WEEK 1 INTENDED LEARNING OUTCOMES: (INFLAMMATION)

- Define the term inflammation
- Describe the pathophysiology of the inflammation and identify where it may occur in the body
- Discuss the role of the nurse in patient management related to inflammation
- Identify potential and actual patient problems related to inflammation
- Implement nursing interventions for patients experiencing inflammation
- Evaluate care related to patient experiencing inflammation
- Demonstrate therapeutic use of medicines prescribed to treat inflammation
- Describe, perform and document care for a patient receiving a blood transfusion
- Describe, perform and document care for a patient requiring drain tube removal
- Perform a nursing handover of a patient's management using the ISBAR tool

INFLAMMATION: is an adaptive response to injury that brings fluid, dissolved substances and blood cells into the interstitial tissues where the invasion or damage has occurred

Inflammations

- Appendicitis – inflammation of the appendix
- Bursitis – inflammation of a small sac of fluid (bursa) near a joint
- Cellulitis – inflammation of soft tissue under the skin
- Dermatitis – inflammation of the skin
- Endocarditis – inflammation of the inner lining of the heart
- Epiglottitis – inflammation of the epiglottis
- Gastritis – inflammation of the stomach
- Hepatitis – inflammation of the liver
- Myositis - inflammation of muscle
- Nephritis - inflammation of the kidney
- Osteomyelitis - inflammation of bone and bone marrow
- Otitis - inflammation of the ear
- Pharyngitis - inflammation of the throat
- Thrombophlebitis - inflammation of a vein

5 cardinal signs of inflammation are: heat, redness, swelling, pain + loss of function

Tissue injury occurs
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Inflammatory Response (heat, redness, pain, swelling, loss of function)
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Pathogen enters the body
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Mast cells release histamine
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Increased blood flow to the injured area – Vasodilation
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WBC, neutrophils and T-Cells, enter the interstitial space, phagocytes engulf bacteria
↓
Oedema
↓
Platelets move out of the capillary to seal the wound
↓
Healing process begins

Pathophysiology of Inflammation

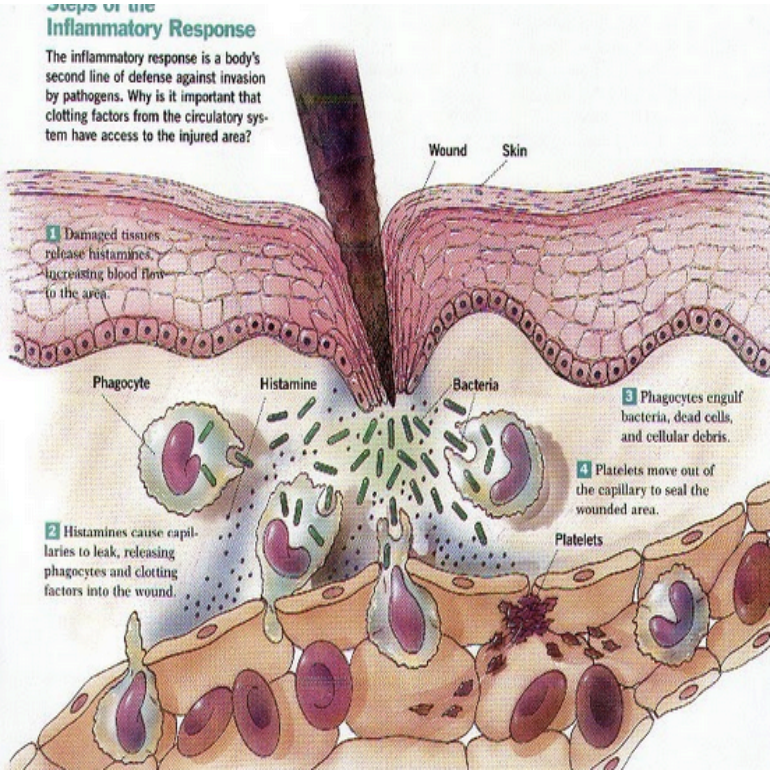
1. A break in the skin introduces bacteria, which reproduce at the wound site. Activated resident macrophages engulf the pathogens and secrete cytokines and chemotaxins.

2. Activated mast cells release histamine.

3. Histamine and cytokines dilate local blood vessels and widen the capillary pores. The cytokines also make the blood vessel wall sticky, causing neutrophils and monocytes to attach.

4. Chemotaxins attract neutrophils and monocytes, which squeeze out between cells of the blood vessel wall, a process called diapedesis, and migrate to the infection site.

5. Monocytes enlarge into macrophages. Newly arriving macrophages and neutrophils engulf the pathogens and destroy them.



THE SERIES OF EVENTS IN THE PROCESS OF INFLAMMATION ARE:

VASODILATION: LEADS TO GREATER BLOOD FLOW TO THE AREA OF INFLAMMATION, RESULTING IN REDNESS AND HEAT.

VASCULAR PERMEABILITY: ENDOTHELIAL CELLS BECOME "LEAKY" FROM EITHER DIRECT ENDOTHELIAL CELL INJURY OR VIA CHEMICAL MEDIATORS.

EXUDATION: FLUID, PROTEINS, RED BLOOD CELLS, AND WHITE BLOOD CELLS ESCAPE FROM THE INTRAVASCULAR SPACE AS A RESULT OF INCREASED OSMOTIC PRESSURE EXTRAVASCULARLY AND INCREASED HYDROSTATIC PRESSURE INTRAVASCULARLY

VASCULAR STASIS: SLOWING OF THE BLOOD IN THE BLOODSTREAM WITH VASODILATION AN FLUID EXUDATION TO ALLOW CHEMICAL MEDIATORS AND INFLAMMATORY CELLS TO COLLE AND RESPOND TO THE STIMULUS.

VASCULAR RESPONSE	CELLULAR RESPONSE	PHAGOCYTOSIS
▪ Tissue damage causes brief, initial vasoconstriction, rapidly followed by vasodilation (resulting in redness + warmth) ▪ Inflammatory mediators (histamine, prostaglandins, bradykinins) released in the innate immune response and by damaged tissue dilate local blood vessels and increase the permeability of capillaries in the area ▪ Protein-rich fluid (exudate) accumulates in interstitial spaces, causing swelling and pain ▪ Resulting oedema slows blood flow and with clotting, helps localise and prevent microorganisms from spreading	▪ <1hr after injury, phagocytic blood cells brought into damaged tissue ▪ Loss of serous fluid from capillaries increases blood viscosity in the area and slows blood flow ▪ Leukocytes move to the vessel periphery and adhere to the capillary endothelium --> endothelial cells separate, allowing leukocytes to transmigrate through vessel walls into tissue spaces ▪ Chemotactic signals draw the leukocytes to the site of injury/infection	▪ Once attracted to the inflammatory site, phagocytes engulf the foreign agent by projecting pseudopodia ("false feet") in all directions around it ▪ It produces a phagosome containing the antigen, which is ingested into the cytoplasm ▪ Once engulfed, lysosomes fuse with the phagosome, killing any live organism and releasing digestive enzymes, which destroy the antigen

TABLE 11.2 Major chemical mediators of inflammation

FACTOR	SOURCE	EFFECT
Histamine	Mast cells, basophils and platelets	Vasodilation and increased capillary permeability producing tissue redness, warmth and oedema
Kinins (bradykinin and others)	Plasma proteins	Histamine-like effects; chemotaxis and pain inducers
Prostaglandins	Formed from arachidonic acid found in cell membranes	Histamine-like effects; chemotaxis, pain, and fever inducers
Leukotrienes	Formed from arachidonic acid	Smooth muscle constriction (especially bronchoconstriction), increased vascular permeability, chemotaxis