Case Study: Vasovagal (Neurocardiogenic) Syncope

An integrative case study using syncope to teach the neural control of cardiovascular hemodynamics with an optional part II study on fetal response to maternal changes in hemodynamic parameters.

Juliet Jones (aka “JJ”) was admitted to the hospital at 37 weeks pregnant. The baby seemed to be measuring small for gestational age and the doctors suspected intrauterine growth restriction (IUGR). Upon admission, the following notes were taken by the nurse.

Patient “JJ”:
Age: 25
Term: 37 weeks gestation, 1st pregnancy
Allergies: N/A
Meds: Prenatal vitamin
Height: 5'2
Weight: 135 lbs
BP: 118/79
HR: 70 bpm
History: no history of heart disease, hypertension, or diabetes

A Doppler monitor was strapped to JJ’s abdomen for continuous monitoring of maternal and fetal heart rates. The nurse began preparing to administer the i.v. in preparation for an induction. JJ informed the nurse of her aversion to having blood drawn because of previously feeling faint. The nurse reassured her that it would not be a concern because JJ was lying down. The nurse began to insert the i.v. line and instructed JJ to be calm and breathe normally. The nurse was able to successfully insert the needle into the vein. However, she began to struggle with advancing the catheter over the needle and fully into the vein. The nurse was unaware of the extreme anxiety that JJ was experiencing during the process that the nurse was struggling to finish the i.v. placement. In fact, JJ’s heart began sinus tachycardia (152 bpm). As soon as the i.v. was placed, JJ began feeling the following symptoms: nausea, sweating, pallor, and lightheadedness. JJ remarked of feeling faint. The nurse quickly increased i.v. fluids and held smelling salts under JJ’s nose. JJ recovered quickly from her episode. However, as she looked up she observed the Ob-Gyn on staff in the doorway of her room flanked by two nurses. He said, “JJ, your heart rate just went rapidly bradycardic (34 bpm) and your pressure fell to 65/30”. “Your heart rate and blood pressure are recovering, but your baby’s heart rate is not coming back up.” JJ was rushed back for an emergency C-section and within 10 minutes of her fainting episode had a healthy baby girl.
QUESTIONS PART 1: PHYSIOLOGICAL HOMEOSTATIC RESPONSE

1. The autonomic division of the peripheral nervous system has two subdivisions, the sympathetic and parasympathetic divisions. What neurotransmitter is released by the postganglionic neurons to the effector cells from each subdivision? Give a generalized activity summary for each subdivision.

2. The heart is said to be dually innervated, whereas most arterioles are not, what does that mean with regard to the sympathetic and parasympathetic subdivisions?

3. JJ experienced considerable anxiety at the start of the i.v. placement. Which system was overly active at that time? Describe how that activation affects the heart and resulted in JJ developing sinus tachycardia. What receptors mediated that effect?
4. Sympathetic stimulation, in addition to increasing heart rate, also affects myocyte contractility. As a result, would heart contractility be stronger or weaker? Define stroke volume? Predict how sympathetic stimulation will affect her stroke volume.

5. Give the equation for cardiac output. As a result of JJ’s tachycardia and heart contractility what will happen to her CO? The equation for mean arterial pressure (MAP) is MAP = CO x TPR. What will happen to her MAP simply based on the change in her CO if TPR does not change?

6. Our bodies seek to maintain MAP within a homeostatic range in order to adequately perfuse all of our tissues. Therefore, our bodies exhibit sensory mechanisms to detect alterations in MAP. Name the specific receptors that detect changes in MAP. Where are they located? What do you predict would happen to the firing rate of these receptors as a result of the change in JJ’s MAP?

7. These receptors elicit a reflex response to bring MAP back to normal. These receptors alter the autonomic control of blood vessels often through hormones and vasoactive mediators that affect blood vessel diameter. What is TPR? What needs to happen to TPR in order to bring MAP back to normal? Which autonomic system primarily controls blood vessel diameter?
8. Describe how arterioles respond to sympathetic activation. What receptors mediate this response? Would sympathetic tone need to increase or decrease in order to reduce TPR and maintain MAP? In addition to the sympathetic response, active local arteriolar vasodilation (by a currently unknown mediator) would have what effect on total peripheral resistance?

9. If Tachycardia (high heart rate) is sustained for a greater length of time it can also independently affect the heart itself. Define end-diastolic volume (preload). Predict what a sustained increase in heart rate would do to end-diastolic volume (preload) independent of any other alterations? As a result, what happens to the firing rate of the cardiac mechanoreceptors (stretch receptors)? Describe the Frank-Starling mechanism and how it applies in this scenario. Taken together, what do you predict the response of stroke volume to be now following a lengthy tachycardic period given your answers to the previous parts of this question?

SUMMARY PART 1.

At this point the body has adjusted TPR to maintain MAP. In addition, if HR remains high with low TPR then EDV and consequently SV will decrease (as in number 9) in order to further lower MAP. In a normal situation, this will keep MAP steady until the stressor has been alleviated and the sympathetic tone has fallen back to normal. No further adjustments would be necessary and the individual would experience no symptoms or illness as a result. This represents a typical physiological response to a stressful situation.
QUESTIONS PART 2: PATHOPHYSIOLOGICAL RESPONSE

Note: Recall that JJ had been told that her baby was measuring small and was growth restricted. The doctor’s did not know the health of the baby. JJ was very concerned and scared. To make matters worse, JJ also does not like to get blood drawn due to many previously unsuccessful attempts in her past.

10. Just as quickly as JJ’s heart rate went up a normal antagonistic reflex response began to attempt to compensate. In some individuals, extreme pain and/or anxiety heightens and prolongs the antagonistic response. This response is characterized by profound sympathoinhibition and parasympathetic outflow. Two regions of the CNS are thought to be involved. The first region serves as the master neural control center, especially for the autonomic system in the brain, called the _______________? The second region is known for emotional processing and is known as the _______________?

11. Increased parasympathetic outflow to the heart is mediated by what nerve? What receptor mediates its effects? How does it affect heart rate and contractility? How might this response be exaggerated in some individuals who are sensitive to pain/anxiety?

12. Recall the equation for cardiac output from question 5. Given your answers to question 9 and 11, what is the effect on cardiac output due to JJ’s heightened antagonistic response?

13. In these situations, the vasodilation is sustained and TPR continues to fall. TPR does not reflexively increase as in a normal response. Recall the equation for mean arterial pressure (MAP), MAP = CO x TPR. Given your responses to question 12, predict what will happen to JJ’s MAP as a result of her vulnerability to this emotionally stress driven physiological response.
14. Give the equation for the estimation of MAP based on measured systolic and diastolic pressures. Calculate JJ’s MAP during her syncopal episode.

15. Describe arteriolar autoregulation. The ability of the brain to autoregulate blood flow is between 65-140 mmHg. What happens when MAP falls below the cerebral autoregulatory range? Is JJ’s MAP below the cerebral autoregulatory range?
QUESTIONS PART 3: LABORATORY EXERCISE

Warning: Do not complete on any student with a known cardiovascular condition or history of fainting.

16. Describe in detail how systolic and diastolic measurements are made by a sphygmomanometer. What are the sounds of Karotkoff? Take your lab partner’s blood pressure following instructions provided by your instructor or in your lab manual. Record the pressures below. For each lab member calculate MAP.

Your blood pressure:__________________________________________________

Lab Partner’s blood pressure:__________________________________________

Your MAP:__________________________________________________________

Lab Partner’s MAP:___________________________________________________
17. One response of transient global cerebral hypoperfusion is syncope (fainting). The nurse informed JJ that she was unlikely to faint because she was lying down.

A. What makes you less likely to experience syncope when you are lying down vs. sitting or standing?

B. Take your lab partner’s blood pressure lying down (after 5 minutes), sitting (after 5 minutes), and standing (after 5 minutes).

   Blood pressure lying down: _______________________

   Blood pressure sitting: _________________________

   Blood pressure standing: _______________________

   Were they different?

   Why?

C. What symptoms should have alerted the nurse that a syncopal episode was beginning? What steps can be taken to reduce the likelihood of vasovagal syncope in an individual prone to the condition?
QUESTIONS PART 4: EXTENSION EXERCISE ON CARDIOVASCULAR RESPONSES IN PREGNANCY
(These questions may require the use of outside resources to complete.)

18. A. What effects does pregnancy have on the parameters we have discussed so far: CO, HR, MAP? Therefore, is a pregnant woman more or less likely to experience vasovagal syncope?

B. Why is lying in a supine position for an extended time a concern in a pregnant woman (think anatomically)? How would lying in a supine position (such as for i.v. administration that was sustained due to complications with the insertion) contribute to the likelihood of a pregnant woman experiencing vasovagal syncope despite her increased CO, HR and MAP?

19. What is IUGR? What are possible risks? Why is IUGR a concern?
20. How does maternal blood pressure and HR influence fetal heart rate?

21. Why are fetal heart rate decelerations a concern especially when they are sustained? Why is an IUGR baby more likely to experience a reduced heart rate than a healthy baby?

22. Why is an emergency C-section the best treatment to resolve fetal bradycardia/distress?
The answer key for this case study may be obtained by contacting the author, Jan Foster, at jfoster@ngu.edu.