

Veterinary Bioscience: Cardiovascular System



WEEK 4 – DISORDERS OF COAGULATION AND FLOW

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INTENDED LEARNING OUTCOMES

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

- Define and utilise appropriately the terms embolus, embolism, thromboembolism, ischaemia and infarction
- Understand the circumstances that may lead to pulmonary versus systemic thromboembolism
- Predict the factors that can influence the outcome of reduced blood flow to or from tissues and hence the prognosis for the affected animal

KEYWORDS

embolus, emboli, embolism, thromboembolism, saddle thromboembolus, pulmonary thromboembolism, systemic thromboembolism, septic embolus, bland embolus, lipid embolism, gas embolism, foreign body embolism, parasitic embolism, fibrocartilaginous embolism, hypoxia, anoxia, hypoxaemia, ischaemia, coagulative necrosis, liquefactive necrosis, malacia, infarction, infarct, collateral circulation, arterial infarct, venous infarct

LECTURE 20 – PERTURBATIONS OF FLOW 3 – EMBOLISM, ISCHAEMIA AND INFARCTION

An embolus is a solid, liquid or gaseous mass that is transported in the bloodstream to a site distant from its point of origin. Embolism is the process by which a downstream blood vessel is blocked by an embolus that arose upstream. Most emboli are thromboemboli. The consequences of pulmonary versus systemic thromboembolism will be reviewed before we encounter several other types of emboli: lipid droplets, air bubbles, foreign bodies, neoplastic cells, other tissue cells, placental components, parasites, bacteria, fungi, and fibrocartilage fragments derived from intervertebral discs.

Thrombi, thromboemboli and other types of emboli are capable of causing diminished blood flow and hence ischaemic injury to tissues, resulting in **infarction**. The factors that influence the outcome of diminished blood flow and hence the prognosis for the patient include the anatomic location of the infarct, whether the affected blood vessel is arterial or venous, the availability of an alternative arterial supply or route of venous drainage, the rate of development and degree of impairment of blood flow, the size of the affected blood vessel, the duration of tissue hypoxia, the tissue vulnerability to hypoxia, the general cardiovascular and haematological health of the animal, whether or not reperfusion injury occurs, and whether or not there is secondary bacterial invasion of the hypoxic tissue. In general, cerebral, myocardial, intestinal and pulmonary infarcts are the most likely to prove fatal.

Infarcts are typically wedge-shaped, with the occluded vessel located at the apex of the wedge. Infarcts arising from occlusion of arterial branches are usually grossly paler than the surrounding viable tissue, whereas those arising from venous occlusion tend to be dark red-purple and haemorrhagic. However, the anatomic structure and vascular pattern of the affected tissue, the duration of the infarct, and whether the infarct is septic or bland (sterile) also influence the colour and gross appearance of an infarct. If the affected animal survives, the necrotic debris within an infarct is gradually liquefied and phagocytosed by leukocytes infiltrating from the vasculature of adjacent viable tissue. In infarcted stable or labile tissues, parenchymal regeneration may commence at the periphery of the infarct during the subacute phase. Ultimately, most infarcts are replaced by scar tissue, leaving residual pale contracted areas.

FURTHER READING

DA Mosier. Vascular disorders and thrombosis. In: *Pathologic Basis of Veterinary Disease*. 4th ed. Ed. MD McGavin and JF Zachary. Mosby Elsevier, St Louis, USA (2007). pp. 63-99 (**emphasis on pp. 86-91 and 93-95**)

DO Slauson. Disturbances of blood flow and circulation. In: *Mechanisms of Disease. A Textbook of Comparative General Pathology*. DO Slauson and BJ Cooper. 3rd edition. Mosby, Inc. St Louis, USA (2002). pp. 76-139 (**emphasis on pp. 89, 99-102, 104-107, 110-116 and 119-124**)