Baroreceptor Reflex: Anatomy & Physiology

Physiology > Cardiovascular > Cardiovascular

Baroreceptor reflex Overview Mean arterial pressure (MAP) is the driving force for blood flow; it is regulated by two main mechanisms: The Baroreceptor Reflex, which is fast-acting (within seconds) and acts via changes in cardiac output and total peripheral resistance. The Renin-aldosterone-angiotensin system, which acts slowly and via blood volume changes. **Key Measurements** Mean arterial pressure = Cardiac Output x Total Peripheral Resistance Cardiac output = Stroke volume x Heart rate Total peripheral resistance Primarily determined by the degree of arteriolar constriction, much like a faucet on a hose, and (to a much lesser extent) blood viscosity.

Key Anatomical Structures

The heart, aorta and aortic arch, and the common carotid artery: include its bifurcation into the internal carotid and external carotid arteries. The carotid sinus lies at the origin of the internal carotid artery; the aortic arch baroreceptors lie in the aortic arch. The carotid sinus and aortic arch baroreceptors are baroreceptors, which respond to stretch changes in the arterial vasculature due to changes in blood pressure. Brainstem & spinal cord; we show an expanded axial section of the medulla.

AUTONOMIC INNERVATION

We'll indicate parasympathetic elements in green and sympathetic elements in blue. **Parasympathetic Innervation** Solitary tract nucleus, within the medial dorsal medulla. Cardioinhibitory center lies (nucleus ambiguus) in front of it the solitary tract nucleus. Ventrolateral medulla with vasomotor area; it comprises areas A1 (the cardiac accelerator center) and C1 (the vasoconstrictor center).

CNs 9 and 10

The glossopharyngeal nerve (CN 9) projects from the carotid sinus to the solitary tract nucleus. The vagus nerve (CN 10) projects from the aortic arch baroreceptors to the solitary tract nucleus. The solitary tract nucleus excites the cardioinhibitory center, which produces parasympathetic branches that project via the vagus nerve to the heart to produce: *Heart rate deceleration and reduced stroke volume.*

 SYMPATHETIC INNERVATION

We show the sympathetic ganglia (paravertebral and prevertebral) and a portion of the spinal cord (the intermediolateral cell column from T1 to L2). The vasomotor center provides tonic sympathetic stimulation to the spinal cord (the intermediolateral cell column from T1 to L2). Presynaptic sympathetic branches innervate the sympathetic ganglia. Post-synaptic sympathetic nerve fibers innervate the heart to produce: *Heart rate acceleration and increased contractility.*

MECHANISMS

Peripheral Vascular Resistance

Sympathetic innervation of the vessel produces vasoconstriction (which increases total peripheral resistance). To better understand this, draw a section of the aorta, which is like a large hose. Show a series of organs (like buckets). Show a few arteries emerge from it in parallel, which each have equal pressure to one another. Now add variable arteriolar resistance to each artery (like a faucet); show greater degrees of resistance from top to bottom. Then, show the resultant flow, which is variable. Indicate that as a result the organs, receive variable amounts of blood. Thus, one mechanism to adjust blood flow is via arteriolar constriction (peripheral resistance).

RENIN-ANGIOTENSION-ALDOSTERONE SYSTEM

Sympathetic innervation triggers the renin-angiotension-aldosterone system and adjusts blood pressure via a slow

mechanism; it works through change in blood volume, and involves low-pressure baroreceptors and atrial stretch receptors.

CNS MODULATION

The brain modulates the solitary tract nucleus: after all, we can affect our heart rate through our own conscious fear and anxiety. Key modulators include the hypothalamus and amygdala, and parabrachial pontine nucleus.

ACUTE HYPOTENSION

Drop in Blood Pressure, such as from hemorrhage (blood loss) or orthostasis, triggers the baroreceptor reflex to increase blood pressure via the following steps:

• A drop in mean arterial pressure produces a drop in baroreceptor stretch.

• This reduces baroreceptor activity and thus reduces cranial nerve 9 and 10 firing.

• This triggers an increase in sympathetic activity and a decrease in parasympathetic activity, which produces

an increase in cardiac contractility and stroke volume, heart rate, venous constriction which increases venous return, and arteriolar constriction. These processes are the keys to increases in cardiac output and total peripheral resistance. And, as shown at the beginning, indicate that when they increase, so does MAP – in this case back to normal (the set-point).

CLINICAL CORRELATIONS

Key Clinical Correlations:

• Global Cerebral Ischemia (Presyncope, Syncope)

• Arterial Dissection

CARDIOVASCULAR RESPONSE & ORTHOSTASIS

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Cardiovascular response

A simple way to test the cardiovascular response is by varying your pulse.

• Take your pulse and get a good sense of your heart rate.

• Then, take a deep breath and hold it for 5 or 6 seconds.

• Your heart rate should speed up because when you inhale deeply, you open up lung tissue and shunt blood into the lung capillaries, which reduces your effective circulating blood volume (ie, your stroke volume).

• Cardiac output is stroke volume multiplied by heart rate; therefore, to compensate for a decreased stroke volume, your heart rate increases (typically by 8 beats per minute).

Orthostasis

• An additional, slower response to a reduced stroke volume is to increase the effective circulating blood volume, itself.

• For instance, when we stand, blood pools in our veins, so after we stand upright for a full minute, T5 sympathetic splanchnic fibers command our abdominal vessels to shunt roughly 1.5 units of blood from our abdomen into our peripheral vasculature.

• Because there is a delay in the shunting of blood between systems, when we check orthostatic blood pressure, we must wait at least a few minutes in between measuring supine and standing blood pressure (and possibly longer, even).