Veterinary Bioscience: Cardiovascular System

WEEK 3 – REGULATING FLOW AND PERTURBATIONS OF FLOW

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LECTURE 14 – PERTURBATIONS OF FLOW 1 – HYPERAEMIA, CONGESTION AND OEDEMA

INTENDED LEARNING OUTCOMES

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

1. Distinguish the mechanisms responsible for hyperaemia, congestion and oedema
2. Interpret the consequence of hyperaemia, congestion and oedema
3. Categorise the likely causes of hyperaemia, congestion and oedema

KEYWORDS

hyperaemia, active hyperaemia, passive hyperaemia, passive congestion, cyanosis, oedema, hydrostatic pressure, colloid osmotic pressure, albumin, lymph, lymphatics, oedema, hypoalbuminaemia, lymphoedema, vascular permeability, inflammation, hypernatraemia, transudate, modified transudate, exudate, ascites, hydrothorax, hydropericardium, hydrocoele, anasarca, pitting, dependent oedema, brisket disease, bottle jaw

Hyperaemia is an increase in the volume of blood within blood vessels. It can be an active or passive process, and a localised or generalised phenomenon.

Active hyperaemia of tissues involves arteriolar dilation and hence increased perfusion of the downstream capillary beds and is a localised phenomenon. Active hyperaemia is a common physiological process and is also responsible for the redness (rubor) and heat (calor) of actively inflamed tissues.

Passive hyperaemia of tissues is more commonly referred to as **passive congestion** or simply **congestion**. It is a passive process in which impairment of venous outflow causes pooling of blood within upstream venous channels and capillary beds. Congestion may be a localised or generalised phenomenon and can result in oedema and hypoxia of the affected tissues.

Extracellular edema is the accumulation of excess extracellular fluid in the interstitial tissues and/or cavities of the body. All forms of extracellular oedema can be explained mechanistically by an alteration in Starling's equilibrium forces (plasma and interstitial hydrostatic and colloid osmotic pressures that control the movement of water, electrolytes and small solutes across vessel walls within the microcirculation), by an increase in the permeability of vessel walls or by a decrease in lymphatic drainage.

The major causes of extracellular oedema in domestic animals are severe hypoalbuminaemia (e.g. severe malnutrition, protein-losing enteropathies, protein-losing nephropathies, chronic hepatic disease), increased plasma hydrostatic pressure (particularly venous hypertension), increased vascular permeability (e.g. active inflammation, vascular injury) and lymphatic obstruction. Depending on the underlying cause, oedema may be a localised or generalised phenomenon. In some forms of oedema, activation of the renin-angiotensin-aldosterone system exacerbates the oedema by promoting renal retention of sodium and water, thereby increasing the plasma hydrostatic pressure and decreasing the plasma albumin concentration by dilution.

Wound healing is typically impaired within oedematous tissues and the latter are susceptible to secondary bacterial infection. Chronic interstitial oedema promotes fibroplasia and ultimately fibrosis. Severe diffuse oedema of the brain or lungs can be rapidly fatal.

The characteristic gross features and microscopic appearance of tissues affected by active hyperaemia, passive congestion and extracellular oedema will be presented, using common veterinary examples. We will also explore how the anatomic distribution and the protein concentration and total nucleated cell count of extracellular oedema fluid provide important diagnostic clues to the underlying causal mechanism.

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. 67-73 and 92-93)**

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. 78- 83 and 124-136)**