Veterinary Bioscience: Cardiovascular System

WEEK 4 – DISORDERS OF COAGULATION AND FLOW

LECTURER: DR JOANNA AITKEN

After graduating from The University of Sydney in 2013, Joanna started her veterinary journey in mixed practice in rural NSW. She has since worked in both private practice and university settings across Australia and the United Kingdom. In 2017 she completed a Small Animal Rotating Junior Clinical Training Scholarship (internship) at the Royal (Dick) School of Veterinary Studies, Edinburgh, which led her to a private practice residency in Small Animal Cardiology. Her interest and training in cardiology, emergency and critical care and teaching led to a position as a Teaching Fellow at the University of Surrey, followed by recognition as a Fellow of the Higher Education Academy. During this time, she was also a permanent member of the Vets Now Emergency team in Southern England. In 2020 Joanna moved back to Australian soil and started her residency under the American College of Veterinary Emergency and Critical Care at the University of Melbourne (U-Vet), transitioning into a Lecturer role in the pre-clinical department in 2021. Her research interest is cardiac ultrasound use in the intensive care environment and cognitive task analysis for this technique for veterinary students. Outside of the University she is a co-founder of 'Vets: Stay, Go, Diversify (Down Under)' an organisation that celebrates the multiple career options available to those that hold a veterinary degree.

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INTENDED LEARNING OUTCOMES

At the end of this lecture you should be able to:

- Define and utilise the terms hypertension and hypotension in relation to domestic animals
- Understand the causes and potential consequences of systemic hypertension and explain why systemic hypertension tends to be a self-perpetuating phenomenon
- Revise the terms cardiac output, stroke volume, contractility, afterload and preload
- Define the term 'shock' and relate this to the 'tree of life'
- Describe the six main categories of shock and understand their aetiopathogenesis (hypovolaemic, cardiogenic, distributive, obstructive, metabolic and hypoxaemic)

KEYWORDS

Hypertension, vasoconstriction, vasodilation, peripheral vascular resistance, systemic hypertension, epistaxis, cerebrovascular accident, hypotension, hypoperfusion, cardiac output, stroke volume, contractility, afterload, preload, shock, hypovolaemic shock, cardiogenic shock, haemorrhagic shock, metabolic shock, distributive shock, obstructive shock.

LECTURE 21: SHOCK, HYPERTENSION AND HYPOTENSION

Hypertension refers to a sustained increase in blood pressure. The pressure is a product of the volume of blood and the resistance to its flow, with the latter being influenced by the diameter of the blood vessel. Counterregulatory mechanisms exist to counteract sustained elevations in blood pressure.

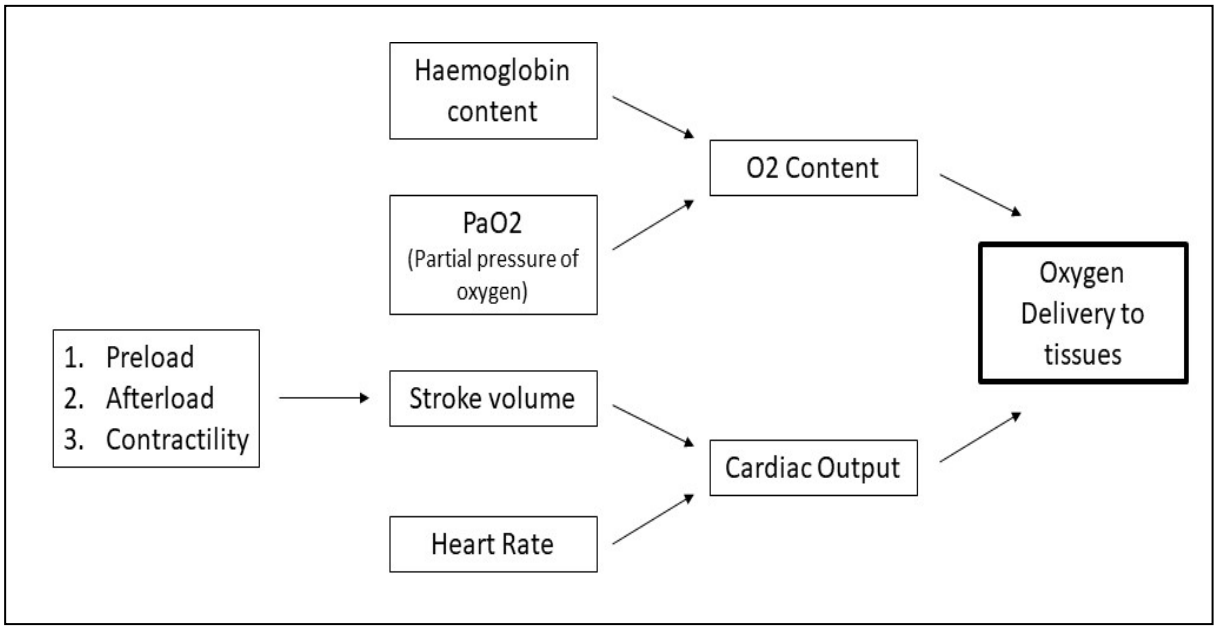
Systemic hypertension is a sustained increase in the systemic arterial blood pressure. The systemic arterial blood pressure is the product of cardiac output from the left ventricle and the total peripheral vascular resistance. Total peripheral vascular resistance is chiefly determined at the level of the arterioles in which vascular tone depends on the competition between various vasoconstrictor and vasodilator stimuli.

Systemic hypertension is highly prevalent amongst humans and is also well documented in cats and dogs. It can be broadly categorised as idiopathic (or primary) hypertension or secondary hypertension. Diseases associated with development of secondary systemic hypertension in small animals include renal disease (especially glomerular disease), feline hyperthyroidism, canine hyperadrenocorticism, functional catecholamine-secreting tumours of the adrenal medulla, and obesity. Although systemic hypertension may remain clinically silent until late in the disease course, its detrimental effects are progressive and self-perpetuating. Chronic systemic hypertension promotes hypertrophy of arteriolar smooth muscle and hence reduction in the arteriolar luminal diameter, thereby exacerbating the hypertension. It also constitutes a pressure overload on the left ventricle during systole, promoting concentric hypertrophy of the left ventricular myocardium over time.

Hypertensive arteriolar spasm may lead to damage to the downstream capillary beds. Animals with systemic hypertension are at increased risk of intra-ocular haemorrhage, progressive glomerular injury and fibrosis, haemorrhagic cerebrovascular accidents (strokes) and hypertensive encephalopathies.

Hypotension refers to a drop in systolic arterial blood pressure. Causes include decreased blood return to the heart (leading to decreased preload), decreased vascular tone (vasodilation) and cardiac dysfunction.

Shock is a generalised phenomenon of peripheral circulatory failure and is characterised by a decrease in the delivery of oxygen to tissues (DO_2) compared to oxygen consumption within the tissues (VO). It is the final common pathway of many disorders and may prove fatal by inducing lethal hypoxic injury to vital organs. The 'tree of life' helps us to understand the components that lead to oxygen delivery to tissues and consequently better understand where it might go wrong.



We will introduce the six main functional classifications of shock: cardiogenic, hypovolaemic, haemorrhagic, obstructive, distributive and metabolic. The causal triggers and pathogenetic mechanisms responsible for each of these forms will be discussed with case examples.

FURTHER READING

Ettinger et al, (2017). *Textbook of Veterinary Internal Medicine,* 8th Edition, Chapters 157 (Pathophysiology and Clinical Manifestations of Systemic Hypertension) and 159 (Systemic Hypotension) and Chapter 127 (Shock).

Silverstein and Hopper (2015). *Small Animal Emergency and Critical Care,* 2nd Edition, Chapter 5 (Shock)