>> PROFESSOR: All right. So, today's going to be chapter 10 on the cardiovascular system. So the first topic is going to be anatomy and physiology, and so basically I'm going to talk to you about the heart and how it functions. Okay? First of all, the heart is made up of how many chambers? Four. Great. All right. When I talk about the heart, I very often divide the heart into the right side and the left side. Okay? And the right side receives blood from where? Anybody know? Generalization? What is it? Where's the right side receive blood from?
>> STUDENT: [Inaudible].
>> PROFESSOR: Good. From the veins. Great. And the veins bring blood from where? The rest of the body. Okay. So the blood in the venous system is going to be either what high in oxygen or low in oxygen? It's going to be low. Great.
Okay. So technically, when blood comes to the heart, it enters through the right atrium, it then passes through the right ventricle, it then goes through the pulmonary trunk to the pulmonary arteries, okay, to the lungs for what? Oxygenation. Great. When the blood is oxygenated, then it comes back to the heart through the pulmonary veins, and it comes to the left side of the heart. So then the left side of the heart receives oxygenated blood, the right side receives blood that is low in oxygen or poorly oxygenated. Okay?
So the first chamber that receives blood from the lungs is what? Anybody know? Left atrium. Right. Fills up with blood, and then it passes to the left ventricle, and then the blood leaves the heart through what vessel? The aorta. Great.
So the chambers of the heart, you got the right atrium that receives blood from the body, right, then blood passes from the right atrium to the right ventricle and then it passes through those vessels to the lungs back to the left atrium and the left ventricle. The left atrium, the right atrium, are thin-walled vessels, they receive the blood, and then they on taken the blood and then they empty into the ventricles. Now, basically three vessels bring blood to the right atrium. There's the inferior vena cava, and the superior vena cava. Inferior vena cava brings blood from the body, basically the lower extremities and everything, and the superior vena cava which brings blood to the right atrium from the head, and then a smaller vessel called the coronary sinus, all right. The coronary sinus brings blood
from the heart muscle back to the heart. All right? And then it goes through the right side of the heart to the lungs, and then back. Okay? So the blood flow is going to be from the vena cava and coronary sinus to the right atrium, right ventricle, and then to the lungs, back through the left atrium, left ventricle, through the aorta. Okay? Now, when the ventricles contract, what do we call that? Systole. Systole. When they relax, we call that diastole. Okay?

So when the ventricles contract, what they're doing is they are pumping blood out of the ventricles, all right, and away from the heart. And we'll talk about the left ventricle. All right? When the left ventricle contracts, it pumps blood, it squeezes itself, the walls come close together and it pumps blood through the aorta. Okay? This is during systole. Then, when all the blood is out, the left ventricular wall, the muscles within the muscle fibers, within the left ventricular wall have to relax. Why? Because the ventricles have to open up, because the left atrium has to empty the blood that it's been collecting into the left ventricle. Okay? So we call that diastole, so the ventricles relax, they fill up with blood. Systole, they contract. And of course then what words do we get from systole and diastole? Systolic blood pressure and diastolic blood pressure. Okay?

So therefore in a lot of ways, when you have a blood pressure taken, the top number is, what, systolic or diastolic? It's the systolic. So the systolic pressure measures the pressure within the arteries, okay? And the pressure is the work that the left ventricles have to do to get blood through the circulatory system. Okay? The diastolic pressure is the pressure of the ventricles at work -- I'm sorry, at rest. So that's like the resting blood pressure. All right? So you have why are systolic blood pressure, your diastolic, your systolic is higher. So as you can see, if I tell you that normal blood pressure is going to be 100 over 70 or 110 over 70, or for some people can go as low as 90 over 60, that's the amount of work the heart has to do. So someone who has a blood pressure of 160 over 110, all right, what's that mean? That means the heart muscle has to work at a much greater level because it has to pump blood out where? Not at 90 millimeters of mercury, but at 160. Okay? So that heart has to do more work. When heart muscles have to do more work, what does that require? More what? More oxygen, and more muscle. Right.

So that's why sometimes people have chronic hypertension. They get to go for a chest x-ray. It's one of the screening tools for hypertension. If they get a chest x-ray, and it's a crude evaluation, but if they've got cardiac enlargement,
it's due to the left ventricle, and if they've got left ventricular enlargement, or now that you've taken survey, you know the term, left ventricular hypertrophy enlargement, they've had longstanding, chronic hypertension, it's not a new event, they've had it for a while and their heart musculature has compensated. Okay? So the blood pressure, all right, is going to be lower in the lungs than it is in the systemic circulation.

Therefore, the right ventricle is not going to be as muscular, and it's not going to require as much oxygen. Therefore, risk of heart attack on the right side of the heart is minimal. It's the left side that you have to worry about. Okay? That's where heart attacks occur. All right. So we've established blood flow through the heart. Any questions on that?

All right. Then we need to talk about heart valves. Okay. So when the ventricles contract, all right, when they contract, the walls come together, all right, and they pump blood out, okay, and once again, we're talking about the left ventricle for a moment, you pump blood through the aorta, okay, when the left ventricles then are complete systole, they've contracted and all the blood is out, but they can get out, what happens, they relax. When the walls of the left ventricle relax, how come blood doesn't flow from the aorta back into the left ventricle?

>> STUDENT: [Inaudible].

>> PROFESSOR: Right. It's the aortic valve. Okay? And it closes. Now, what does close mean? It basically means that the valve is made up of three semi-lunar flaps. They're shaped like a moon or a cup, and as blood goes through the aorta, the semi-lunar valves, are thin walled, are pressed against the aorta. As soon as the ventricles stop contracting and they start to relax, blood will start to flow back into the left ventricle. When that happens, it fills the semi-lunar valves up, okay, they're like cups, and there's three of them.

One percent of the population has aortic valve that has two semi-lunar cups instead of three. Okay? One percent. That percent, later on in life, is subject to developing aortic stenosis, basically where the valves harden, and they may not open totally, and they may not close totally, and sometimes they have to be replaced.

All right. And the same thing for the right ventricle, all right? The right ventricle pumps blood where? To the lungs. It pumps it through the pulmonary trunk, so when it gets totally contracted, at the end of systole, needs to relax, there is three semi-lunar valves, all right, the pulmonary valve, they close up and they prevent blood from flowing back into the ventricles. All right? Now, the ventricles receive blood from where? Where do they receive
blood from?
>> STUDENT: [Inaudible].
>> PROFESSOR: From the atrium. Okay? Directly from the atrium. So what goes from the atrium into the ventricles. When the ventricles contract during systole, what prevents the backflow of blood into the atrium? Set of valves, the AV valves, atrial ventricular valves. On the right side of the heart, there's three flaps to what we call the tricuspid valve. On the left side of the heart, there are two flaps, all right, or leaflets, to the mitral valve. Okay? So on the right side, in between the right atrium and right ventricle, is the tricuspid valve. On the left side, there is the mitral valve. So, during ventricular systole, what happens is these leaflets meet and prevent blood from flowing back into the atrium. All right? Any questions on that?
Okay. You'll see diagram, in the outline later on, they will show you the mitral valve, and then there are what we call violin strings called chordae tendineae. Okay? These chordae tendineae attach to the leaflets, okay, and then they're attached to papillary muscle, projection from the muscular wall. All right? And what happens is when the ventricles contract, the papillary muscles come closer together and the leaflets close, and that's how they work, and we'll go more into detail with that. All right? So, we've got the anatomy down, okay, the circulation of blood throughout the heart. Now, remember that the heart is made up of four chambers, okay? And the purpose of the heart is to receive blood from the body, pump it to the lungs, put oxygen into the blood. It comes back to the heart, gets pumped out to the body. The heart muscle itself needs oxygen, and it does not get it from the circulation. It has its own independent circulation. Okay?
When blood leaves the left ventricle and goes through the aorta, the first two vessels that branch off the aorta are the right and left coronary artery. Okay? Left coronary artery brings blood to the left side of the heart. Right coronary artery brings blood to the right side of the heart. All right?
There's the left anterior descending artery, which also receives blood from the left coronary artery. All right. They have a diagram in the book. We'll go over that diagram.
And so what happens then is the branches -- those two branches of the aorta, they're the very first branches, okay? So it's very interesting the way the human body is made. The heart requires a lot of oxygen, okay? So the blood, with the highest oxygen content in theory is going to be the blood that's closest to the aortic valve. So the vessels that are closest to the aortic valve, we see them
most -- or most highly oxygenated blood, it's going to be the coronary arteries. Okay?
Then, as you go further out from the aortic valve, you're going to have blood to other vessels, whether it's going to be the thoracic arteries, the internal mammarys, the spleen, liver, later on further down, the kidneys, the femoral arteries. So the coronary arteries bring blood to the heart, and then the blood then returns to the heart through the coronary sinus. All right? Which is a smaller vessel, empties into the right atrium. All right?
Now, the division between the right and left ventricle, okay, is called the septum, ventricular septum, and that's a wall in between the left and right ventricles. There's also a wall in between the right and left atrium. All right?
And that's called the atrial septum.
Now, kids can be born, children can be born with defects in the septum, and the defects can be very small, can be pinhole size, or they can be totally absent. Sometimes they're not diagnosed until later on in life. A lot of times, they can be diagnosed at birth.
So if someone has a defect, all right, a communication, a small pinpoint hole in the ventricular septum, okay, which, then, you have blood that can flow from one ventricle to the other during systole when the ventricles are contracting. So my question is, during systole, okay, someone has a ventricular defect, will blood flow from the right ventricle to the left or from the left ventricle to the right ventricle? Yes?
>> STUDENT: Do the ventricles contract at the same time?
>> PROFESSOR: Yes. Yes. Yeah, they contract at the same time. So what would be the direction of blood flow? Right to left or left to right?
>> STUDENT: Left to right.
>> PROFESSOR: Good. Left to right. Why?
>> STUDENT: [Inaudible].
>> PROFESSOR: Because the left ventricle pumps blood at a higher pressure. All right? The blood pressure in the pulmonary circulation is a lot lower than the blood pressure in the circulatory system. All right?
So now, a murmur is blood flow over an abnormal area. Okay? So when children are diagnosed with heart murmurs, basically you're hearing the cardiologist hears blood flow over an abnormal area so they can determine by the sound and where it occurs in the cardiac cycle as to what type of defect is present. All right? And very often, the smallest defect makes the loudest noise. If you want to think about a hose, garden hose. Turn on the faucet. Water comes out of the garden hose, okay? So you hold the garden hose and maybe it comes as a stream and it hits the ground five, six feet from where you're standing. Put your finger over the aperture,
the opening of the garden hose, and reduce the size of the
opening by half or three-quarters and how much further does
it go? It goes much, much further. It's the same thing
about defects. Okay? The smaller the defect, very often
the louder the noise. All right. So we've talked about the
anatomy of the heart. You guys know the left ventricle is
more muscular, does more work than the right ventricle,
pumps blood at a higher level. We've talked about systole,
diastole, systolic blood pressure, diastolic blood pressure,
and we have a couple other things to talk about. Okay?
In utero, within the uterus, when the baby is in the uterus,
all right, are the lungs opened up and expanded or are they
unexpanded?
>> STUDENT: Unexpanded.
>> PROFESSOR: Right. They're unexpanded. So what happens
to the blood when it's in the right ventricle? When it's in
the right ventricle, it's supposed to go to the lungs. But
the lungs aren't expanded, so then how does the blood, you
know, what's the blood do? How's it get to the left atrium?
Umbilical. Anybody know? All right. There's two passages
of communication. One is called the ductus arteriosus, and
what that is is that's a communication, or a duct, between
the pulmonary trunk and the aorta. So the right ventricle
contracts, pumps blood through, or into the pulmonary trunk,
and then once blood's in the pulmonary trunk, it goes
through the ductus arteriosus into the aorta. All right?
The other one is the foramen ovale, and what that is is a
communication, all right, in the septum of the atrium. So
it's a communication, it's an oval window, communicating
between the right atrium and left atrium. So that blood
that comes into the right atrium, some of that blood can go
then into the left atrium and from the left atrium, it goes
into the left ventricle. Yes?
>> STUDENT: What happens to those structures when --
[Inaudible].
>> PROFESSOR: When the baby is born, and they initiate the
first breath, most the time they close.
>> STUDENT: So they're still there?
>> PROFESSOR: They are still there, okay, the foramen
ovale, if you do anatomy of the heart, you can find the
little depression. Where it was.
If ductus arteriosus, in theory, should close. It usually
does. It may not close right away. It may take a day.
Most of them close right away with the first couple of
breaths. There is an entity called PDA, patent ductus
arteriosus, where it doesn't close. Patent. It stays open.
It's patent. And so with PDA, you can give them medication
to try to close it. If that doesn't work, you can do
surgery.
So all right. Now, as far as how's the heart function? It
functions with a pacemaker. It's called the sinal atrial node and it's in the right atrium. And the impulse, then, okay, the electrical impulse is conducted from the sinal atrial node to the AV node, atrial ventricular node, located at the junction of the atrium and ventricles, it then goes down the side wall of the septum, okay, and into the bundle branches, there's right and left bundle branch, and electrical impulse then terminates in the purkinje fibers. All right? So each cardiac muscle fiber is innervated by a purkinje fiber. So the coordination of the heart muscles is accomplished through an electrical conducting system, and it is independent, all right? It has its own pace. Now, it can be modified, okay, with you guys I think pretty much all you heard of the fight or flight response, okay, higher epinephrine levels can stimulate the heart, greater cardiac demands, and as a result, heart rate increases. Okay? But, the important thing is is that the muscle fibers function in a coordinated fashion. All right? There's two terms, flutter and fibrillation. All right? Sometimes you hear about atrial flutter or atrial fibrillation, and that's where the atrial muscle fibers do not contract in a coordinated fashion. Usually that can be treated with medication, and the person does well. Ventricular fibrillation is different. Ventricular fibrillation is when the cardiac muscle fibers contract totally independently and out of sync, okay, out of organization. I've seen it demonstrated, and it looks like a bag of worms. That's the only way I can describe it. It's a bag of worms, where it's all worms just moving. It's the craziest looking thing. All right? Ventricular fib, or V-fib, as we call it, is incompatible with life. It's incompatible with life. And if you ever watch these medical shows, hospital shows, the ER, which I do not watch at all. I guess I've seen too much of that stuff, so I'm done with it, but if you ever watch, you know, very often, why do they give someone the paddle? Well, they give them the paddle because they're asystole or they're in V-fib. They've got ventricular tachycardia or they're in V-fib and they want to shock the heart to go back to a normal sinus rhythm. Okay? And what they'll do is very often they'll give them an intravenous dose of medication to make the heart less irritable, and then they'll shock them. Anybody know what illegal drug increases the risk of V-fib? Cocaine. Right. That's why cocaine has some unexplained deaths. Why people die. Apparently good health, but what it does is increases heart rate, increases ventricular contracts, the rate and force of ventricular contracts, which requires greater oxygen and it makes the myocardium more irritable. When it makes it more irritable, you have a
greater risk of ventricular fib occurring. All right? So let's see, we've discussed the valves, the electrical conducting system. We've talked about foraminal valley, and the ductus arteriosus. Okay. So let's take a look at this. So now, this will be a little bit of a review from what we talked about.

So the function of the heart is muscular pump. It propels blood through the lungs and tissues. All right. So what's heart disease? Disturbance of function. So where's the heart located? It's located within the mediastinum. The mediastinum is the central portion of the chest cavity. Okay? So you've got your right and left lungs, lung fields and you have your mediastinum, which contains the heart, the aorta, also the esophagus, lymph nodes, all right, let's see. Now, also, something else I wanted to say is that the inner most lining -- like, if you do a biopsy or cross section, all right, of the ventricle, the inner most lining is called the endocardium. Okay? Then you have the myocardium, the heart muscle, and the outer most layer or covering is the epicardium. And the heart is located within the pericardial sac, or pericardium. The pericardial sac is lined with a layer of epithelium that's derived from the mesothelium. It's flattened cells, they're shiny. It's a shiny surface. It's one cell layer thick. And the purpose of that covering is to reduce the friction between the heart and the sac when the heart contracts. Okay?

Areas in the body where there's movement, they are covered by mesothelial derived tissue. The heart is an example. The pericardium. So the outer most portion is a shiny layer of mesothelial derived tissue, and the same thing for the lining of the sac.

We have the same thing in the abdomen. All right? The abdomen is the organs. The intestines are covered with what we call peritoneum, and the abdominal cavity, the wall is covered with peritoneum, also lined with peritoneum. When it covers the organ, we call it visceral peritoneum. When it lines the abdominal cavity, we call it the parietal peritoneum. And, of course, what word do we get from peritoneum? Anybody? What is it? Right. Peritonitis. Right. Peritonitis. Anybody want to tell me one of the common causes for peritonitis? A ruptured what? Valve, yes. I'm thinking specifically appendix. Okay? Somebody gets an appendix, appendicitis, if it ruptures, pus leaves the appendix and enters the peritoneal cavity and enters the peritoneal cavity, itis means inflammation, so you've got inflammation of the peritoneum, we call it peritonitis.

All right. So let's see here. So the heart coverings, you've got the pericardium, double-walled sac, the outer layer of tough connective tissue. The visceral layer of pericardium covering myocardium. You know, your visceral,
or -- and then you have your parietal. Layers of the heart wall, the epicardium is the outer layer. You got your myocardium, which is the muscular layer, the middle layer, and then within it you have the endocardium. Okay. So the chambers in theory have no direct communication. In other words, the right and left atrium don't communicate, right and left ventricles don't. Okay? And let's see, the right half pumps blood into the pulmonary trunk. Okay. Another thing I want to bring out, okay, the pulmonary circulation, and the systemic circulation, you told me that the -- okay. The right atrium receives blood from the superior vena cava and inferior vena cava. It brings blood from the veins of the body, it's low in oxygen. Okay?

So the veins then carry blood that's low in oxygen, right, from the body, and the arteries carry what type of blood? It's going to be high in oxygen. Right. Oxygenated blood. All right. So therefore we say arteries carry blood that's high in oxygen, and veins carry blood that's low in oxygen. A more better generalization is that the arteries carry blood away from the heart, veins carry blood to the heart. The blood pressure in the vessels, is it going to be greater in the arteries, or the veins? Great. Greater in the arteries. The arteries are thicker, more muscular. Okay? And so then what about in the pulmonary circulation, when the right ventricle pumps blood to the lungs, okay, to get oxygenated, what's the name of the vessels it goes through? It goes through the pulmonary artery. But, the pulmonary artery contains blood from the right ventricle. Is that going to be high in oxygen or low in oxygen? Low. Right. So the pulmonary circulation is reversed. The blood in the pulmonary artery's going to be low in oxygen, and the blood in the pulmonary vein's going to be high in oxygen, all right? So you need to remember that.

All right. So let's see, atrium are thin walled. Okay. Blood flow into the right atrium from the superior vena cava, the inferior and the coronary sinus. Okay. So the cardiac valves prevent the flow of blood in one direction, right. You got your AV valves, which is going to be the mitral valve, and the tricuspid valve. They're flaps. All right. They prevent the backflow of blood. Now, the semi-lunar valves are the valves of the aortic valve and the pulmonary valve. Okay? So the pulmonary-systemic circulation. Let's see. Blood returns to the heart. Okay. We've talked about this. Going to be low in oxygen. High in CO2. Pulmonary circulation, oxygen poor in blood enters the right atrium. Question.
>> STUDENT: [Inaudible].
>> PROFESSOR: You know what? No. It has two cusps and I refer to it as the mitral valve and on the test I will refer to it as the mitral valve. Okay? Oxygen, poor blood enters the right atrium, goes to the right ventricle, and when it goes to the right ventricle, it goes through the tricuspid valve. Okay. To the right ventricle, pulmonary artery to the lungs. And then oxygenated blood leaves the lungs, goes through the pulmonary veins, left atrium and then left ventricle through the mitral valve and then to the aorta and the rest of the body.

All right. Here's a picture of the chordae tendineae. Remember I told you that there are pictures of the chordae tendineae, and here we go. Right here. These are what we call violin springs, all right, and they attach to the flap here of the valve, and then over here is the papillary muscle. When the ventricles contract, back here is the wall of the ventricle. When the ventricle contracts, these papillary muscles come closer together, right here and here, and allows it to close. One of the concerns is that if someone has a heart attack, all right, basically we haven't talked about it yet, but a heart attack occurs when there's lack of oxygen to muscle fibers. When there's lack of oxygen to muscle fibers, the muscle fibers die. They become necrotic and they die. All right? Very often, those fibers are replaced with scar tissue. Sometimes, it's hard to replace it with scar tissue. Someone gets an infarction, okay, they get an infarction -- let me see if I can get this up here. There we go. They get an infarction that involves this area of the ventricular wall down here. It can infarct the papillary muscle. When it infarcts the papillary muscle and there's necrosis, tissue dies. As a result, what? These chordae tendineae flap in the breeze. As a result, this valve doesn't close properly. So as a result, they can develop cardiac insufficiency. When the left ventricle goes to contract and close, that flap doesn't really meet the mitral valve and as a result, blood flows from the left ventricle back into the left atrium. So what happens then is you have decreased cardiac output, and if the cardiac output is decreased enough, you're going to have a decrease in function. Someone who has decreased cardiac output, they may not be able to walk from the end of the parking lot to our building. They may not be able to go up a flight of stairs. They may have to take the elevator and sometimes it can be worse, so sometimes these valves have to be replaced. All right. Also, here you have, on the lower picture, you have a picture of the aortic valve, okay? And you can see that like there is the semi-lunar, or cusps. Okay? And they point to the opening of the right and left coronary artery. Here we go. You got here. You see that little bit
of opening right there, and then right here. And then you see how they meet. Okay? And these are -- this area right here is depressed, and that's what fills up with blood. All right. So the coronary circulation is the main blood supply of the heart. Shortest circulation in the body, and the myocardium is very thick, and what they're saying is basically it's so thick that you just can't have nutrients diffuse from within the ventricular chamber through the full thickness of the wall, so the aorta branches to the right and left provide the coronary arteries. Blood passes through the capillary beds of the myocardium and then it collects in the cardiac veins into the coronary sinus, which empties into the right atrium. All right?

So this is what we talked about. Now, let's see, the right coronary artery supplies the back wall, and the posterior part, posterior means back, so the posterior portion of the intraventricular septum. Left coronary artery, all right, branches into the left anterior descending artery, which is significant, all right, it supplies the front wall of the -- especially the left ventricle, and also the left circumflex. Circumflex means it curves around to the back of the heart. All right? So it supplies the lateral wall. Lateral wall is going to be the left ventricle. Anterior, the left ventricle also in the front.

All right. So adult cardiac muscle does not proliferate. Nervous tissue, okay, nervous tissue, muscle fibers really do not proliferate. They don't get replaced. All right? All right. People who have spinal cord injury, paraplegics, quadriplegics, very hard for them to regain their function because nervous tissue really does not regenerate. That's the problem with heart attacks. When heart tissue dies, it's not replaced by a healthy myocardial fibers, it's replaced by fibrous tissue, fibrotic tissue. As a result, this fibrous tissue does not stretch, and it doesn't contract well. So people who have myocardial infarctions, the dead tissue is replaced by scar tissue, and as a result, they can have decreased cardiac output, all right, because it doesn't stretch as well, and it doesn't contract as well. All right. So most areas of cell death are repaired with non-contractile scar tissue.

So here you have your diagram, you see your left coronary artery with the circumflex, the left anterior descending, and the right coronary artery at the posterior descending. All right. So angina pectoris. What's angina pectoris? That's chest pain, and it's chest pain that comes from a temporary decrease in blood flow to the cardiac muscle, and we call that temporary decrease, okay, and myocardial ischemia, i-s-c-h-e-m-i-a. And the big thing to understand is that ischemia can occur from a decrease in oxygen, but
there's not infarction. There's no tissue death. So when someone has a myocardial infarction, when there's prolonged blockage of blood flow to an area of myocardial fibers, what happens is you get tissue death. When tissue dies, the cell walls dissolve, and cardiac enzymes, enzymes that were within the cardiac muscle fibers, are then released into the circulation. And the three that they examine the most are troponin T and troponin I. And creatine kinase. It's called CK, and then specifically MB. Yes.

>> STUDENT: [Inaudible].

>> PROFESSOR: Yep. It's the resulting. Okay. So the tissue stops getting blood. It dies, and then it starts to disintegrate, so that's necrosis. Okay?

So with angina pectoris, there is no elevation of cardiac enzymes. The myocardial infarction, there is. Now, with angina pectoris, chest pain, it's typically brought on by activity or exercise. Okay? Increased activity, increased exercise, and when you have increased activity, increased exercise, what's that do to oxygen requirements? Does it increase it or lower it? It's going to increase it, so you're increasing cardiac demand. You're increasing oxygen demand, and if the vessels can't bring the amount of oxygen that it needs, then you get ischemic changes. All right? You get angina pectoris, chest pain. Now, what's the most common medication they use to relieve chest pain, or angina pectoris? Not aspirin. Anybody? Nitroglycerin. All right. How many here have heard of nitroglycerin before?

Right. And typically, it's administered what we call sublingually. Underneath the tongue. A small little pill, they put it underneath the tongue, and what's it do? It causes vasodilation. It causes the coronary arteries to dilate a little bit. By causing dilation, you then increase blood flow to that area. When you increase blood flow to that area, then the tissue gets more oxygen, and ischemic changes go away. Okay? And there's different degrees of angina pectoris. There's related to physical activity. There's angina pectoris at rest, and then there's prinzmetal, where people are sleeping, and they're woken up with severe chest pain. Okay?

So in a lot of ways, you can look at angina pectoris as a warning signal, okay, as a warning signal for the heart. So what's causes? Narrow coronary arteries from arterial sclerosis, which we'll talk about, and stress induced spasm of the coronary arteries. Now, remember I said, it's reduced blood flow. The next line, prolonged coronary artery blockage. We're not talking about blockage, we're talking about reduced flow. Okay? Maybe the coronary arteries go into spasm a little bit, they narrow down, so
not as much blood flow to an area within the heart. Okay? But not a blockage.

All right. So the conducting system of the heart, it's a group of specialized muscle cells that initiate electrical impulses. The impulses are initiated in the sinoatrial node, in the right atrium, near the opening of the superior vena cava. And what happens is then the cardiac muscle depolarizes, the contraction is intrinsic, it doesn't depend on the nervous system. It can be modified or influenced, okay, by the nervous system, but it does not -- it's not controlled.

So here you go. Sinoatrial node. The impulse is generated there. It then goes through the intranodal tracks and to the AV node, and then the impulse leaves the AV node through the bundle of his, and then you get your right and left bundle, right and left bundle branch. One of the typical syndromes, there are complexes that diagnose people with cardiac issues can have a right bundle branch block or a left bundle branch block. You can see that if you have, okay, an MI, okay, a cardiac myocardial infarction, in an area of the heart where the conducting system is, it can affect cardiac function.

I'm trying to get this pointer to come up, and it's not going to come up. But at the beginning of the left bundle branch block, if you have an infarct that occurs in that area with tissue death, it can affect the function of the heart.

>> STUDENT: [Inaudible].

>> PROFESSOR: Electrical fibers. They're really not nerve fibers.

All right. So the cardiac cycle consists of all events associated with blood flow through the heart during one complete heart beat. So you've got atrial systole, where the atrium fill up with blood. Sorry. They contract, they fill up with blood, they contract, and then they relax. Okay? Atrial diastole, and then you have ventricular systole and ventricular diastole. So systole refers to the contraction period. Diastole refers to the relaxation period.

Cardiac output typically, five liters of blood are pumped per minute, okay, by each ventricle.

Blood pressure. Blood pressure is the blood flow in the arteries which results from the force of the ventricular contraction. Pressure is highest when ventricles contract. It's lowest when ventricles relax.

All right. EKG. It measures the electrical activity of the heart, a diagnostic tool. It is way too complicated to go into a discussion on EKGs. All right? There are basically three waves. You got your P wave, and the QRS complex, is sort of like a wave, and then you got your T wave. Okay?
The P wave is when the atrium basically depolarizes, so you've got atrial contraction. All right? The QRS refers to ventricular systole, when the ventricles contract. And then the T wave refers to when the ventricles relax. Okay, during diastole.

And you can tell a lot from EKGs, really a lot. The PR interval basically refers to the time from the peak of the P wave to the top of the QRS complex, which is the time for deep polarization. Basically the electrical impulse is initiated in the P wave passes during time to the QRS. Okay?

So and the EKG can detect disturbances in the rate, rhythm, conduction, muscle injury, extent of muscle damage. For example, I've seen you can have -- when people have atrial flutter, they don't really have a clear cut P wave, because remember, flutter or fib, it's not contracting in an organized fashion. But they have a basic rhythm, the QRS complex. All right?

Now, if the QRS complex is not at a sufficient speed, all right, then cardiac output is going to be decreased and people may have passing out episodes, syncopal episodes, and then they may need a pacemaker. All right. A pacemaker is instilled so that you can control the heart rate and usually it's instilled because the heart -- the basic -- there's cardiac damage, and the heart beats at a slower rate than is acceptable for their level of function.

All right. So cardiac arrhythmias. We're going to go into that. Arrhythmias refers to disturbance in the heart rate or rhythm. There's atrial fib, ventricular fib and heart block. All right. So as I mentioned, atrial fib, it's where the atrium basically quiver, the muscle fibers contract independently.

And the ventricles may be irregularly and faster, shortening diastole. Okay? Ventricular fib, ventricles are unable to contract normally. It's incompatible with life. Heart block, complete or incomplete, refers to a delay or interruption of the transmission from the atrium to the ventricles. Okay?

And it can be from arteriosclerosis, which we'll talk about later.

So here, what they've done in this chart is they have said, likened the heart to a pump. And with a pump, you can have mechanical abnormality, faulty construction, faulty valves, plugged fuel line, overloaded pump or malfunctioning pump. So the faulty pump construction refers to congenital heart disease. And, you know, there are children who are born with heart disease, we call it basically we say heart disease referring to heart defects. May be a simple little thing like an atrial-septal defect that's really small. Doesn't really interfere with cardiac function that much.
Or, they can be missing a septum, and the ventricles. So instead of having two ventricles, they have one, because they're missing that septum that separates the right ventricle from the left. Obviously that type of child is going to be compromised because they're going to be receiving oxygenated blood and de-oxygenated blood, all right, in one chamber, pumping it through.

So there are also, with congenital heart disease, there's different complexes, where there's, like, three or four features, not just one abnormality, but three or four abnormalities. There's one where the aortic valve receives blood from the left ventricle, but also overrides the septum, so it receives blood from the right ventricle also. Okay?

And in managing cardiac abnormalities, the degree of oxygenation in the arteries is a key component. Okay? So very often, when kids are born with congenital heart disease, they prolong or put off doing surgery until it's really necessary. And the reason being is that very often these kids need surgery more than once. Okay? So they put it off as long as possible. Hopefully, the growth requirements will be met, and then as the growth parameters are not met, then very often they will operate on the child to do the repair, with the hope that the child will then take off and be, you know, benefit and continue to grow for quite a long while.

All right. So faulty undirectional valve means valve disease. It can be from birth. It also can be from a heart attack. Remember I told you how the chordae tendineae can be injured, depending on where the area of necrosis is. Rheumatic fever is another disease that can affect the heart valve. I talked to you, already told you about the aortic valve. Normally there's three semi-lunar valves or flaps, but one percent of the population is born with two flaps. And they have a greater risk for developing aortic valve stenosis, and insufficiency. Insufficiency is where it doesn't meet. Doesn't close well.

All right. And the coronary heart disease basically, where the coronary arteries have blockage and prevent or slow down oxygenated blood to the muscle. Overloaded pump would be hypertensive heart disease. I told you you can get a chest x-ray on someone, check the size of the heart. If they have cardiomegaly, or cardiac hypertrophy, you know they've had chronic hypertensive disease.

And then there are certain conditions where they have primary myocardial disease, disease of the myocardium. All right. So congenital heart disease. What are some of the causes? German measles, down syndrome, high percentage of down syndrome babies have cardiac disease.
Certain drugs can affect cardiac disease. Years ago, there was a -- the Phen-Phen diet. Very successful. People lost a lot of weight. Very high percentage developed cardiac abnormalities of the valves and needed cardiac surgery. All right?

Also, congenital heart disease can be determined to cause by genetic factors. The genetic factors that determine cardiac heart disease, okay, are multi-factorial. It's not just one genetic factor. And so you can have a family history of heart disease, all right, and -- but, the abnormalities can be different. All right? Grandparents -- grandmother may have something, her children don't, but her grandchildren do. And she may have a septal defect, and the children could have a mild valvular defect. It's like -- it's hard to tell.

All right. So fetal bypass channels fail to close normally. I mentioned this, patent ductus arteriosus, or patent foramen ovale. You can have combined septal defects. You can have obstruction, narrowing, of the pulmonary valve. Aortic stenosis, where there's basically narrowing or doesn't function well. Also, coarctation of the -- that's a narrowing of the vessel itself. All right? Tetralogy of Fallot is basically a collection of cardiac abnormalities that are present at birth. That's one of the complexes that I mentioned, and it affects the great vessels that are in an abnormal location.

All right. Let's see, so for prevention, protecting developing fetus from intrauterine injury. Right. That's why the concern about German measles and medication in pregnancy to prevent heart disease.

All right. So valvular heart disease, rheumatic fever, causes rheumatic heart disease. You have non-rheumatic aortic stenosis, I mentioned that to you, two flaps. Mitral valve prolapse is one, and infective endocarditis.

All right. Let's see. So valvular heart disease. We're going to take a break soon. Rheumatic fever, it's commonly encountered in children, and the problem is it is caused by strep. So how many here have had a strep throat infection? All right. Everybody. Right? Anybody not ever have a diagnosed strep infection? Really. You guys have never had it, huh? Okay. All right. So most people get it, and they get more than one. Okay? And the treatment for strep throat infection is what? Anybody know? What type of antibiotic? Amoxicillin or penicillin. Penicillin derivatives. All right?

And when you take a child to the pediatrician, they have a sore throat, what happens? The doctor does a culture, all right, a healthcare person does a culture, and on the back of the throat, they put it in a culturette, and it gets sent to the lab. At the lab, they take that cotton tip
applicator that was touched to the back of the throat, they plate it on a special media, and they see what grows out, all right? It takes 24 to 48 hours. They see the organism that -- the predominant organism that grows out and what it's sensitive to. The way they determine sensitivity is they put like a couple disks of different antibiotics and they'll see which disk the organism will not grow in there. And typically, beta strep is very sensitive to penicillin. It's not resistant to penicillin. All right?

So if it comes out beta strep, then what happens is the lab reports it to the doctor. The doctor then prescribes amoxicillin. Now, in this day and age, what happens is child comes in, sore throat, doctor does a culture, and also the doctor does something else. Anybody know what else they do? Anybody been with a child or niece, nephew, or whatever? They do a rapid strep. Rapid strep is an office test, which is quite accurate, over 90% accurate. If the rapid strep comes out positive, they send the mother and child out with a prescription for antibiotics so the child starts right away. Okay?

And in this way it saves waiting a day or two for the lab to call with the strep, whether it's positive or negative, and then trying to contact the parents, get the prescription to the pharmacy, picked up, the whole bit. And so because it's a much more efficacious way of dealing with it, what happens is the child gets treated. Okay? When it needs to be treated.

The problem is if the child's not treated appropriately, then they have a strep infection, and they run the risk, then, of getting sequelae of strep infections. The first sequelae is scarlet fever. It's a sandpaper rash and occurs a month, two months, three months after the initial infection. Red rash, lasts for a while, and then it goes away. And then what happens later on is they develop rheumatic fever, and what rheumatic fever is, it's basically, it's an unusual entity in that you have circulating antigen antibody complexes. Okay? It is antibody against strep antigen. And when antibody binds to antigen, what gets activated? Compliment. So you set up an inflammatory process. As a result, these children have rheumatic fever, and there are certain organs that are affected. The brain is affected. They develop chorea. Chorea are involuntary movements, expressions. They can develop inflammation of the mitral valve. The antibodies associated with rheumatic fever cross react with antigens on the mitral valve, so it becomes thickened, scarred, doesn't open all the way, doesn't close all the way. Those antigen antibody complexes that are circulating in the bloodstream get deposited where? What organ filters the blood? The kidney. So these antigen antibody complexes get stuck in
the kidney. And I told you, when antibody binds to antigen, compliment gets activated, you have an inflammation of the kidney as a result. The medical term is glomerulonephritis. All right. So and it affects the joints also. So they get a fever, inflammation of connective tissue, affects the heart, affects the joints. And they are severely compromised.

All right. So rheumatic fever, anti-Streptococcal antibodies against strep antigens, and what happens is they cross react with similar antigens in the host tissue. So that's why it also affects the mitral valve. I've taken care of patients from emerging countries, developing countries that had rheumatic fever. We don't have -- thankfully, because we have good medical care here, rheumatic fever is not common at all. Okay? But, in other countries, where there's not as good medical care, rheumatic fever is more common, and as a result, with medical care, poor medical care, what happens is these children are not treated properly. They get rheumatic fever, and then that rheumatic fever, those antibodies cross-react with the antigens in the mitral valve. The mitral valve gets scarred and the mitral valve needs to be replaced. Okay? So they need to have heart surgery. And I've taken care of patients who were pregnant who needed that. And we had to deliver them earlier because of the fact that their cardiac output was decreased due to the complications from the mitral valve.

So let's see, we'll go through this slide a minute. And the antigen antibody reaction, what it does, injures connective tissue, causes fever, you get scarring of the heart valves, you get death from severe inflammation, acute heart failure and once you get it, recurring strep infections can lead to additional damage.

So let's see. We'll take a break here.

(Part 2 starts now).

>> PROFESSOR: All right. So with valvular heart disease, we're going to discuss these. So rheumatic fever is commonly encountered in children. It's not a bacterial infection, really, when we talk about rheumatic fever, but it's an immunologic reaction that develops weeks after an initial strep infection. It's complication of group A beta hemolytic strep. They first get the sore throat. Later on they get the scarlet fever.

Now, a small percentage get the scarlet fever, and a small percentage get rheumatic fever. It's not a hundred percent. Believe me. And there's fever and inflammation of connective tissue, heart and joints. It affects multiple joints. They get arthritis.

All right. And basically, it's the anti-Streptococcal antibodies that react against the strep antigens. And this
antibody-antigen reaction can injure connective tissue, all right? It causes a fever, and as a result, you get healing with some scarring. You can -- patients can die from this. Okay? And with recurring strep infections, they get the disease progresses. All right? So, complication, you get scarring of the heart valves, primarily affects the mitral valve and the aortic valve, and what happens if the valves don't close properly, you get what we call regurgitation, so you get aortic regurge, where the aortic valves don't close, so what that means is during diastole, when they should be closed, they don't close totally, so you get regurgitation of blood back into the left ventricle. Or you get stenosis, where the aortic valve is narrowed. Okay? And impairs cardiac function, puts a strain on the heart, and eventually leads to heart failure. You know, it's what happens, left ventricle is trying to pump blood out of the left ventricle, and blood keeps falling back into it. All right? So prevention, you treat the beta strep promptly, and penicillin is given for, you know, kids with the infections. All right. Non-rheumatic aortic stenosis, all right, this they say occurs in 2% of the population, and it's secondary to two cusps of the aortic valve. Not three, but two. People who are born -- I said it was 1%. They say 2%. It's around 1.5, 1%. And basically, instead of having three cusps of the aortic valve, there's just two. And over time, it gets stenosed. It gets scarred and stenosed. So people later on in life, what happens is they end up with stenosis. It doesn't open up the whole way. It becomes scarred, calcified, it doesn't open the whole way and by not opening the whole way, what it does is it creates decreased cardiac output. Okay? Eventually it becomes rigid and doesn't function well. Nonrheumatic aortic stenosis, all right, so let's see. Once again, they undergo scarring and they become fibrotic and rigid. Okay? Puts an increased strain on the left ventricle, hypertrophy and heart failure. All right. So here's an example, over here, on the top right, where you see some early scarring of the aortic valve. It has two cusps, not three. Then on the top left, you see mitral valve damage. Notice the irregularities, those raised bumps. Those are what we call vegetations, and those are accumulations of platelets, thrombin, antigen-antibody complexes, inflammatory white blood cells that cause the mitral valve to become thickened, and you see the chordae tendineae protruding from the valve leaflets. So let's see, valvular heart disease. Mitral valve prolapse. All right. What this refers to, remember I told
you the mitral valve has two leaflets. When the left ventricle contracts, the two leaflets meet, and it prevents blood from flowing back into the left atrium so all the blood flows out of the -- out of the left ventricle through the aortic valve into the aorta.

With prolapsed mitral valve, what happens is the valve leaflets don't meet properly. One of them falls back or prolapses into the left atrium a little bit. And we don't know exactly why this happens, but it may be that the one valve is a little -- the one leaflet is a little too big. It's a little redundant, so it falls back, and therefore, so they don't fit tightly, leaks back into the left atrium, you get what we call mitral regurge. Blood passes during ventricular systole, some of that blood passes through the mitral valve and the left atrium. So when you listen, there's a click sound in systole followed by a faint systolic murmur, okay, which comes from the reflux of blood in between the closed leaflets. It can be diagnosed by an echocardiography, and people with prolapsed mitral valve, if they have blood flowing back in the left atrium, what happens is they recommend that they get antibiotics for any type of surgical procedure or dental procedure. The concern is when you have any type of cardiac abnormality, maybe atrial septal defect, ventricular septal defect, you then have surgery or any procedure, where bacteria can gain entrance into the bloodstream, what happens then is you're at increased risk for valvular damage and a heart infection.

All right. So here you have the normal on the left, okay, you see the chordae tendineae. Papillary muscle, and then you see the prolapse. Okay? Mitral valve prolapse, where a little bit of blood will regurge back into the left atrium. So valvular heart disease, also you can get an infective endocarditis, which can infect the mitral or aortic valves. It can be chronic, low -- with a low infective endocarditis. It doesn't have to be a very virulent. Caused by organs of low strength. And may cause mild infections. But what happens is platelets and fibrin get deposited on the damaged valves from the endocarditis, and this, then, serves as a site for bacteria to implant. Thrombi can form and the concern is when you form a thrombus, which is clot, it can break off. When it breaks off, it then becomes an embolus, travels in the bloodstream and will eventually block blood flow to some part of an organ somewhere and cause an infarct. Okay? Once again, prophylactic antibiotics should be given for dental or surgical procedures.

Infective endocarditis, acute infective endocarditis caused by a highly pathogenic organisms, commonly staph, all right, and the at-risk group, IV drug users. Okay?

So they're going to use illegal drugs to be injected. Okay? Now, so these type of drugs, they're injected. Where are
they injected? In arteries or veins? They're in veins. So blood, all right, within the veins, where does it go?

>> STUDENT: [Inaudible].

>> PROFESSOR: To the heart. What side of the heart? The right side. Great. So it's going to enter the right atrium, and then what happens, it passes through what?

>> STUDENT: [Inaudible].

>> PROFESSOR: Think back. What's it pass through? Tricuspid valve, into the right ventricle, so it's the tricuspid valve that IV drug users tend to have being scarred and inflamed, okay, and it becomes stenotic. So intravenous drug users, affects the tricuspid valve instead of the mitral or aortic valves. And it's because they use unsterile materials, will even take drugs by mouth, you know, pills by mouth, grind them up, and then inject them, trying to get the high. And of course you're injecting in foreign material into the bloodstream. Okay?

All right. So here you see basically a clot forming here. The vegetation. All right. And also you see a perforation of the leaflet on the right side.

All right. So now, coronary heart disease. It refers -- arteriosclerosis refers to scarring or hardening, basically it's an old term. It refers to deposition of lipid within the coronary arteries. Okay? The deposition of lipid, lipid deposits, lipid plaques, narrows the lumen of the coronary arteries and reduces blood flow through the coronary arteries to the myocardium. So what happens? The initial event is the endothelium, which is the inner most lining of the artery gets injured. We don't know exactly why or how, but it gets injured. All right? And then part of that injured process is cells proliferate, okay, in the intima, which is the layer directly below the endothelium. As these cells proliferate, cholesterol accumulates, okay, and lipids accumulate in the area. The cholesterol then gets taken up by cells, all right, and it precipitates in these cells called foam cells, and they crystallize and they cause cellular necrosis, cells break down. And then what happens is the enzymes leak out, cholesterol crystals are present in the intima, and then there's some scarring, calcification, and degenerative changes in the arterial wall. All right? So therefore, this process leads to formation of an atheroma. And the atheroma can have a rough -- excuse me, rough ulcerated surface. This ulcerated surface, all right, it's rough, predisposes to the formation of clots -- or platelets sticking there and then a clot forming. Now, the atheroma can grow out from the artery. If so, very often it doesn't cause a problem. Or it can grow into the artery. If it grows into the artery, then it's going to reduce blood flow, and those are the ones that are the concern. Because it reduces blood flow, and it can
cause an infarction. The other concern is that you have
platelets sticking to this rough area that can set up a
clot, and then the clot breaks off. And when it breaks off,
it's in the cardiac blood -- it's in the circulatory system
for the heart, so it's just going to break off, travel along
the vessel, and end up in a smaller diameter vessel
obstructing blood flow and causing an infarction.
So the atheroma, or atheromatous plaque, it's irregular mass
of yellow, mushy debris. It can encroach on the lumen of
the artery, okay, and it can extend into the muscular wall.
All right. So let's see. A stable plaque basically is one
that's not growing, and it's covered by fibrous tissue.
All right. So here you see early atheromatous plaques
inside the aorta. Very small. This, the lower picture,
is -- has severe atherosclerosis. These lipid plaques
almost everywhere. This is the type of aorta that then is
damaged and ends up dilating. Okay? I told you you can
have a dilatation of the descending portion of the aorta.
All right. So let me just see here.
All right. This, I think, is a great diagram. Okay? And
it's great because it's dramatic.
So what it shows is on the left, say you have a vessel
artery, and the diameter of the artery is four millimeters,
okay? And the flow rate through the artery varies to the
fourth power of the diameter, so the fourth power is the
diameter, which is four, so it's four times four times four
times four. Okay? Which ends up to be 256.
All right. So then so you put a plaque, say this artery
then develops a plaque over a couple years, and the diameter
is no longer four millimeters, it's two millimeters. Okay?
And remember, we're talking about a small area, all right?
Just remember, the -- for, let's see, an inch is made up of
200 -- no. An inch is made up of 25 -- basically 25
millimeters, okay? So when we're talking four millimeters,
it's a small area. A small size, small diameter. So four
millimeters rings all right, cubed, and -- I'm sorry, not
cubed, but to the fourth power, it's 256. If you cut the
diameter in half, so we go from four to two, all right, the
milliliter per minute flow rate is not cut in half from 256
down to, what is it, 128, it's not halved. It goes from 256
to 16. So then, you take that diameter of two millimeters
and you cut it in half to one millimeter, and the flow rate
is one milliliter per minute. So it's a dramatic drop. All
right? And that's why the obstruction, or partial
obstruction, of current and coronary arteries with
atheromatous plaques can have devastating effects. It can
cause dramatic heart attacks and death due to that.
So, this diagram emphasizes the importance, and the
significance of the formation of atheromatous plaques
because they narrow the diameter. All right? So we'll go
back to that picture. So you have some atheromatous plaque surrounded by connective tissue. All right. Question?

>> STUDENT: [Inaudible].

>> PROFESSOR: No. It varies. It varies. Depending on where you're going to be. But they use four because it's easy to halve.

All right. So for coronary heart disease, what are the risk factors? Elevated blood lipids, high blood pressure, cigarette smoking, diabetes. The likelihood of coronary heart disease and heart attack, all right, if you have one risk factor, it's double the risk. Two risk factors, it's four times. Three risk factors, it's seven times the normal risk.

Other risk factors are obesity, and they say the type A personality. All right? Compulsive, hyper, you know, very active type personality, may be another risk factor.

All right. So coronary heart manifestations, all right, also referred to as ischemic heart disease due to decreased blood supply to the heart from narrowing, or obstruction of the coronary arteries. Now, sometimes, blood flow can be blocked in a coronary artery, and the smaller branch. If it's blocked in a smaller branch, sometimes there's not myocardial infarction, because there can be blood supply from surrounding arteries that may get to that area that's deprived, through the capillary system. And we call that collateral blood flow. So people who develop plaques over a long time, a very slow process, you have the advantage of development of collateral circulation. All right?

So, let's see. So with coronary heart disease, people can have it and be symptom-free. They may have angina pectoris, it's a pain in the chest. That's a warning signal. Or they can have bouts of severe chest pain that can radiate in the neck, up into the neck, or into the arms, caused by decreased oxygen to the myocardium. We call that once again, myocardial ischemia.

So stable angina refers to mid sternal pressure, discomfort on exertion. Subsides with nitroglycerin, we talked about that.

Unstable is when it lasts longer and occurs frequently. Very often it's not even relieved by nitroglycerin, and also you can have prolonged myocardial ischemia and it can cause an infarction, and then a cardiac arrest. Here's our diagram. So let's see. Basically, these are all events that can cause ischemic changes. Formation of a clot in a coronary artery, an atheromatous obstruction. You can bleed into an atheroma or it may grow quickly, so then they have hemorrhage into a plaque or spasm, narrowing of the coronary artery.

So with a myocardial infarction, you have necrosis of the heart muscle, all right, and this results from insufficient
blood flow through one of the coronary arteries. Okay? And inadequate collateral blood flow. The term transmural means full thickness. Subendocardial means only part of the wall, okay? Not all of it. Part of it. So myocardial infarction, the location often involves muscles of the left ventricle and septum. Thicker walls require rich blood supply, they work harder to pump blood into the systemic circulation. Rarely does it involve the right ventricle or the atrium. Okay? And very often, the location depends on the obstruction and the collateral blood flow. All right? So what triggers a heart attack? You can have sudden blockage, bleed into an atheromatous plaque, arterial spasm or sudden demand, increase in demand and myocardial oxygen requirements. When I lived in New Jersey, I practiced in New Jersey, and periodically every winter you'd hear about people who would go out, shovel snow, and have a heart attack. These basically were people, older people, they had a very sedentary life. Okay? They'd sit in front of their TV a lot, which is totally fine, but just describing their life. Maybe their biggest activity was going to the food store, okay, once a week, and all of a sudden there's a big snow, and, you know, they want to have their sidewalk shoveled, and, you know, the neighborhood kid is busy shoveling his parents' own walk, so he hasn't done their walk so they decide to do it themselves, put a heavy jacket on, go out, they start shoveling, their heart's not used to that type of activity. Requires increased oxygen requirements, which they can't meet. So they end up with a heart attack. So the cardiac arrest can result from prolonged or severe myocardial ischemia. All right? Can be involved with ventricular fibrillation. So let's see, complication, someone gets a heart attack, the biggest concern is arrhythmias, irregular heart beats. Okay? And what happens is is the concern is that when we have an infarction, you have destroyed tissue, it may set off or may act as a focus or center for abnormal beats. So when someone has an infarction, they give them medication, lidocaine is an example, where it's given intravenously to reduce the irritability of the heart muscles. Okay? Also, there's the concern about forming a thrombus, a clot in the heart that can then break off. And also, cardiac rupture. If it goes -- if it's a full thickness, transmural. All right. So let's see. The bottom picture here shows you cardiac rupture through a transmural infarct. All right. Area of infarcted heart muscle. All right. So factors affecting survival include the size...
of the infarct, the age of the patient, complications, other
diseases. You know, do they have hypertension? That's
going to complicate things.
All right. So they're telling us in the book that 6%
mortality rates for small infarcts, all right, and up to --
greater than 50% for large infarcts with severe heart
failure.
90% of hospitalized patients survive MIs. However, the real
question is what percentage of patients get to the hospital,
okay, with an MI. And how many don't get there. Okay? You
can have a silent MI and not know it, not have it be
diagnosed, and basically sort of recover from it, you know,
so that's a hard statistic to really fully evaluate.
All right. So how do you diagnose it? You can take a
history, okay, may be inconclusive. They may have pain.
All right. Remember, I told you about when I was an intern,
the GW emergency room, that a student who worked in the book
store came in and said I feel like I have an elephant
sitting on my chest. That's how he described. He had
severe angina. That's how he described the pain. Physical
exam, usually not that significant. You use it to -- use
EKG and enzyme tests. Okay? To diagnose the heart attack.
You put things together, with the history, physical exam,
EKG and you wait for the blood test to come back. Troponin
T and troponin I, they start to peak within 24 hours, may
remain elevated for up to 14 days. CK-MB also, creatine
kinase, there's several different types of creatine kinase.
All right. The isoenzyme MB is the one that's specific to
cardiac muscles and that rises within a few hours, peaks in
24 and lasts for a few days.
All right. So what are the things to watch for? Severe
unstable angina, obviously is going to be a warning signal.
So let's see. Now, how do you treat it? All right. One of
the ways to treat it, if they get to the hospital soon
enough, you get them thrombolytic therapy, and remember we
talked in the coagulation chapter about plasmin. Plasmin
will dissolve a clot. Plasminogen tissue, plasminogen
activator. Plasminogen activator is given intravenously,
and what that does is it activates plasmin within the
person, and the plasmin then will break up the clots. It's
called the clot buster, thrombolytic therapy. Also,
Streptokinase is another medication that can be given. Best
results if it's given within an hour of the heart attack.
By resolving the clot, you restore blood flow to the area.
After six hours, there's very little benefit, okay?
Streptokinase, thrombolytic therapy does not really work
that well. It's not that effective. The problem with it is
that it disturbs blood flow, disturbs coagulation process so
all of a sudden these people are more susceptible to
bleeding.
All right. Here we have myocardial revascularization, prior procedures, there's an obstruction here, coronary artery and they attach a vein to bypass the obstruction. Also, there's angioplasty. What angioplasty is is they insert a catheter, goes -- usually it's inserted in the femoral artery, and it goes up the artery, up the aorta into the coronary artery, and then to an area where there's a blockage, okay, you see that yellow area that indicates a plaque. They then advance the catheter and then there's a balloon on the end of the catheter. It's inflated. And what that does, it breaks up the plaque, pushes it into the wall. They then insert a stent over the balloon, and that stent serves to hold that plaque back so it doesn't come back into the artery. So as a result, there's a good blood flow through the area. Okay? I mentioned about cocaine causing arrhythmias and infarcts. Okay? Blood lipids. I need to go over this.

All right. Triglyceride, what that is is basically, it's a glycerol, three carbon molecule with hydroxyl groups on each of them, and then combines with a fatty acid, and that's triglycerol. All right? And the fatty acid chains, if they have no double bonds, they're considered saturated fats. They predispose to forming lipid plaques, atheromatous plaques. Unsaturated, all right, they have double bonds, they can be polyunsaturated with several double bonds. The saturated fats tend to be solids at room temperature. So butter is an example. Animal fats. Okay. Cholesterol also is synthesized in the body. All right. High levels of cholesterol are correlated with atherosclerosis. The two diagnostic entities that we follow are the LDL and HDL. LDL stands for low density lipoprotein, and what that does is it binds cholesterol. Cholesterol by itself is not soluble in blood and it can travel in the bloodstream if it's bound to lipoprotein. Also, HDL, high density lipoprotein. That's called the good cholesterol. All right? So let's see, the HDL can be increased with regular exercise, stopping smoking, less alcohol intake.

All right. So here, in the wall of the vessel, remember I told you the first thing is endothelial injury? When a plaque forms, and then there's high concentration of cholesterol in the area? LDL binds cholesterol, drops it off at the area of injury. HDL picks up the cholesterol, brings it back to the liver. Okay? So that's why the HDL is considered protective because it tends to remove the cholesterol from an injured area. The LDL brings it there. So you want to keep your HDLs, or high density lipoproteins, high and you want your LDLs, or low density lipoproteins to stay low. And there's certain medications called the statins and what they do is they serve to lower LDL, all right, to low levels, and by low levels, it reduces the risk
of plaque formation. Now, metabolic syndrome. Also, the whole thing with obesity, hypertension, high lipid results, impaired glucose tolerance, we'll talk about this more in the chapter with adult onset diabetes, or type II, and they are resistant to insulin.

Aspirin. Aspirin. 30 milligrams of aspirin per day can activate thromboxane A2, which is essential for the stickiness of platelets. And platelets turn over every ten days. They live for ten days, so you take 30 milligrams of aspirin, I believe baby aspirin is 32 milligrams. You take 30 milligrams of aspirin per day. That's all you need. And what it does, you take it for ten days, and your platelets are less sticky. And how that is protective, with less sticky platelets, then they don't tend to stick to an atheromatous plaque, you don't form a clot and run the risk of that breaking off. Okay?

Hypertension. All right. Hypertension causes excessive vasoconstriction of the small arteries and we don't really know why this happens. Most hypertension is what we call essential hypertension. We don't know why it happens. It happens. But remember when I told you narrowed down the lumen of a garden hose, how water goes further? Same thing with the excessive vasoconstriction, increases the pressure. All right? Therefore, increases the diastolic pressure, increases the force of ventricular contraction. Systolic pressure has to go up. As the heart works more, it enlarges. It then enlarges. It can develop heart failure. All right. Also, with higher pressure, you run the risk of injury to the arterioles, they sometimes can rupture. Diabetics can have retinal hemorrhages. The eye is one area that, you know, when you go to the doctor, you can look into the back of your eye if he dilates your vessels. You can see the retinal arteriole, and you can see if someone has diabetes from the changes at the back, and they can rupture. One of the things that diabetics are at risk for is retinal hemorrhages. And the concern is when they rupture, they bleed, and then as the bleeding stops and the healing process occurs, it pulls the retina off the back, and into the vision -- interferes with the vision. Also, the kidneys get affected, okay? They get injured, and as a result, it affects kidney function. Let's see. So you have basically essential hypertension. We don't know why it happens. Most people have essential hypertension. Secondary can be from unknown disease such as chronic kidney disease, maybe secondary to diabetes. Let's see. Also, you can have systolic hypertension, which is a mild to moderate rise in systolic blood pressure, but the diastolic stays low. We don't really have an
understanding of that well. Heart failure, where the heart no longer can pump adequate amounts of blood. All right. They develop chronic heart failure. All right. There's forward failure, where you get reduced blood flow to the tissues. There's backward failure, where you get blood backing up in the veins and in the liver. Okay?

Acute pulmonary edema. What that refers to, basically, is decreased output from the left ventricle, so as a result, decreased output from left ventricle, where's the left ventricle get blood from? The lungs. So as a result you have a backup of fluid in the lungs, and there's going to be pulmonary edema.

Let's see. Aneurysms, dilated or outpouchings of the arterial wall, commonly caused by arteriosclerosis. All right. The wall weakens, okay, and as a result, with atheromatous plaques forming, whenever the wall weakens and it starts to balloon out due to the increased pressure in the arteries.

Here you see a dissecting aneurysm on the right. There's a tear into the endothelium, into the wall, and as a result there's dilatation and there can be accumulation of blood within the separation. Okay.