Veterinary Bioscience: Cardiovascular System

WEEK 3 – REGULATING FLOW AND PERTURBATIONS OF FLOW

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INTENDED LEARNING OUTCOMES

It is hoped that this lecture will facilitate your understanding of the mechanisms involved in the short term regulation of blood pressure and cardiac output, and long term regulation of arterial pressure. In particular, at the end of this lecture you should be able to:

- identify the sensory receptors, afferent pathways, central integrating centres, efferent pathways, and effector organs that participate in the arterial baroreceptor reflex
- state the location of the arterial baroreceptors and describe their operation
- describe how the sympathetic and parasympathetic outputs from the medullary cardiovascular centre change in response to changes in arterial pressure.
- describe the chain of events that are initiated by the arterial baroreceptor reflex, to compensate for a change in arterial pressure.
- Describe baroreceptor adaptation
- Describe the influence of changes in body fluid volume on arterial pressure
- Explain in general terms how mean arterial pressure is regulated by the kidneys, through regulation of blood volume, and total body fluid content.

LECTURE 13 – MODULATING FLOW AND THE BAROREFLEX

Systemic arterial pressure is the single most important requirement for proper operation of the cardiovascular system. It is the pressure gradient that ensures adequate perfusion of tissues. Elaborate control mechanisms have evolved for regulating blood pressure.

THE DETERMINANTS OF MEAN ARTERIAL PRESSURE

Through the basic flow equation, cardiac output, resistance and blood pressure are linked. Cardiac output (flow) is directly proportional to pressure gradient, and inversely proportional to resistance. Conversely, mean arterial pressure is directly proportional to cardiac output and to arteriolar resistance. MAP= CO X TPR

Hence, an increase in HR, stroke volume (contractility & venous return) or arteriolar resistance will affect mean arterial pressure.

THE BAROREFLEX

The Baroreceptor Reflex is a continuously operating control system, that regulates MAP in a **negative feedback manner** It comprises all the usual components of a reflex pathway, namely a stimulus, sensory receptors, afferent pathways, an integrating centre in the CNS, efferent pathways and effector organs.

The stimulus is a change in arterial pressure or in blood volume, that is detected by mechanoreceptors. These mechanoreceptors are of two types.

the baroreceptors located in walls of the aorta (aortic arch) and the internal carotid arteries (carotid sinus). An increase in blood pressure stretches the vessel wall, causing an increased rate of discharge of the baroreceptors. If arterial pressure remains elevated over a period of several days, firing rate gradually returns to normal. That is, baroreceptors adapt to long term changes in blood pressure, so that the baroreflex is therefore not a mechanism for long term control of blood pressure the low pressure (cardiopulmonary) baroreceptors, located in the atria, ventricles and pulmonary vessels. The cardiopulmonary baroreceptors are stretch receptors that sense central blood volume. Decreased stretch of the cardiopulmonary receptors following a reduction in central blood volume activates the baroreflex to increase sympathetic stimulation to the heart and blood vessels.

Afferent pathways from the carotid sinus travel to the medulla in the ninth cranial nerve, and from the aortic arch and the cardiopulmonary receptors in the vagus nerve.

Central integration of the baroreflex occurs in the **medullary cardiovascular centre** located in the medulla oblongata of the brain stem. There are two more or less distinct regions in the vasomotor centre: the pressor region that provides normal tonic stimulation to blood vessels, via sympathetic nerve fibres, and the depressor region that has inhibitory action on sympathetic nerves and excitatory action on parasympathetic nerves. Descending tracts from the hypothalamus and from the cerebral cortex also modify basic vasomotor reflexes. (e.g. pain, fear)

The efferent pathways for both heart and blood vessels are **sympathetic and parasympathetic nerves**.

Increased sympathetic drive has effects both on blood vessels (vasoconstriction) and on heart rate (increased rate). The **vasoconstrictor** influences are on both veins (capacitance vessels) and arteries (resistance vessels). Capacitance vessels are more responsive to sympathetic stimulation than resistance vessels. Increased sympathetic drive to veins increases venous return to the heart, and thereby stroke volume. (Very few blood vessels receive parasympathetic innervation.)

The **heart** receives both sympathetic (excitatory) and parasympathetic (inhibitory) innervation from the vasomotor centre of the medulla, that effect both rate and force of contraction. The increase in contractility increases stroke volume, that together with the increase in heart rate increase cardiac output.

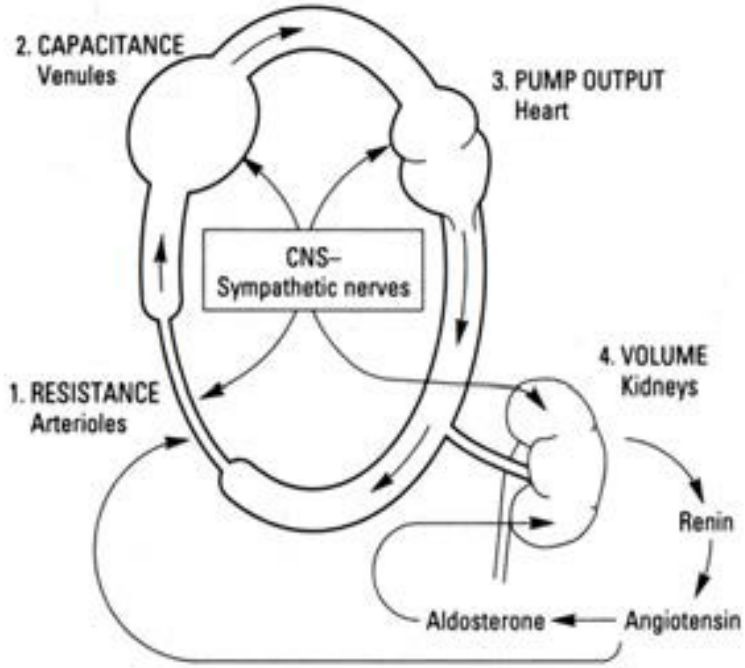

Figure 11–1. Anatomic sites of blood pressure control.

Katzung "Basic & Clinical Pharmacology"

An important feature of the baroreflex is that it adapts over time. When MAP increases suddenly, baroreceptor firing increases, but if the increase in pressure is maintained, firing rate declines due to receptor adaptation.

Long term regulation of arterial pressure is achieved by regulation of circulating blood volume: The renin-angiotensin-aldosterone system. A fall in blood pressure leads to sympathetic stimulation of the juxtaglomerular apparatus and increased renin release. In addition, reduced blood pressure leads to reduced renal perfusion, that also directly stimulates rennin release. Renin catalyses the conversion of angiotensinogen to angiotensin- resulting in vasoconstriction, increased aldosterone release, and increased activity of antidiuretic hormone. The resultant increase in blood volume and arteriolar resistance restore blood pressure.

OTHER CARDIOVASCULAR REFLEXES

Chemoreceptor reflexes: Chemoreceptors located in **carotid bodies** and **aortic bodies** are activated by hypercapnia, hypoxia, and acidosis. The resultant reflex is mainly involved in stimulating breathing, but has cardiovascular effects as well. The reflex results in sympathetically mediated arteriolar and venous constriction, and tachycardia that helps to maintain blood flow to the brain at blood pressures too low to activate baroreceptors.

The Dive reflex Aquatic animals respond to diving with a remarkable bradycardia and intense vasoconstriction in all organs except brain and heart. This allows prolonged submersion by limiting oxygen use and directing blood flow to essential organs as a result of unusual combination of parasympathetic stimulation of heart and sympathetic stimulation of blood vessels. This reflex involved receptors in face and upper airway and stretch receptors in lungs.

Extrinsic reflexes Stimuli external to the cardiovascular system also cause changes in cardiovascular function through activity on the vasomotor centre. e.g. moderate pain causes tachycardia and increased MAP, severe pain may cause bradycardia and decreased MAP, fear etc.

FURTHER READING

Klein BG, Cunningham's Textbook of Veterinary Physiology, 6th edition 2020.
Berne RM & Levy MN, Cardiovascular Physiology, 6th Edition, 2008
Mohrman DE & Heller, Cardiovascular Physiology. McGraw Hill 9th Edition, 2018. eBook available in unimelb library.
Sherwood, L, Human Physiology from Cells to Systems, 8th Edition 2013.
Rhoades RA & Bell DR Medical Physiology: Principles for Clinical Medicine 5th Edition 2017