# Wake-Up Call

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

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**Part I—"Panic!"**

It was 4:36 a.m. She was in a cold sweat and having difficulty breathing. She felt as though she had run a marathon. Fear swept through her—something terrible was going to happen. Panic-stricken, she woke her husband, Jeremy.

"Denise, what is it? Is it a nightmare?"

"No, it's like I'm having an asthma attack. I feel lightheaded and I can't catch my breath. My heart feels like it's beating a thousand times a minute."

Afraid to upset her husband further, Denise didn't tell him that an immense feeling of apprehension suddenly overcame her. She got up to drink some water. Her mind was racing. Jeremy had a family history of heart disease. This couldn't be happening to her. It was his problem. A few months earlier Jeremy was diagnosed with coronary artery disease. He was only 49 years old, the same age as Denise. The scare had encouraged him to gradually end years of chain smoking and adopt a healthier lifestyle. He was currently working on giving up the occasional cigarette for good.

"No," Denise thought to herself. "There's no way this was a sign of heart troubles. I didn't have a pain in my chest, I'm physically fit, and I have no family history. There's just no way."

**Questions:**

**1. Which cells in the heart are responsible for setting heart rate?**

**2. Which branch of the autonomic nervous system is responsible for increasing heart rate?**

**3. Draw a diagram of the heart showing the location of contractile cells (myocytes) and autorhythmic cells (pacemakers). Show where nerves from the autonomic nervous system would synapse on these structures. Include receptors and neurotransmitters!**

**4. Can the autonomic nervous system modify contractile strength directly?**

**5. How is contractile strength (stroke volume) modified? Define stroke volume. List all factors that can influence stroke volume. What is the Frank Starling Law of the heart? Discuss how optimal length for a cardiac muscle fiber is the same/different from the optimal length for a skeletal muscle fiber.**

**6. True or false: in a normal heart both the right and left side pump the same amount of blood.**

**7. What is a pressure volume loop? Draw a normal pressure volume loop for the left ventricle and discuss all parts.**

**8. How would a pressure volume loop for the right ventricle look like – how is it different from the loop you have drawn above? Why?**

**9. Define: venous return, end-diastolic volume, end-systolic volume, stroke volume, contractility, systole, diastole, cardiac output.**

**Part II—"A Voice from Within"**

The next day at work, Denise was having a hard time focusing. Maybe the stress of her job was finally catching up with her. Managing a catering business was no easy task. On top of that, her only daughter, Emily, had left for college this fall and, being the overprotective parent that she was, Denise found herself constantly worrying about how her daughter was faring in a different city, away from the comforts of home. Also, Denise was starting to go through the early stages of menopause. The hormonal changes, combined with fatigue, stress, and her general worrisome nature, were catching up to her. Not only that, she couldn't get last night's scary episode out of her thoughts. Was it just part of the whole perimenopause thing or was it more? Her body was trying to tell her something, but Denise wasn't sure she was ready to hear.

*"I wonder if Denise realizes how all those years of second-hand smoke have taken a toll on her lungs and on ME, her heart! All that tobacco inhalation has constricted her coronary arteries. Sure, Denise tries to stay physically active but genetics and her food choices have brought her blood cholesterol up pretty high - to 245 mg/dl. She could be headed for heart disease. A person's total cholesterol level shouldn't get above 200 mg/dl. That's right. I ought to know! Denise has hypercholesterolemia, a major contributor to heart disease. Geesh. Get with it, Denise.*

*That was a major warning last night. I'm oxygen-starved! Luckily, only a small area of my left ventricle had a big decrease in blood flow and oxygen supply (cardiac ischemia). Thank goodness. If nothing else happens, my body will start growing some new collateral vessels (bypass channels) and I can get some repair work done. Denise didn't experience chest pain (angina pectoris). But her rapid heart beat and shortness of breath sure got her attention. She had better shape up because I don't know if I can handle much more oxygen deprivation. And, hey, all this unstable plaque lurking around is not a good sign either. No indeed. Who knows when it may rupture? I don't like the looks of this at all."*

**Questions:**

**1. Draw a sketch of the heart and show where the coronary blood vessels lie.**

**2. List in order the blood vessels and heart chambers that a drop of blood would enter as it makes a complete journey around the body starting with the right atrium. Include all heat valves.**

**3. Make a table clearly listing the major differences between an artery, capillary, and vein.**

**Part III—"Heart Attack Basics"**

It appears that Denise has suffered mild heart trauma, which may lead to a more severe **heart attack** if not treated. But wait ... isn't a heart attack when the heart stops beating? Not exactly.

**Cardiac arrest** is the term used when the heart muscle literally stops pumping blood. **A heart attack**, also known as a **myocardial infarction**, may lead to cardiac arrest, but it's defined as a sudden event where at least one of the three major coronary arteries (right coronary artery, left anterior descending coronary artery, and left circumflex artery) becomes partially or totally blocked, usually by a blood clot (**thrombus**). A more rare cause of coronary occlusion is an artery spasm that shuts down blood flow to the heart. This can occur with cocaine use and severe emotional stress. Other rare causes of heart attack include sickle cell crisis, allergic reactions, carbon monoxide poisoning, extreme hypoxia, and an unmet increased need for blood flow to the heart such as may occur during extreme physical exertion, shock, or hemorrhage.

Heart cells can live for about 20 minutes without oxygen. The loss of oxygen-rich blood to the heart cells during a heart attack leads to cell damage, which may be permanent and lead to cell **necrosis** (death), depending on the severity of the attack and the amount of heart tissue that the blocked artery supplies. The area of infarction is where cell necrosis occurs, if it does. Surrounding it is the area of injury, which may or may not suffer permanent damage. The outermost affected area is the zone of **ischemia**, which is weakened but regains function within two to three weeks.

Besides the possibility of cardiac arrest, other possible complications include the following: **cardiogenic shock** (where the heart is too weak to adequately pump blood), **pulmonary edema** (where a weakened heart causes blood backup and leakage of plasma into the lungs), irregular heart rhythm (**arrhythmia**), rupture of a heart wall or valve, or death.

It is a misconception that having a heart attack leads to **chronic coronary artery disease** (CAD). In reality, CAD and accompanying **atherosclerosis** (hardened, narrowed arteries) is the number one cause of heart attacks. What causes CAD? The main culprit is arteriosclerosis, or **plaque** buildup in the coronary arteries. Plaque is a material composed mainly of lipids, cholesterol (lipoproteins), and calcium. **Cholesterol** (a type of lipid necessary for synthesis of hormones, vitamin D, and bile) is carried through the bloodstream by two main types of lipoproteins**: high-density lipoproteins** (HDLs) or "good" cholesterol, and **low-density lipoproteins** (LDLs) or "bad" cholesterol. Studies by the American Heart

Association and the well-known NHLBI-supported Framingham Heart Study show that HDLs help prevent heart disease by transporting lipids and cholesterol from the arteries to the liver. LDLs, which contain more fat and less protein, are unstable and stick to artery walls to help contribute to plaque formation.

LDLs (cholesterol-handling system) produce toxins that form tiny lesions on the inner walls of arteries. These lesions attract triglycerides and other substances in the bloodstream. White blood cells (inflammatory system) rush to the injury site, but cause the inner wall to become stickier and thus attract more LDLs. Platelets (blood-clotting system) collect at the lesion site, only to trap more lipids and white blood cells. Plaque build-up slowly occurs. (Note that cholesterol is not the sole cause of plaque formation.) Over time, some of the plaque can develop a thick, hard, calcified fibrous cap and is called stable plaque, yet causes the arteries to become narrower and harder (atherosclerosis). Other plaque can develop a large lipid and macrophage core, decreased smooth muscle cell content, and a thinner, softer, more unpredictable fibrous cap (due to increased metalloproteinase enzyme activity). This can rupture, producing a thrombosis (artery blockage), cardiac ischemia, and a heart attack can ensue.

**Questions:**

1. **One of the clinical diagnosis tools for heart attacks are ECG recordings. The ST-segment appears elevated in patients experiencing an acute myocardial infarction. Draw a normal ECG tracing, labeling each wave form. Indicate where atrial <u>contraction</u> and ventricular <u>contraction</u> occur. Think about excitation-contraction coupling.**
2. **List the path electrical current takes in the heart, beginning with the SA node.**
3. **Another heart problem, in which the ST-segment of the ECG is elevated, is heart block. Explain what heart block is. In addition to an elevated ST-segment, how would an ECG recording from a patient with heart block look different from normal?**
4. **Compare a normal ECG tracing to one in which the vagus nerve was cut.**
5. **Why is tetanic contraction of the heart impossible? Be specific!**