Roger Crockett, a 68-year-old man with a 40-pack-year smoking history and recent complaints of angina (sub-ternal chest pressure) upon exercising, goes to you, his primary care provider.

1. While listening to his heart with a stethoscope, you notice a high-pitched, blowing, systolic murmur, heard best directly under the left nipple. A review of Roger's medical records shows no prior history of a heart murmur. What is causing this new murmur?

   From this location, Roger’s systolic murmur is most likely from the mitral valve. During systole, the mitral valve should be closed, therefore if we are hearing a murmur, the valve must be insufficient, or not closing properly. If we were viewing this valve via Doppler echo, we would see a large blue plume of color moving from the left ventricle into the left atrium.

2. Is the cause of the murmur in any way related to his heart attack? Explain.

   How is the mitral valve kept in the closed position? It is stabilized by the papillary muscles and chorea tendinae. If Roger’s heart attack damaged those papillary muscles, then the valve is probably not stable in the closed position and causing a turbulent leak of blood during systole.

   Questions that arose in class -

   What if the valve that produced the sound was the aortic valve? Then with a systolic murmur, the aortic valve would be stenotic, or not able to open properly.

   Why do some patients with mitral valve prolapse receive antibiotics? As Dr. Katz explained the echo lab, certain bacteria have receptors for proteins found on the valves and those with prolapsed valves are vulnerable to endocarditis.
A 50-year-old airline pilot complained of severe, intense, precordial, crushing sensation with pain radiating to the left shoulder and down the inside of the left arm, triggered by an off-duty tennis match. The chest discomfort brought on by the exertion was relieved by rest. Emergency room examination resulted in the following information:

Heart rate (HR) 98 b/min
Blood pressure (BP) 160/110 mm Hg
ECG: ventricular extrasystole arrhythmia (premature ventricular contraction [PVC]) as well as S-T segment depression and decreased R wave height.

The following day an exercise tolerance test was performed to test the functional response to graded stress. This symptom-limited test gave an ischemic ECG response during exercise, characterized by a downward-sloping S-T segment. Mild exertion resulted in chest pain, which was relieved by sublingual nitroglycerin. Coronary angiography showed lumenal obstruction >70% (88%) in three major coronary vessels, including the left anterior interventricular (descending) coronary artery. Nitroglycerin, beta-blockers, and calcium channel blockers were tried as pharmacologic therapy. Angioplasty, the procedure in which a balloon-tipped catheter is inserted into the partially obstructed vessels, was able to increase coronary flow to near normal values.

1a. What is the term for the chest pain experienced by this individual? 1b. What is the cause of this pain?

Our pilot is experiencing angina, a type of chest pain or chest discomfort that occurs when the heart muscle is not receiving enough oxygenated blood. This pain is triggered by exertion in the case and is therefore called stable angina. It is relieved by reducing exertion and taking nitroglycerin.

2. In a normal ECG tracing the ST segment should be isoelectric. For the individual in this case study, the ST segment is down sloping or depressed. What normally occurs during an isoelectric ST segment and why should a clinician be concerned when the ST segment is either depressed or elevated?
Isoelectric events indicate that a tissue is completely depolarized or repolarized. For example we are concerned about the duration of the PQ segment as a measure of AV delay. The duration is of the ST segment is not as critical but it should be present! The ST segment is the isoelectric event between the QRS complex and the T wave. If the ST is not isoelectric, ischemia is affecting the electrical activity of the cardiac myocytes. Depolarization may not be spreading through the conduction pathway normally. There may be abnormal depolarizations occurring in a “triggered way”. An alteration in the ST segment is a sign of cardiac ischemia and is of great concern.

3. What is the site of action for each of the pharmacologic drug therapies?

a. Nitroglycerin: is a coronary vasodilator. By dilating the coronaries, nitroglycerin bring more oxygenated blood to the myocardium and treats angina.

b. Beta-blockers: are antagonists at Beta receptors. By blocking the beta receptors, the heart rate is slowed. Coronary blood flow is reduced during systole, especially in the left ventricle. By slowing the heart rate and increasing the time for diastole, the amount of time of coronary blood flow is increased. A side effect of beta-blockers is a reduced contractility. Both the decreased heart rate and decreased contractility will reduce the work of the heart. This decreases the demand for oxygen.

c. Calcium channel blockers: Calcium blockers can be used to treat certain dysrhythmias. The identification of supraventricular arrhythmias is beyond the scope of this course. However, calcium blockers can also relax smooth muscle and treat hypertension. This decreases afterload, decreases the work of the heart and decreases the heart’s demand for oxygen.

SUMMARY – if oxygen delivery does not match demand, then dysrhythmia and heart attack can occur. Two strategies to treat this mismatch is to increase delivery (nitroglycerin) or decrease demand (beta blockers and calcium blockers).
Recall your patient, Roger Crockett, a 68-year-old man with a 40-pack-year smoking history and recent complaints of angina (sub-sternal chest pressure). In an earlier module, it was determined that Roger had a mild heart attack and some valve damage. The valve damage has not decreased the ejection fraction significantly, so you and Roger’s cardiologist refer Roger to smoking cessation and lipid lower drug therapy.

Unfortunately, Roger collapses while mowing his lawn. Paramedics arriving at the scene found him unconscious, not breathing, and without a pulse. CPR was successfully performed and Roger was transported to the hospital. An ECG was suggestive of an anterior wall myocardial infarction, and he was given an intravenous solution of tissue plasminogen activator (TPA). Elevated blood creatine phosphokinase (CPK) levels measured over the next 2 days confirmed the diagnosis. Coronary angiography was performed a week later, revealing the following results:
- Circumflex artery: 20% blocked
- Right coronary artery: 15% blocked
- Left anterior descending artery (LAD): 95% blocked
  
("Anterior intraventricular artery")

3. While listening to his breathing with a stethoscope, you hear some wheezing and inspiratory rales ("crackling noises")? There are two answers to this question. One is a controllable health risk—the other is the result of a leaky ventricular valve and its effects on pressure.

First Roger is a smoker and some of his lung sounds may be due to pulmonary disease. Second, Roger has had a heart attack. If his heart is losing pumping effectiveness, blood may be backing up into the pulmonary circulation and affecting his breathing.

4. A chest X-ray taken two weeks after his collapse shows a markedly enlarged cardiac silhouette and generalized haziness at the bases of the lungs. What has caused this increased silhouette? Hint: Think about cardiac output

Well, we have some evidence of the pump failing. Roger’s heart is enlarged and we see signs of blood backing up into the pulmonary circulation. In a later section, we discussed the Starling’s forces. This back up of blood is increase pulmonary capillary hydrostatic pressure and pulmonary edema.

Roger is stabilized and ultimately discharged from the hospital. Three months after the heart attack, he comes back to his physician for a checkup. He complains of dyspnea ("shortness of breath") at rest and difficulty breathing while lying down ("orthopnea"). He says he can only sleep when he is propped up by two large pillows.
5. Why is he having these symptoms?

Yep, that pump is failing. The back up of blood and the increased pulmonary capillary hydrostatic pressure is increasing capillary filtration. There is no place for that filtrate to go but the alveoli. The alveolar edema is interfering with oxygenation and Roger is short of breath. Why do the pillows help? His hydrostatic pressure and edema are lower in the upright position and worsened when he is supine.

6. Which term more accurately describes the stress placed upon Roger's heart -- increased pre-load or increased aferload?

The left heart is failing and an already overstretched heart is being stretched even more by the backup of blood. This is a preload problem. We could also say that Roger has a contractility problem because of his heart attack but that was not part of the question.

7. What is creatine phosphokinase (CPK) and why are elevated CPK levels in the blood suggestive of a myocardial infarction (heart attack)?

CPK is an intracellular enzyme for the short term production of ATP. If this enzyme is in the blood, heart cells had to die and release it to the blood. Another protein that is a specific indicator of MI is the cardiac isoform of troponin.
1. Why was weight loss indicated as the initial form of treatment?

_When a person gains weight, the blood has to be transported over a longer distance. The length of a blood vessel has a direct effect on vascular resistance and can contribute to hypertension. Weight management is part of the care plan._

2. Explain the sites of action for the three pharmacologic agents prescribed for this individual

   a. **Oral diuretic** - A diuretic increases the diuresis of fluid by the kidneys. It reduces plasma volume and decreases blood pressure.

   b. **Beta-blocker** – A beta blocker blocks the effects of epinephrine, decreases heart rate and decreases blood pressure

   c. **Vasodilator** – Vasodilators decrease peripheral vascular resistance and decrease blood pressure and afterload.

3. Why was the vasodilator not used before the other drugs were added?

_Often diuretics are used first in the treatment of hypertension and then other drugs are added later to manage the blood pressure. This is not something I expect you to know for the exam._

_What is the take home here? If a person has hypertension it could be because the kidneys are retaining too much fluid, it could be because the HR is too high or the vessels are too contracted. These therefore are all avenues for treating the problem._

4. Write the formulas for determining mean arterial blood pressure?

_A common formula is \( \text{DP} + \frac{1}{3} (\text{SP-\text{DP}}) \)

So before treatment, his MAP was \((115 + \frac{1}{3}(180-115)) = 137 \text{ mmHg}\)

After treatment his MAP was \((90 + \frac{1}{3} (140-90)) = 108 \text{ mmHg}\)

5. Would you have any concerns if this patient presented to your office for dental care?

_Yes, you would want to check his blood pressure before the examination. Any procedures requiring local anesthetic with epinephrine would be of concern._
David is a 14 year old, avid baseball player. This morning as David was playing short stop he fielded a grounder which took a bad bounce and knocked out his two maxillary central incisors and gave David a bloody nose that bled profusely. David’s parents brought him home and began to apply cold packs and pressure to the area of trauma and called his dentist.

David, an uncommonly curious boy, understands that inflammation is caused by fluid moving from the blood and accumulating in the space between the cells. He also understands that this inflammation process is caused by paracrine agents, like histamine, cytokines and leukotrienes.

How does the stress response act upon blood vessels? (hint: adrenergic receptors)

ANS Norepinephrine from sympathetic neuron,s bind to alpha 1 receptors and cause vasoconstriction. This maintains blood pressure in the face of stress and bleeding. Epinephrine from the adrenal cortex will bind to both alpha 1 receptors for vasoconstriction but will also bind beta2 receptors for relaxation of smooth muscle in the bronchioles and skeletal muscle vascular beds.

1) What physiologic processes oppose bleeding in response to injury?

Local factors - Injured blood vessels release endothelin, vasoconstrictor that causes vasospasm at the site of injury. Damaged tissues release tissue factor, a major initiator of the clotting cascade. Note the discussions from your book about endothelin as a local factor.

2) What causes swelling in this boy?

Starlings forces - Trauma causes the release of histamine, a vasoactive substance that increases capillary permeability. Plasma proteins leak to the extravascular space, bringing fluid with them. Essentially, tissue oncotic pressure increases. This leads to swelling. A possible benefit of this process is the dilution of toxins and bacteria.

3) Before the bleeding stopped, he lost enough approximately 250 mls of blood. What processes help replace that lost volume?

Activation of the renin angiotensin system will increase his plasma volume and maintain vascular resistance.
#6 Blood Pressure

You have an appointment with Herold Crick this morning. Herold’s chief complaint is a throbbing pain in #31. Upon a review of past medical history you notice that Herold had radiation therapy two years ago for a squamous cell carcinoma nodule located on the left-anterior side of his neck just 4 cm lateral from his larynx.

Herold’s vital signs are as follows:

| Normal values | BP 120/80  |
| HR 60 bpm     |  |

| BP 180/95 mmHg | HR 96 bpm |

Herold is not taking any blood pressure medications and has had no history of hypertension. His Cholesterol levels are normal and is a non-smoker.

You have taken the following full mouth panoramic X-ray and notice several small radiopaque lesions located 2.5 cm inferior and posterior to the angle of the mandible; very near the area where Herold’s nodule was removed.

What could be a possible cause of Herold’s elevated BP and HR?

**He has calcification of the carotid sinus. His brain stem is getting some incorrect information about his blood pressure (that it is too low). The brain stem is activating the sympathetic nervous system and raising the blood pressure and heart rate.**

Is Herold at risk for any medical conditions?

**Hypertension is the silent killer. He is at risk for stroke and heart attack, for kidney and retinal disease.**

You decide to perform a root canal on #31 and administer a local anesthetic with epinephrine. Is this an acceptable treatment plan given what you know about Herold? Why or Why not?

**Epinephrine would elevate his blood pressure even more. This treatment plan is contraindicated.**