Baroreceptor Reflex

The baroreceptor reflex stabilizes arterial pressure by activating the central nervous system to alter heart rate and arterial tone in response to changes in arterial pressure in the aorta and in the carotid arteries. This reflex helps to provide uninterrupted flow to vital organs by maintaining the proper perfusion pressure.

The strength of the reflex can be studied by altering baroreceptor perfusion pressure and then noting the subsequent effect on arterial pressure. A decrease in carotid perfusion pressure causes an increase heart rate, and in turn systemic arterial pressure, a relationship that helps to stabilize arterial pressure.

The baroreceptors are spray-type nerve endings. They respond to changes in arterial wall diameter, which is itself a function of blood pressure in the vessel. As pressure increases, the baroreceptors depolarize more easily, leading to an increase in their firing rate. This signal is carried by Hering’s nerve to the glossopharyngeal nerve (carotid baroreceptors) or the afferent vagus nerve (aortic arch baroreceptors) to the medulla in the brain stem. In the nucleus tractus solitarius, increased baroreceptor firing rate inhibits the vasoconstrictor center and excites the vagal parasympathetic system. This leads to dilation of arterioles and veins throughout the body (vasoconstrictor center) and a decrease in heart rate (vagal...
The feedback loop for the arterial baroreceptor reflex links changes in arterial pressure to vasoconstriction and heart rate and contractility.

Parasympathetic center). Together, these factors act to decrease arterial pressure and likewise decrease carotid and aortic pressures, which reduce the baroreceptor stimulus and completes the feedback loop.

Protocol

In this exercise, we’ll replicate the classical protocol while also following the details of the response to carotid pressure changes.

1. Establish initial conditions.
2. Set carotid perfusion pressure up to 80 mmHg and click the carotid perfusion pump switch to on.
3. Advance the solution for 1 minute and record the appropriate neural and hemodynamic data in the table below.
4. Repeat for carotid perfusion pressures of 60 and 40 mmHg.

Questions

1. How do the effects of clamping affect autonomic firing rate versus vagal firing rate?
2. What effect does decreasing carotid pressure to 20 mmHg have?

One thing to note in the above protocol is that the baroreceptor reflex is saturated at around 50 mmHg. Like all reflexes, it has an area of greatest sensitivity, in which it can exert a profound influence on blood pressure and heart rate. Outside of this range, other mechanisms must assume control of pressure changes.

Protocol

June 24, 2015
1. Restart the simulation and run 1 minute to establish a baseline.
2. Change posture control to standing.
3. Run for 5 minutes, observing carotid perfusion pressure, baroreceptor firing rate, autonomic rate, and vagal rate.
4. Change posture control to lying, turn on the carotid sinus clamp, and set it to the “standing” perfusion pressure (use the magnifying glass to find the exact value). Run 1 minute.

Questions
1. What changes occurred after lying down and keeping the carotid sinus pressure clamped to its “standing” pressure?
2. Observe mean arterial pressure. Why did it increase above resting pressure when posture changed?
3. What explanation can be offered for this difference?

The above protocol introduces the idea that other sources might be responsible for changes in sympathetic nerve activity. The following protocol will hopefully clarify what these other sources might be. Lower body negative pressure causes blood to pool in the lower extremities, thereby reducing venous return.

Protocol
1. Restart the simulation and run 1 minute to establish a baseline.
2. Turn compression stockings to -50 mmHg.
3. Run for five minutes, observing sympathetic nerve activity and mean arterial pressure.

Questions
1. Are the changes seen due to baroreceptor firing? Check by observing baroreceptor rate.
2. What other nervous receptor might be responsible for the changes seen?
3. Has cardiac output (and therefore venous return) fallen? What does this suggest about right atrial pressure?

In the above protocol, lower body negative pressure reduces the height of the venous return function, causing a lower intersection with the Starling curve. The new intersection point has both lower right atrial pressure and lower cardiac output. This suggests that the atrial low pressure receptors, also called cardiac baroreceptors, are responsible for the changes in sympathetic activity in the clamped carotid situation and in the initiation of lower body negative pressure.

Summary
The baroreceptors are a potent detector of mean arterial pressure. As pressure decreases, decreased baroreceptor firing rates trigger decreases in vagal firing rates, and increases in cardiac sympathetic nerve activity. These factors act in the short term to counter the decrease in pressure. They are by no means responsible for all changes in sympathetic and parasympathetic nerve activity to the heart.