

Cords, Coffee Tables, and Cadavers: Mini Cases on Cardiac Output

by

Sheri L. Boyce

Department of Biological Sciences

Messiah University, Mechanicsburg, PA

Case 1 – Carl

Carl arrived at the ER suffering from severe shortness of breath (dyspnea) that began approximately 12 hours earlier. He was an active 64-year-old, with no recent illness or shortness of breath before today. He had a prior history of appendicitis but was otherwise healthy, with no known cardiovascular issues or other chronic medical conditions.

A chest X-ray showed some opacity of both lungs, but his heart was not enlarged. Additional tests ruled out a myocardial infarction, but the doctor detected a noticeable murmur when listening to Carl's heart. Carl was surprised to hear this, and reported that he had never been diagnosed with a heart murmur even though he had had regular physicals for the past 30 years. An echocardiogram (an ultrasound of the heart) showed that several of the chordae tendineae connected to the anterior leaflet of the left atrioventricular valve had ruptured.

Use the following data to answer the questions below.

- Heart rate: 102 bpm
- End diastolic volume (EDV): 115 ml (within normal range)
- End systolic volume (ESV): 30 ml (decreased)
- Left ventricle size: normal
- Left atrial size: normal

Questions

1. Explain how the ruptured chordae tendineae are related to the newly detected murmur.
2. Consider the path blood now takes during contraction of Carl's left ventricle and the influence of afterload. Why is his ESV less than normal?
3. Given Carl's ESV and EDV, explain why his calculated stroke volume (SV) and ejection fraction (EF) are elevated.
4. Despite Carl's increased SV and EF, he begins to exhibit signs of cardiogenic shock in which his heart cannot supply enough blood to the tissues and oxygen levels in the tissues fall. Explain why with reference to the ruptured chordae tendineae.
5. Why is Carl's heart rate elevated?
6. How is Carl's shortness of breath related to the ruptured chordae tendineae? (*Hint*: think about where blood will back up, and follow the path "backwards.")

Case 2 – Sofia

Sofia settled awkwardly on the exam table and waited as her favorite nurse Meg checked her blood pressure and pulse. Sofia loved being pregnant, and now that she was seven months along, her large belly and the movements of her baby made motherhood seem closer every day. What she didn't love was having to pee every 45 minutes.

“Pulse 96, BP 110/68. Nothing concerning in your urine specimen either.”

Sofia frowned at Meg. “Isn't 96 a little high? And is my blood pressure still considered normal? It's been around 100/65 for most of my pregnancy, but no one seems concerned.”

Meg smiled. “You don't have anything to worry about. Pregnancy causes some significant changes in your cardiovascular system and there's a wide range of what's considered normal. Dr. Janssen will look at your chart to be sure, but your numbers aren't unusual.”

As she spoke, Dr. Janssen burst through the door and settled her lanky frame on a stool. “Sofia, tell me how you're doing! Third trimester is here! Baby moving every day? Any cramping or contractions? Swelling in your feet and legs? How are you sleeping?”

By now, Sofia was used to Dr. Janssen's rapid-fire conversation; she knew Dr. Janssen would always stop to listen if Sofia had questions. Sofia answered and laid back so the doctor could listen to the fetal heartbeat. As Sofia lifted her legs onto the table, Dr. Janssen pointed at her shin. “That's a spectacular bruise there, Sofia. I think I see every color of the rainbow.”

Sofia grinned. “I know. Hector teased me about secretly joining a rugby team. I did run into the coffee table the other day but not that hard. I don't know why the bruise is so big.”

“Not too surprising,” Dr. Janssen replied. “Your blood volume increases almost 50% during pregnancy, so that there's enough blood to supply the placenta and meet the baby's needs. Some folks think perhaps that's why you bruise more easily, although no one is really sure. Vessels break with the slightest bump, and more blood means more bleeding under the skin ... next thing you know, you've got a bruise the size of Texas.”

She carefully finished the rest of Sofia's exam. “Baby's doing fine and so are you. Rest as much as you can, and we'll see you in two weeks. And tell Hector to move the coffee table.”

Sofia's heart rate and blood pressure at her monthly appointments are shown in the chart below. Refer to it when answering the questions below.

	Pre-pregnancy	Month of pregnancy					
		2	3	4	5	6	7
Heart rate (bpm)	78	82	81	86	90	92	96
Blood pressure (mmHg)	119/78	103/69	100/64	99/62	102/64	105/70	110/68

Questions

- How does Sofia's current resting heart rate compare to her pre-pregnancy heart rate?
 - What effect will this have on her cardiac output? Explain your answer.
- Describe how Sofia's blood pressure has changed over the course of her pregnancy.
 - How did the change in her blood pressure (especially during the first six months of pregnancy) influence stroke volume? Explain your answer.
 - Given your answers to Question 1b and Question 2b, will Sofia's change in blood pressure enhance or counteract the effect of her altered heart rate on cardiac output?

3. What impact does the increase in blood volume during pregnancy have on venous return and cardiac output? Describe the underlying physiology.
4. Given your answers to Question 2 and Question 3, can you assume that Sofia's ejection fraction will likely change as well? Why or why not?
5. Over the last few weeks, Sofia has noticed that lying or sleeping on her back makes her feel a bit short of breath. Hypothesize why this might happen in the later stages of pregnancy. (*Hint:* list all of the factors that influence cardiac output; then consider the position of her enlarged uterus relative to components of the venous system.)

Case 3 – The Cadaver’s Heart

Excited chatter filled the gross anatomy lab as students filed in and prepared to begin the day’s dissection. The syllabus listed it as “Dissection of the Heart and Mediastinum,” but it was better known as “We get to take out the heart!” day.

At Table 5, the students worked carefully to peel back the pericardium and cut the aorta and other great vessels in order to remove the heart from the thoracic cavity. As Raji held the cadaver’s heart, he was surprised at its size. He thought the heart was usually a little larger than the size of your fist, but this heart was huge. The cadaver was a pretty big guy, but even so, his heart was easily bigger than two fists.

Andrea plucked the heart out of Raji’s grasp. “Let me see the...oh, wow, it’s enormous! And look, I think he had bypass surgery...there’s a vessel grafted on to the aorta.”

Raji and Andrea continued to study the heart and it was soon very clear that the donor had suffered from significant cardiovascular disease. The bypass was extensive; a grafted vein ran from the aorta down the anterior left ventricle and then superiorly up the posterior side of the heart. All of the chambers were dilated well beyond their typical size, but the changes were particularly noticeable on the left side.

“The left atrium looks like a flimsy paper bag,” Andrea noted. “And isn’t the left ventricle wall supposed to be a lot thicker than the right? In this heart, they look the same.”

Professor Schultz overheard Andrea’s comment and came over to take a look. “Hmmm, that’s the longest bypass I’ve ever seen; he must have had significant blockage in his coronary arteries. This white area in the anterior wall of the left ventricle looks like scar tissue from a myocardial infarction. But the dilation is what’s really impressive. Terrific example of ischemic cardiomyopathy, which is the most common type of dilated cardiomyopathy.”

Seeing the students’ eyes glaze over, Professor Shultz smiled. “OK, in plain English. He had a coronary bypass to address poor blood supply to his entire left ventricle, probably after the heart attack. However, the heart attack caused a large number of cardiac muscle cells to die, and over time, his left ventricle dilated to compensate. That worked for a while but eventually his heart couldn’t pump effectively to meet systemic needs, which we call heart failure.”

Raji absorbed the professor’s explanation. “So, the donor probably wasn’t very physically active.”

“Probably not,” Dr. Schultz responded. “It’s hard to predict function just by looking at anatomy, but his cardiac function was likely pretty poor, and he might have struggled just to walk across the room. Medications or mechanical assist devices can certainly help, but eventually a transplant is the only option and many folks don’t make it that long.”

Questions

- Consider the impact of the myocardial infarction on left ventricular function. Predict whether the following increased or decreased immediately after his myocardial infarction (before dilation occurred). Explain your answers.
 - Stroke volume (SV)
 - End systolic volume (ESV)
 - Cardiac output (CO)
- Use the Frank-Starling mechanism to explain why slight ventricular dilation could initially compensate for the changes in SV and CO you described in Question 1. Predict what you would observe if you measured EDV.
- Unfortunately, the surviving cardiac muscle cells undergo changes or “remodeling” and the dilation often progresses. Suppose three years after the infarction, the donor’s left ventricular ejection fraction (EF) fell to 25%. Answer the following:
 - Did the increased dilation lead to an increase or decrease in EDV?
 - Given the EF of 25%, did ESV likely increase or decrease?
 - What can you conclude about contractility over the previous three years?
- The donor was likely prescribed one or more medications to lower his systemic blood pressure. Why will a reduction in blood pressure lead to improved cardiac output?

