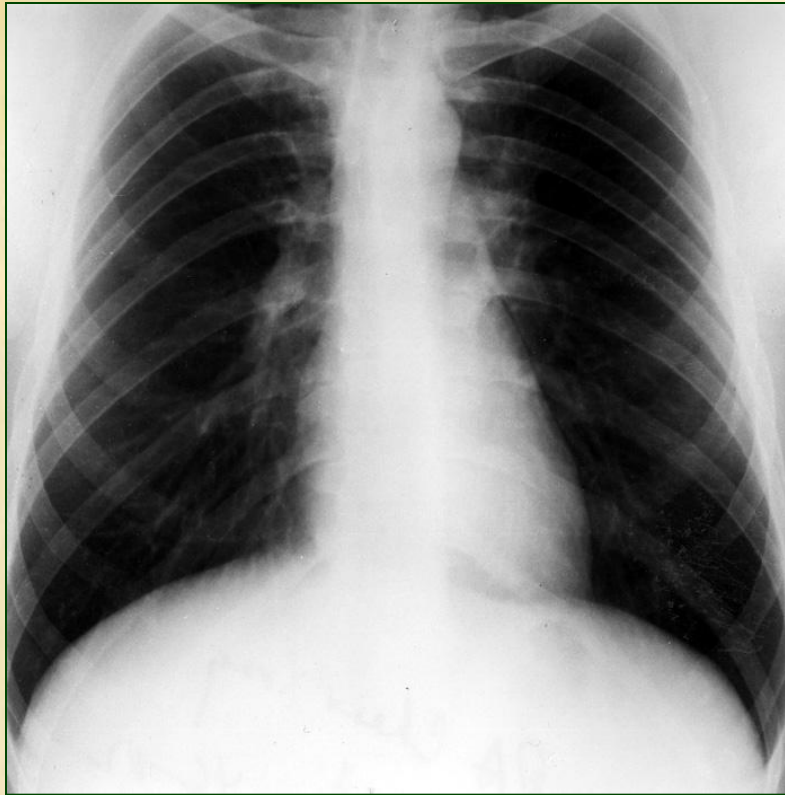
Answer: The ECG shows marked ST segment elevation in the inferior leads with lesser elevation in the lateral leads. There is ST segment depression in leads I, aVL and V1-V3. This ECG is diagnostic of acute inferolateral injury that almost always evolves to infarction.

This ECG also suggests that a significant amount of myocardium is in jeopardy, as patients with anterior ST segment depression often have a larger infarction than those with inferior ST segment elevation alone.

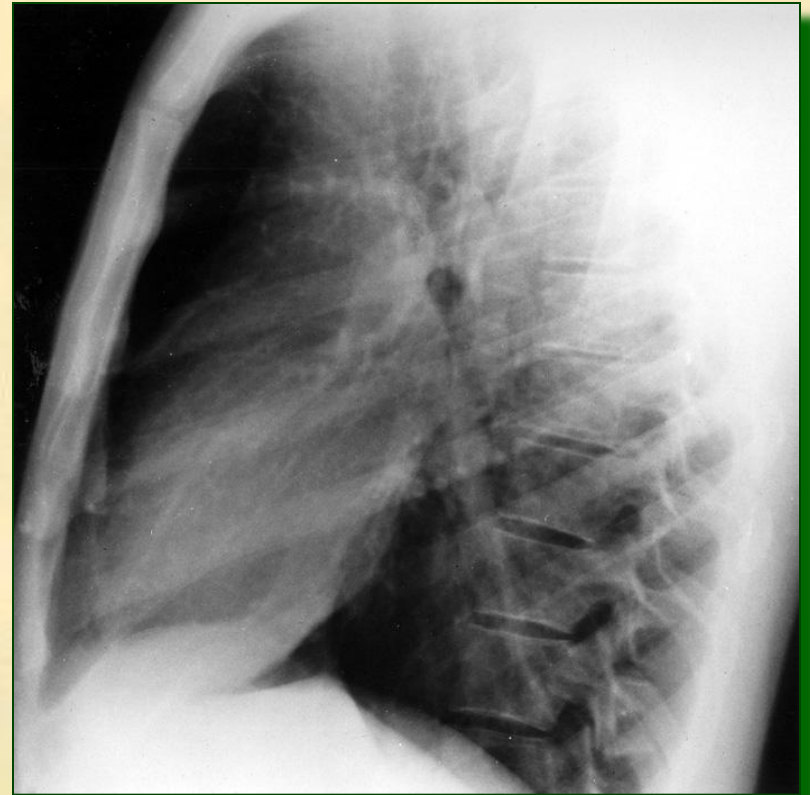
Note that the PR interval is prolonged, suggesting possible AV node ischemia as is commonly seen in inferior wall infarction due to right coronary artery obstruction.

Proceed

CHEST X RAYS



PA



LATERAL

Questions:

1. *How do you interpret the chest X rays?*
2. *Based on the history, physical examination, ECG and chest X rays, what is your diagnosis?*

Answers:

1. The chest X rays are normal.
2. Based on the history, physical examination, ECG and X rays, the diagnosis is an evolving acute inferolateral wall myocardial infarction.

The following rhythm was noted on the monitor.



Question: *What is your interpretation of this rhythm?*

Answer: The rhythm strip shows sinus rhythm with Mobitz Type I second degree A-V block (Wenckebach) as evidenced by progressive lengthening of the PR interval followed by a non-conducted sinus P wave.

A-V block is more common in inferior than in anterior wall infarction. The spectrum from a prolonged PR interval, to second degree A-V block, to complete A-V block may be seen. The conduction defect producing heart block in patients with inferior infarction is usually located in the area of the A-V node, rather than in the bundle of His or the bundle branches. The high incidence of heart block in this setting is due to the fact that in 90% of patients the right coronary artery supplies the A-V junction as well as the inferior wall. This type of heart block is usually transient.

Question: *What is your plan of therapy for this patient?*

Answer: Thrombosis plays an important role in ST-elevation myocardial infarction. Timely reperfusion of the occluded coronary artery can reduce infarct size and decrease mortality. Reperfusion can be accomplished by thrombolytic agents, percutaneous coronary intervention and coronary artery bypass graft surgery.

Unless the patient is allergic to aspirin, it should be given as soon as possible. If thrombolytics are used, the combination of an anti-platelet, anti-thrombin (heparin), and fibrinolytic agent is necessary.

Nitroglycerin increases myocardial oxygen supply, especially when collaterals are present, or if spasm is a component of coronary occlusion. Efforts should also be made to decrease myocardial oxygen demand by use of a beta-blocker.

Proceed

Answer (continued): If full catheterization facilities are available, urgent study and percutaneous intervention is most often the treatment of choice. Thrombolytic agents may also be an effective treatment if administered early in the course of an acute myocardial infarction. Contraindications to such therapy include any event or condition that predisposes to serious bleeding.

Because the catheterization lab was not immediately available, our patient was treated within two hours of the onset of his symptoms with aspirin, a thrombolytic agent and heparin. Beta-blockers were withheld because of his slow heart rate.

Proceed

LABORATORY

Myocardial biomarkers ordered on admission confirmed the diagnosis of infarction. Necrosis of myocardial tissue results in the release of intracellular biomarkers into the blood. In this case, their transient rise was typical.

Creatine kinase isoenzyme (CK-MB) elevation begins approximately 4 hours after symptoms of infarction, and in the absence of early coronary reperfusion peaks at about 24 hours. Troponins I and T rise slightly later and remain elevated longer.

Depending upon the thrombolytic agent used, appropriate clotting studies should be carried out.

This patient's routine blood work and clotting parameters were normal.

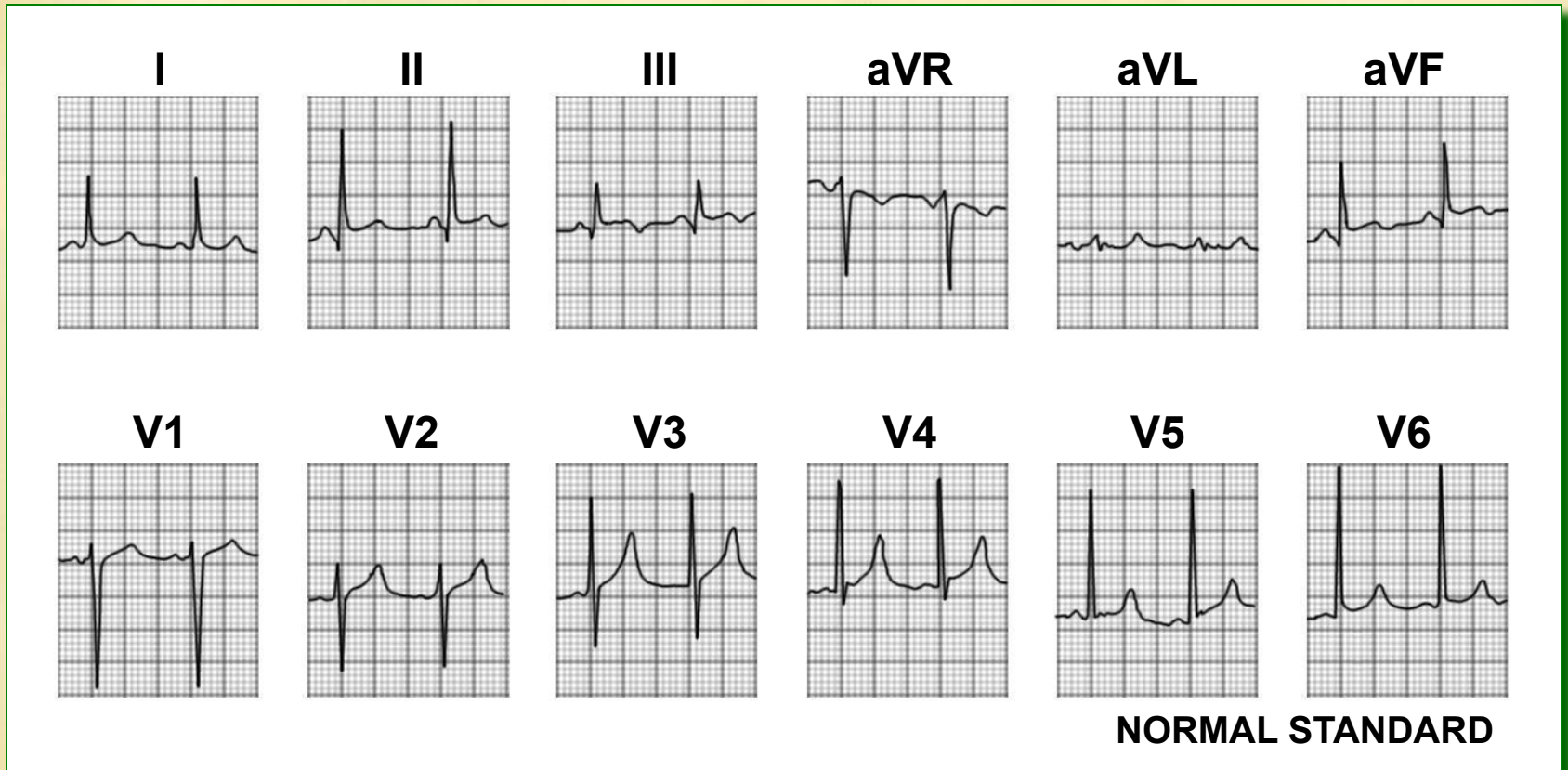
Proceed

The patient was placed at bed rest in the CCU.

Ninety minutes following thrombolytic therapy, the patient showed clinical evidence that was consistent with reperfusion: his chest pain resolved, his blood pressure rose to 120/80 mm Hg. An ECG taken at this time follows.

Proceed

ELECTROCARDIOGRAM

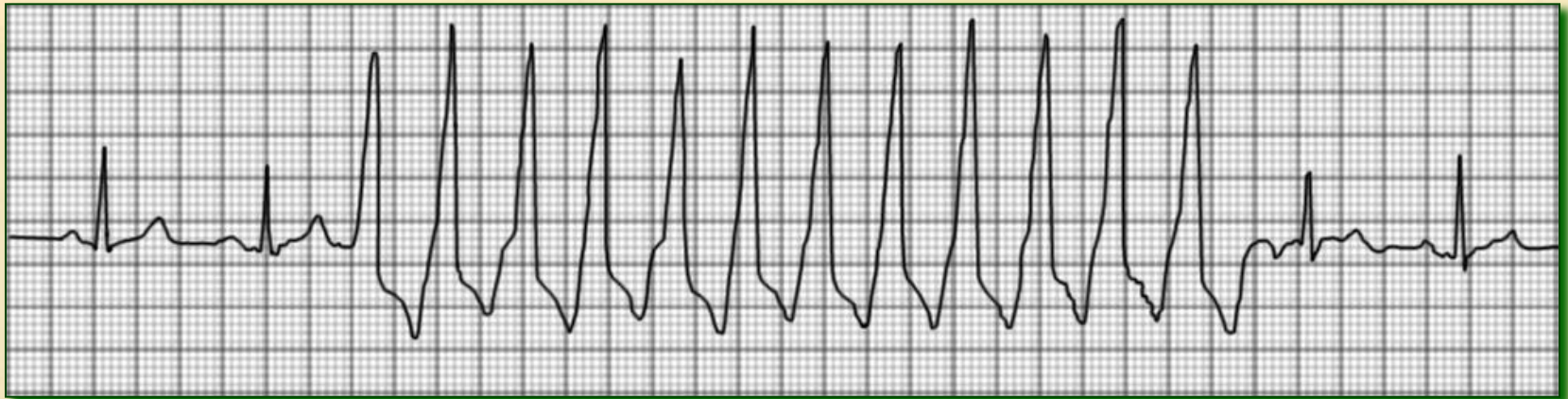


Question: *How do you interpret this ECG?*

Answer: Typical evolutionary changes of an acute inferior wall myocardial infarction are present: Q waves and symmetrically inverted T waves are seen in leads II, III and aVF, and the ST segments have returned to the baseline. Reperfusion has accelerated these ECG changes in the inferior wall and has resulted in the resolution of the other ST-T abnormalities seen on the initial ECG. Note that his first degree heart block has resolved, and the rhythm is sinus.

Proceed

The patient remained clinically stable, although 30 minutes later the following rhythm was seen on his monitor:



Question: *How would you interpret and treat this arrhythmia?*

Answer: There is a run of non-sustained ventricular tachycardia, i.e., three or more ventricular beats in a row at a rate of over 100 but less than 30 beats. Such “reperfusion ventricular arrhythmias” are frequently seen in this setting and do not require therapy.

Proceed

Two hours later the patient became cool and clammy, and his blood pressure dropped to 85/50 mm Hg. Because of its hypotensive effect, the nitroglycerin infusion was discontinued.

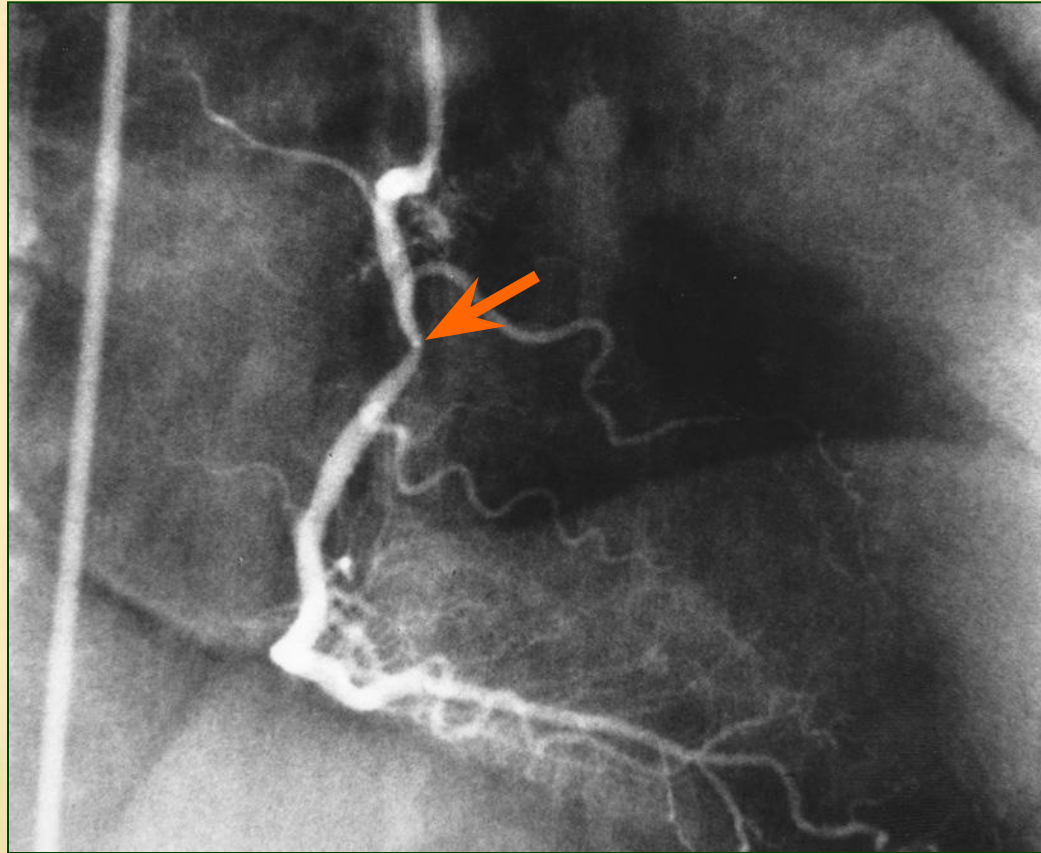
The patient's blood pressure rose to 105/70 mm Hg, but he was still clammy. A bolus of IV saline was then given, as patients with acute myocardial infarction may become hypovolemic, in part due to shifts in intravascular volume related to catecholamine effect. The patient's blood pressure then rose to 120/80 mm Hg, and he appeared alert and comfortable.

Over the next several days, the patient remained asymptomatic and stable and his activity level gradually ambulated.

Proceed

In order to define his coronary anatomy and ventricular function, angiographic study was carried out at a later date.

LABORATORY *(continued)*



Question: *What is your interpretation of this study?*

Answer: The right coronary angiogram shows an isolated non-critical stenosis (arrow) in the proximal right coronary artery. Additional views showed an ulcerated plaque in this area, the probable site of a thrombus that was present prior to thrombolytic therapy. The global ejection fraction was 55% with mild inferior wall hypokinesia. This non-critical degree of obstruction and the well preserved ejection fraction support the success of early thrombolytic therapy in this case.

A Thallium stress test was carried out and demonstrated no significant ischemia.

He was prescribed optimal therapy for secondary prevention that included recommended life-style changes, aspirin, beta-blockers, ACE-inhibitors and statins.

Proceed For Summary

SUMMARY

Coronary artery lesions range from the stable atheroma to complex lesions with thrombotic occlusion. The primary event in most acute infarctions is ulceration and/or rupture of an atherosclerotic plaque that becomes a nidus for platelet aggregation and the development of a thrombus. The resulting abrupt decrease in blood supply leads to cardiac tissue ischemia and necrosis.

There is greater myocardial salvage when efforts are directed towards the prompt reopening of the occluded coronary artery. In order to salvage jeopardized myocardium, therapy must be initiated before necrosis is complete. The goal of early therapy is to decrease the size of the infarction and prevent its complications. There is a significant difference in the ejection fraction and overall mortality of patients in whom reperfusion was successful.

Proceed

PATHOLOGY

This specimen is from a patient with left ventricular hypertrophy who died following an acute inferior wall myocardial infarction (arrows).



Proceed For Case Review

To Review This Case of Inferior Wall Myocardial Infarction:

The **HISTORY** is typical of an acute infarction, with the patient's nausea and epigastric discomfort suggesting the location may be inferior.

PHYSICAL SIGNS:

- a. The **GENERAL APPEARANCE** reveals an anxious, pale, diaphoretic man.
- b. The **JUGULAR VENOUS PULSE** is normal in mean pressure and wave form.
- c. The **CAROTID ARTERIAL** pulse contour is normal, but the blood pressure is slightly decreased.
- d. **PRECORDIAL MOVEMENT** reveals a normal early systolic impulse.

Proceed

- e. **CARDIAC AUSCULTATION** reveals an apical fourth heart sound, reflecting reduced left ventricular compliance. The intensity of S1 was reduced, due to a prolonged PR interval.
- f. **PULMONARY AUSCULTATION** reveals normal vesicular breath sounds in all lung fields.

The **CHEST X RAYS** are normal.

The **ELECTROCARDIOGRAM** shows first degree heart block and ST elevation, typical of an inferolateral infarction.

Proceed

LABORATORY STUDIES include cardiac biomarkers that confirm the diagnosis of an evolving myocardial infarction. A rhythm strip revealed transient Type I second degree A-V block. Following thrombolysis, non-sustained ventricular tachycardia occurred. Catheterization with angiography revealed an isolated 50% right coronary obstruction, mild inferior wall hypokinesia and a normal ejection fraction. An exercise Thallium study was normal.

TREATMENT with a thrombolytic agent resulted in reperfusion. His second degree A-V block and ventricular tachycardia did not require treatment. His hypotension responded well to discontinuation of his NTG and the administration of fluids. The remainder of his hospital course was uneventful. A secondary prevention program was instituted during his hospitalization.