Wake-Up Call Continued

Lisa A. Rubin and Clyde Freeman Herreid, University at Buffalo, State University of New York

Case copyright held by the **National Center for Case Study Teaching in Science**, University at Buffalo, State University of New York

Case study was modified and questions were added by Larissa Eiselein to fit BIO 131A course content.

Part IV—"Call 911!"

It was March. Emily was home for spring break and Denise was enjoying having her 19-year-old daughter around. Unfortunately, it was going to be hard to spend much time with her because it was that time of the year when weddings and other catered events were picking up again after the post-New Year's lull. Denise was feeling the pressure pile up again. She constantly felt fatigued and out of breath, but she attributed these to perimenopause.

Emily could sense that her mother was tense and out of sorts, so she planned a relaxing evening for her parents and offered to cook mushroom lasagna, her mother's favorite dish. All was going well until dessert, when Emily noticed her mother's face growing paler by the minute. Suddenly, just like that night back in October, Denise began to have severe trouble breathing and her heart began racing. The room began to spin and, without warning, she fainted on the dining room floor.

"Oh my God! Dad, call 911!"

"Uh oh. Oh! Oh no! Denise. Denise! Do you read me? I'm in the middle of a heart attack!! I know it. I can feel it! That plaque in your left anterior descending coronary artery just ruptured. Now everything is going crazy. Everyone in the whole body seems to be swimming by. High levels of fibrinogen, C-reactive protein (CRP), and interleukin-6(IL-6-inflammatory markers present in the bloodstream when there's unstable plaque) are combining with your high blood serum cholesterol. BAD things are happening, Denise. Really, really BAD!

Plaque ruptures. Platelets stick to the exposed lipid core at the site of rupture. The blood clot grows...too big. Oh too big. Is it going to break? Say it isn't going to break. Not thrombosis, please....

.... It's been 10 minutes since my heart cells supplied by the blocked artery have been without oxygen. If something isn't done soon, my cells are going to die. Necrosis! I never thought I could say that word. They say a heart attack can take over four to six hours. This first hour is horrible—the most critical period. Parts of the blood clot may break loose, travel in the blood, and stick in some tiny little blood vessel. My God, it could get in a coronary artery or the brain! An embolism. I need help! Now... NOW. HELP!! I've got to get myself in hand. It's the only way in a crisis. Right? Right! Why didn't Denise go to her doctor to complain about her chronic breathlessness, fatigue, and nausea? All this stress elevated her blood pressure and further increased her risk for a heart attack. Alright, so she didn't know that she had a mutation in her LDL receptor gene. How could she know that LDL was not being efficiently removed from her blood? Whatever. At least she should have known her LDL blood levels were very high. So were her levels of lipoprotein (LP a). This stuff increases heart disease risk. Why didn't anyone warn her?

Sure, I know I'm involved. I'm taking it personally. Wouldn't you? But maybe, just maybe, if Denise had been more aware of the symptoms of heart disease she would have sought help. I happen to know that heart attacks are the number one cause of death in the U.S. More people die from cardiovascular disease (including heart attacks, atherosclerosis, and hypertension) each year than the next six leading causes of death combined, including cancer and automobile accidents. It's an epidemic that people need to be educated about. So get it. I'm here to tell you. Denise. If you won't listen to me, who will you listen to?

Questions:

1. Why is the first hour of a heart attack the most critical?

2. What are platelets and what do they have to do with Denise's heart problem?

3. What is an embolism and what is its connection to thrombosis?

4 . Given what you know about cardiac muscle, would you say it most resembles a) a fast glycolytic muscle fiber or b) a slow-oxidative muscle fiber?

Part V—"Emergency Room"

The doctor spoke calmly to Jeremy in the waiting room. "Mr. Belmore, your wife is in no immediate danger but she has suffered a heart attack to her left ventricle. She's in the emergency room right now, with the aid of an oxygen mask. Her ST-elevation has resolved, but we are continuing to monitor her ECG."

"Yeah. I'm actually the one who has been diagnosed with heart disease in the house, and I'm the one with a family history. I don't understand. Where did this come from? Denise is conscious of her weight, and she's healthier than I am. She's the one who usually looks out for me and my daughter."

"Well, from her records, your wife hasn't had her blood pressure and cholesterol tested in a few years. Unfortunately, they were highly elevated, which greatly increased her risk of heart disease. Although she looked fit on the outside, blood work would have revealed hidden dangers. Tell me, had your wife been feeling out of sorts these past few months?" "She has always been an on-the-go person and tends to worry a lot. Her job is pretty stressful. I did notice that these past few months she seemed more tired than usual and acted almost asthmatic. But, don't heart attack victims experience chest pain? Denise has never complained of that."

"That's a good question. The simple answer is that women's heart disease symptoms can be subtler than men's and are often overlooked. Take a look at the charts on the wall over there and you'll see what I mean. Patients may experience all, some, or none of those symptoms. It is even possible to have a silent heart attack."

Women's Symptoms

Angina (chest pain may radiate into jaw and down left shoulder and arm)

Breathlessness (especially at night)

Chronic fatigue (usually overwhelming)

Dizziness or even blackouts

Edema of feet and ankles

Fluttering (rapid heartbeat) and pallor

Gastric upset (nausea) and sweating

<u>Men's Symptoms</u>

Sudden immense pressure or pain in the chest center (may persist or occur on and off)

Pain that radiates from chest center to neck, shoulders, and arms

Dizziness, nausea, sweating

Sudden onset of rapid heartbeat

The doctor continued, "This is a pamphlet that gives you some background on cardiovascular disease and the factors that go into them. You'll notice that some of these are things you can't change. We call them "non-modifiable." They include your gender, age and your hereditary background; we're all stuck with these. Then there are the "modifiable" factors, things like smoking, stress, and a high fat diet. When more than one factor is present, risk further increases. Once Denise is better I think you both need some time together to consider how you might change your lifestyle."

Questions:

А

- 1. In your own words, explain to Denise what a myocardial infarction is and why she might have experienced one.
- 2. Considering your answers from above, explain why necrosis of heart muscle tissue may occur during a myocardial infarction.
- 3. How would a myocardial infarction affect stroke volume? Draw a pressure volume loop to show what is happening compared to normal.

- 4. How would the change in stroke volume affect cardiac output? Use a formula to relate the two variables.
- 5. How would this change in cardiac output affect MAP? What is MAP? Why might a person with a myocardial infarction feel light-headed and dizzy? Why might the person faint?
- 6. Discuss all factors that can influence MAP.
- 7. What is systolic pressure/diastolic pressure?
- 8. What is sphygmomanometry? Explain how you measure BP.
- 9. Why did Denise experience an increase in heart rate during her heart attack? What are baroreceptors? Where are they found? Draw the complete baroreceptor reflex for this situation.
- 10. Why might a person's extremities feel cold to the touch during a heart attack?
- 11. Why was Denise short of breath? What is happening at the pulmonary capillary level? Consider the normal exchange in pulmonary capillaries and think about what is different during a myocardial infarction. Draw a pulmonary capillary. Indicate the arteriolar end, venular end, osmotic pressure, hydrostatic pressure, filtration and absorption. Discuss which of these change(s) during a heart attack. What is pulmonary edema?
- 12. What are lymph vessels and why are they important?
- 13. Where would edema most likely develop during a myocardial infarction of the RIGHT ventricle?

Part VI—"The Aftermath"

"Well, it's been four hours since the chaos began here in Denise's heart. I'm pooped!

Here's the way I see it. A bunch of my cells are dead. So now there's an inflammatory response of neutrophils and monocytes and an elevated body temperature. Enzyme levels in the bloodstream are up. I don't know one enzyme from the other. They're all just proteins to me. But here's what I heard the doctors say—I mean it, they really use these big words:

Creatine phosphokinase (CPK) has become elevated and will peak within 12 to 24 hours since the attack and with luck it'll return to normal within 48 to 72 hours. Its isoenzyme, CK-MB, is also elevated. CK-MB2 undergoes a change to CK-MB when released into the bloodstream. The ratio of CK-MB2 to CK-MB1 is more than 1.5 for heart attack patients, which is a benchmark doctors use to diagnose myocardial infarction within 6 hours of symptom onset. The blood level of aspartate aminotransferase (AST or GOT) has become elevated due to cell injury, will peak in 24 to 48 hours, and will return to normal in five days. In contrast to the rapid rise and decline of these enzymes, lactate dehydrogenase (LDH) will begin to elevate within a day of the attack onset and will persist at high levels for 10 to 20 days.

Cardiac troponins T and I (which help me contract) will remain elevated in the blood for 10 to 15 days after myocardial injury. This means that if the doctors find that the troponins levels are up, they can really be sure the heart has been injured. Well, that's sure to be what happened to me. So now what have I got to look forward to? Some rest and healing time. With luck, four to six weeks from now, Denise's body will have deposited collagen fibers and scar tissue at the plaque rupture site. Some more collateral vessels will have been built. But for me, things will never be the same. Any of my heart tissue that died from oxygen starvation will be lost and replaced with scar tissue ... unless doctors can find a way to regenerate it. Geesh, I never thought this would happen to me. Denise is so young...."

Questions:

- 1. List some life-style changes Denise should think about to prevent another heart attack.
- 2. How would a daily aspirin help in preventing a myocardial infarction?