

PATHOLOGY TEAMWORK

MED 444



INFLAMMATION AND REPAIR (1)

Editing file

OBJECTIVES

- 🔍 Define inflammation.
- 🔍 List cells & molecules that play important roles in inflammation.
- 🔍 Types of inflammation: acute and chronic inflammation.
- 🔍 Recognize the cardinal signs of inflammation.
- 🔍 Describe the sequence of vascular changes in acute inflammation. (vasodilation, increased permeability) and their purpose.
- 🔍 Compare normal capillary exchanges with exchange during inflammatory response.
- 🔍 Define the terms edema, transudate and exudate.

COLOR INDEX:

MAIN TEXT (BLACK)

FEMALE SLIDES (PINK)

MALE SLIDES (BLUE)

IMPORTANT (RED)

DR'S NOTE (GREEN)

EXTRA INFO (GREY)



What is Inflammation ?

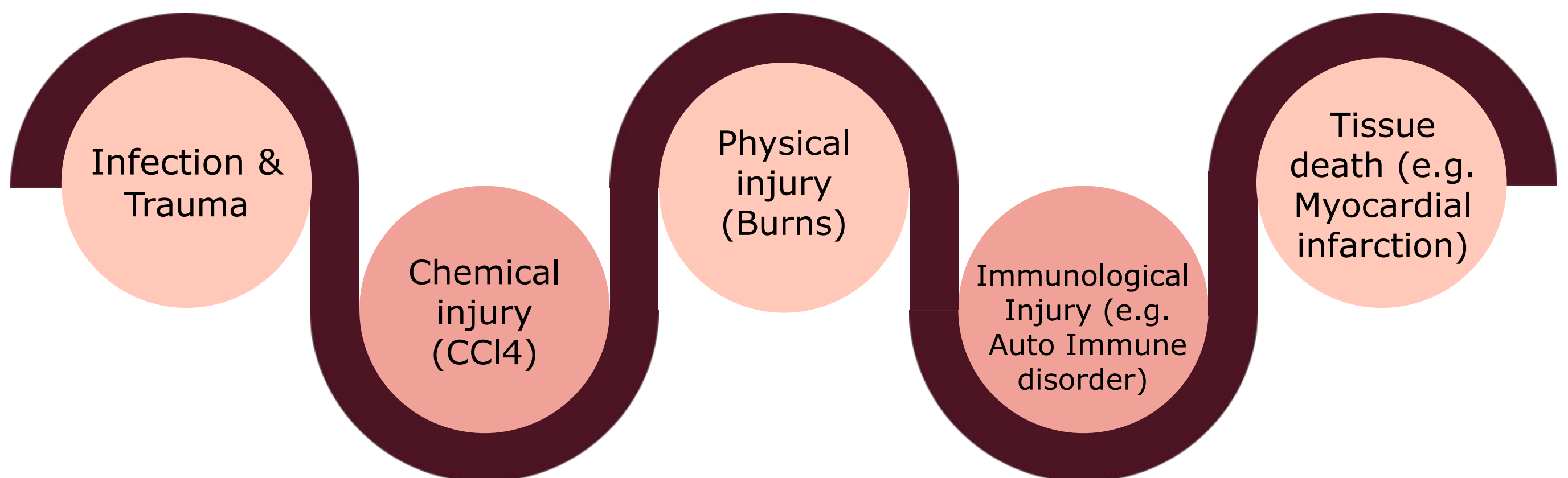
Inflammation is a local response of the vascularized living tissue to infection and damaged tissue (injury/necrosis) that brings cells and molecules of host defense from the circulation to the sites where they are needed.

The aim:

- to localize and eliminate the causative agents.
- limit tissue injury.
- restore tissue to normal.

Therefore, Inflammation is part of immunity: This is a broad protective response (innate immunity) (Immunity towards everything)

Causes Of Inflammation :

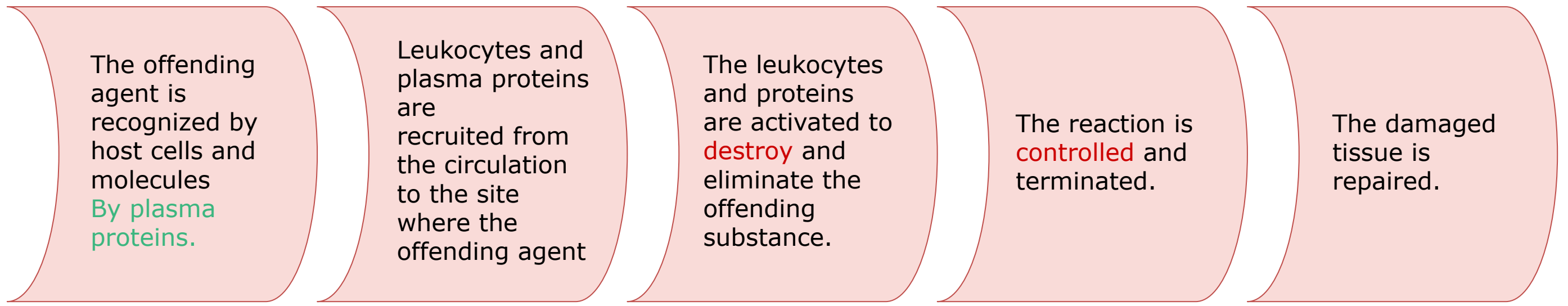


Steps Of Inflammation :

- 1 Injury
- 2 Tissue damage
- 3 Tissue response
- 4 Eliminates the initial cause of injury and the effect of injury (pathogen and necrotic tissue)
- 5 Repair
By regeneration or scar formation



Steps Of Inflammation :



Can Inflammation be harmful? **Yes**

Acute

Acute respiratory distress syndrome, asthma and septic shock

43: The response to sudden body damage, such as cutting your finger

Chronic

تصلب الشرايين
Arthritis, atherosclerosis and pulmonary fibrosis
التليف الرئوي

43: Your body continues sending inflammatory cells even when there is no outside danger. For example, in rheumatoid arthritis

GIRL'S SLIDES

Disorders

Cells and Molecules Involved in Injury

Acute	
Acute respiratory distress syndrome	Neutrophils
Asthma	Eosinophils , IgE antibodies
Glomerulonephritis	Antibodies, Complement, neutrophils, monocytes
Septic shock	Cytokines

BOY'S SLIDES

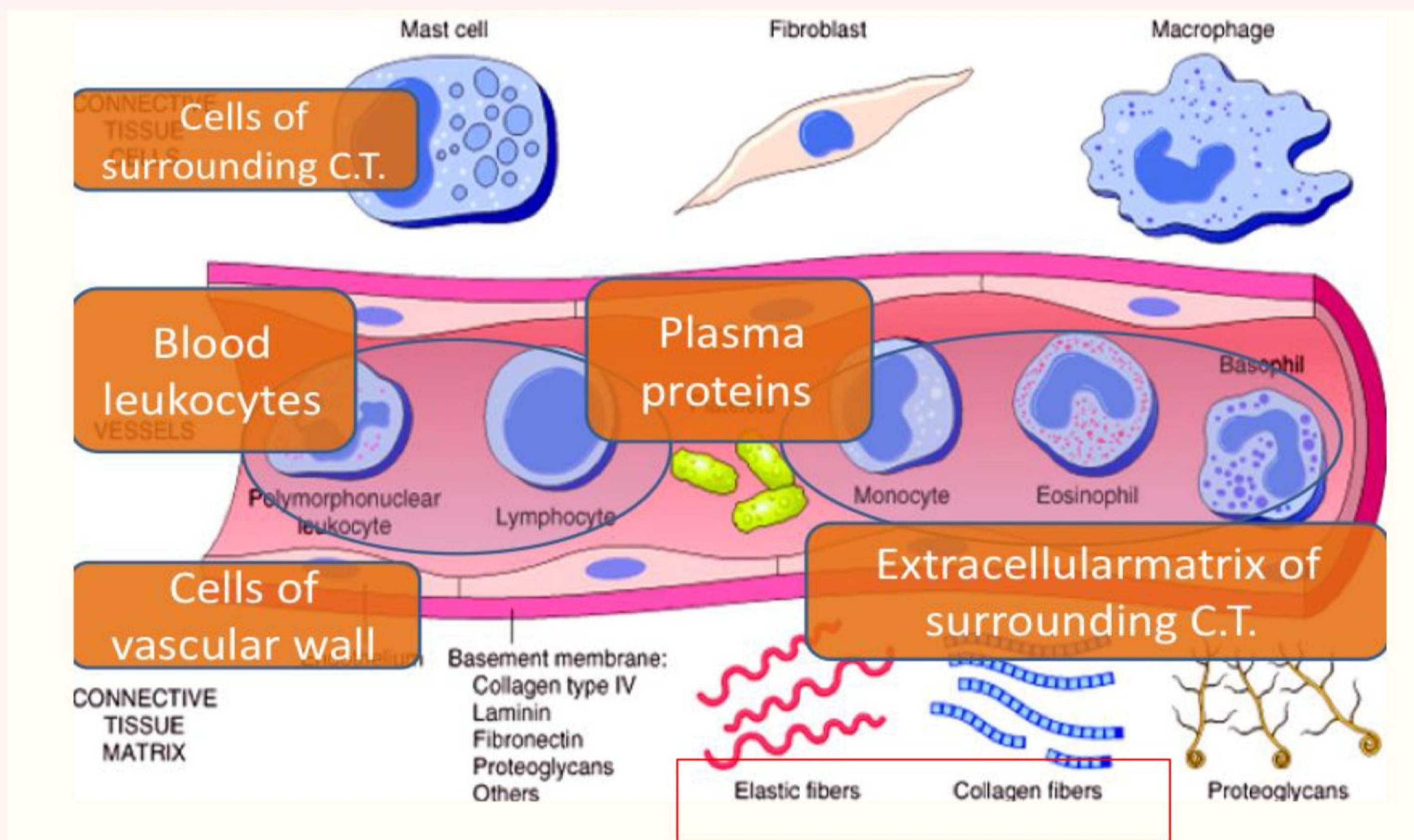
Inflammation

DEFINITION

Inflammation is **terminated** when the **offending agent** is eliminated and the secreted **mediators** are **broken** down or dissipated.

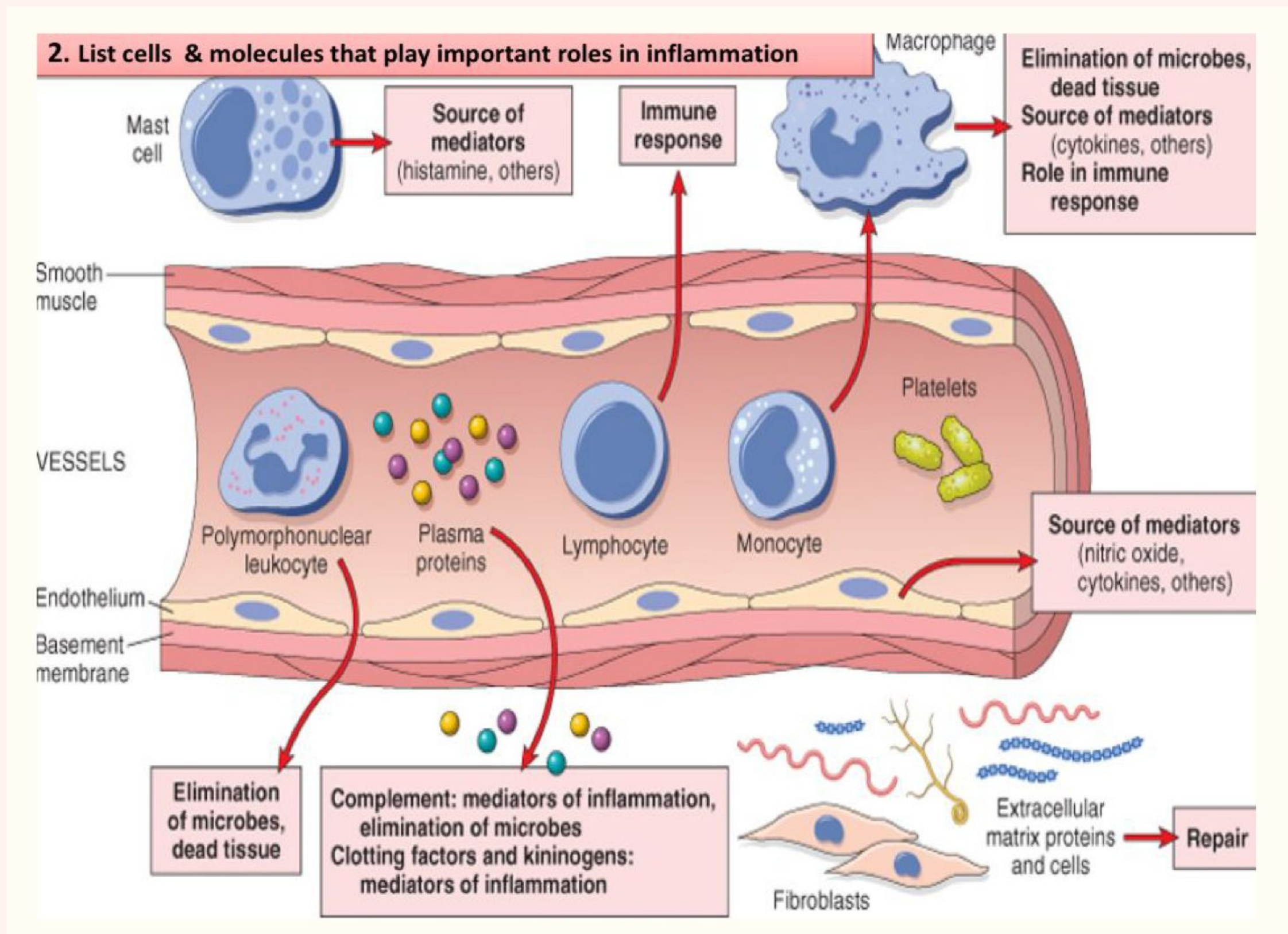
There are active **anti-inflammatory** mechanisms that serve to control the response and prevent it from causing excessive damage to the host.
43: (E.g. Enzymatic lysis of protein)

Cells And Molecules That Play Important Roles In Inflammation



- Inflammation is mediated by chemical substances called Chemical Mediators

Chemical Mediators



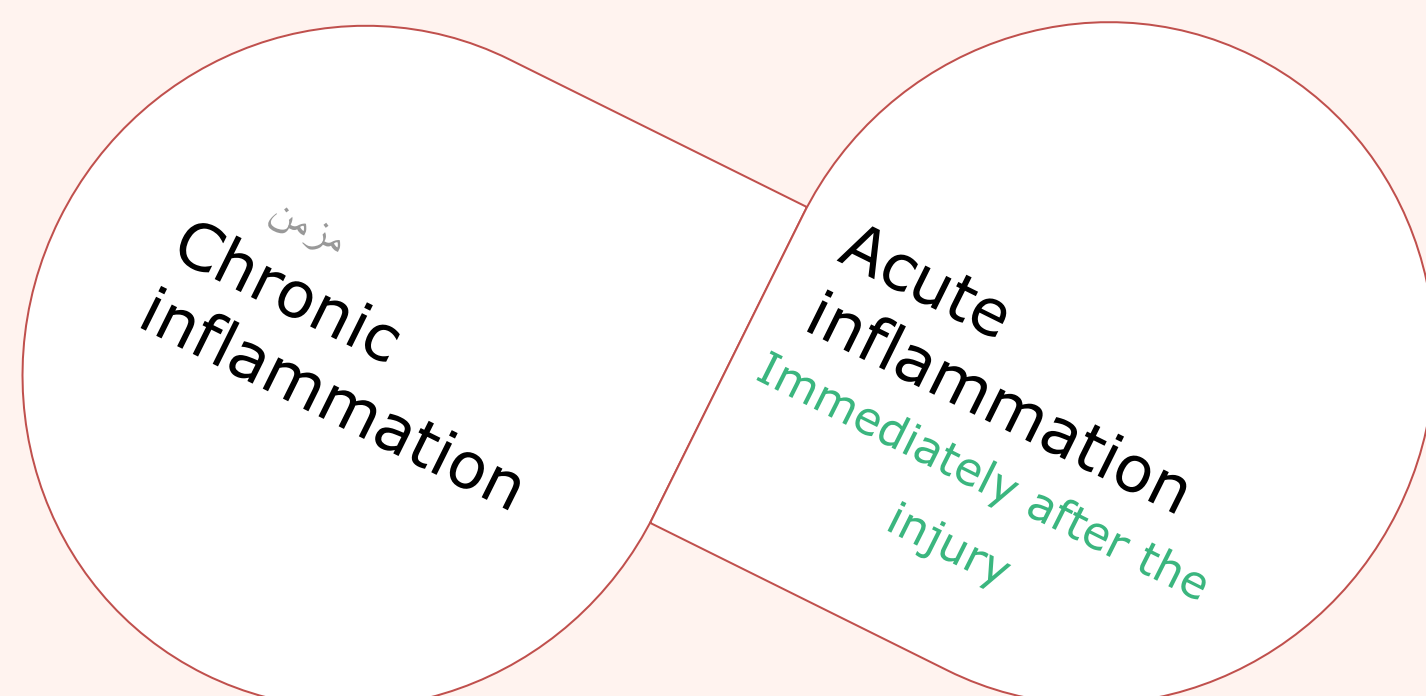
What is the **source** of these chemical mediators?

1. Cells :

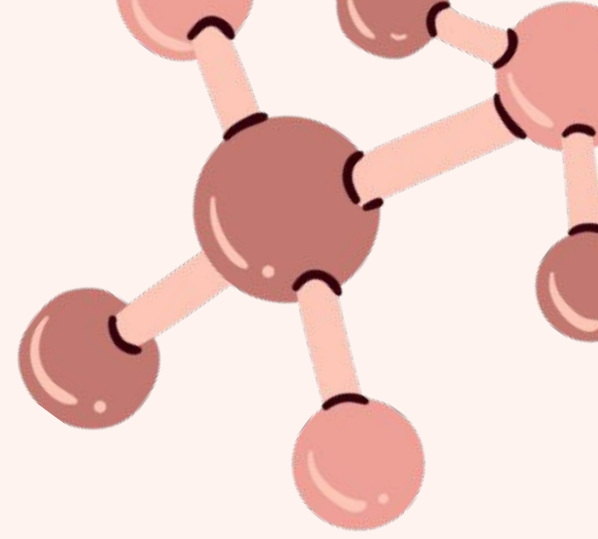
- **leukocytes**
- **Endothelium** : source of nitric , cytokines
- **Mast cells : source of histamine**

2. **Plasma proteins** work as **complement**

Types Of Inflammation



Features Of Acute And Chronic Inflammations



Features	Acute	Chronic
Onset	Fast: minutes or hours	Slow : days, weeks
Cellular infiltrate	neutrophils	lymphocytes and macrophages
Tissue injury fibrosis	Mild, self limited	Often severe & progressive
Local and Systemic Sign	Prominent	Less prominent, may be subtle
E.g (43 Team)	- hepatitis A - Pneumonia (acute lung inflammation)	- hepatitis B - Tuberculosis

Acute inflammation



A **rapid** response to an injurious agent that serves to deliver mediators of host defense-leukocytes and plasma proteins-to the site of injury

The Outcome Of Acute inflammation

1

elimination of the noxious stimulus, followed by decline of the reaction and repair of the damaged tissue

43: Noxious = Toxic

OR

2

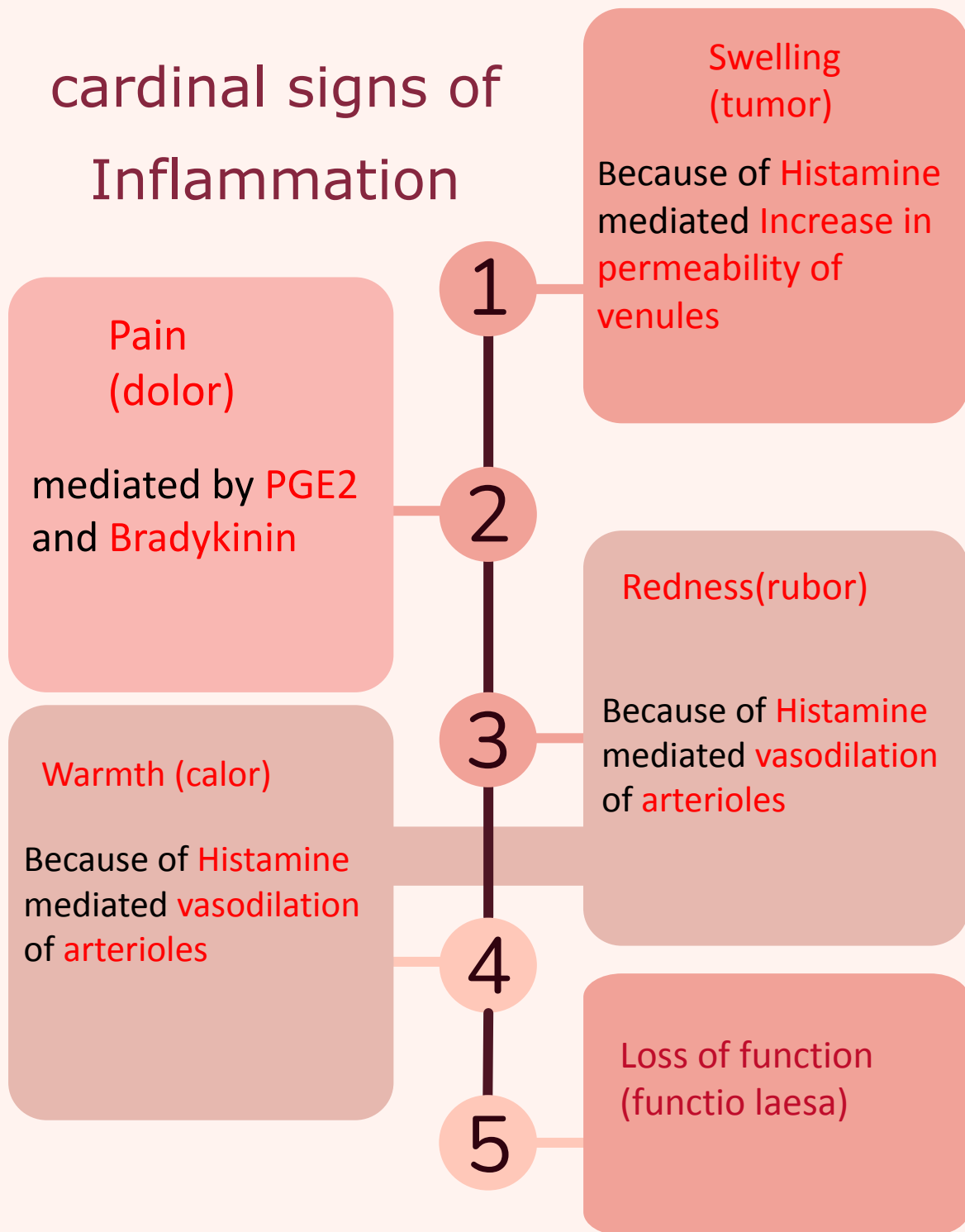
persistent injury resulting in chronic inflammation
E.g: hepatitis

Inflammatory reactions underlie common chronic diseases, such as rheumatoid arthritis, atherosclerosis, lung fibrosis, asthma and life-threatening hypersensitivity reactions to insect bites, drugs, and toxins

Anti-inflammatory drugs would control the harmful sequelae of inflammation

cardinal signs of Inflammation, vascular event sequencing, and events of acute inflammation

cardinal signs of Inflammation

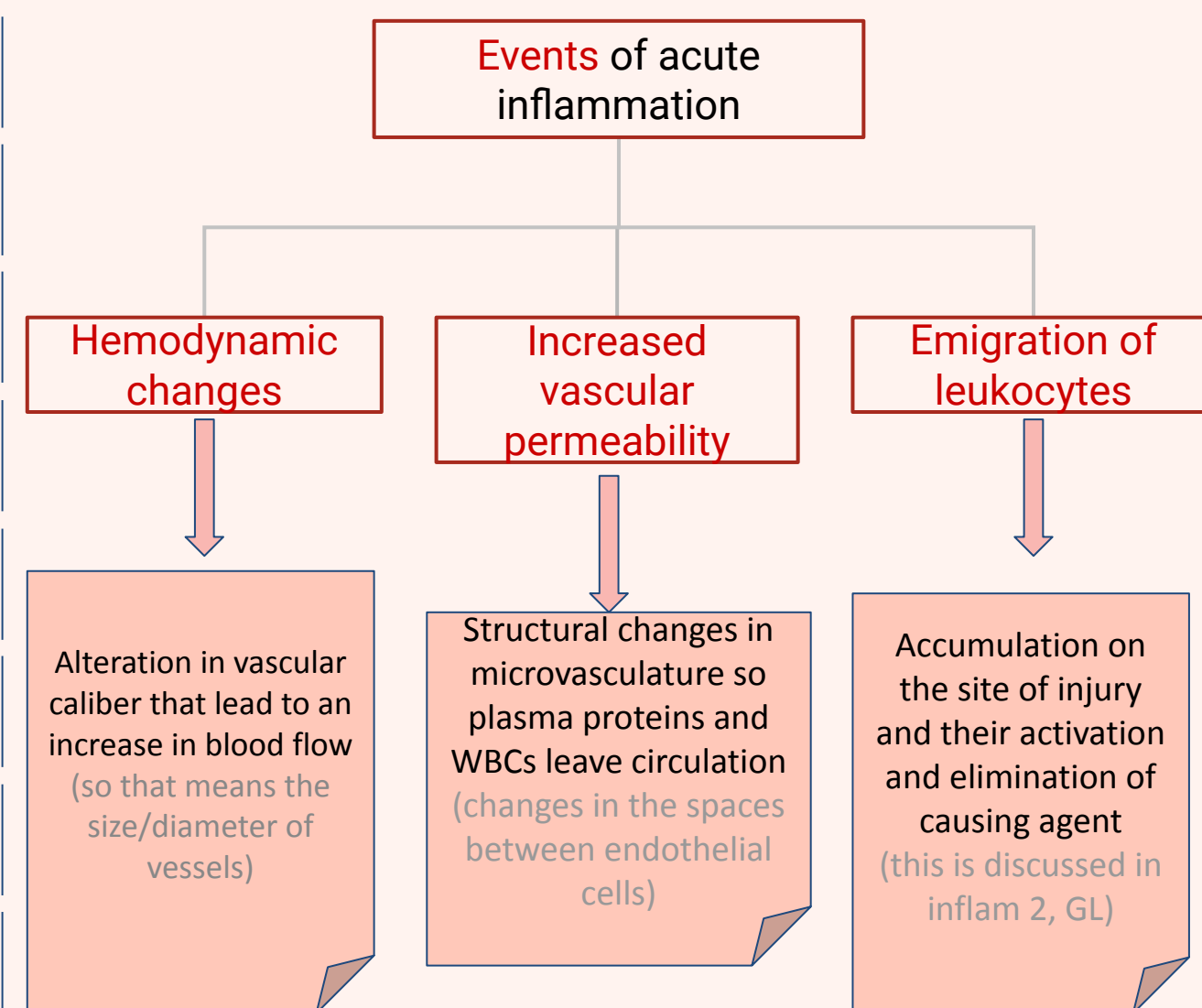


vascular event sequencing in acute inflammation

- 1-Reflex vasoconstriction
- 2-Vasodilation
- 3-Increased capillary permeability

CD IP

events of acute inflammation



SPRAWL



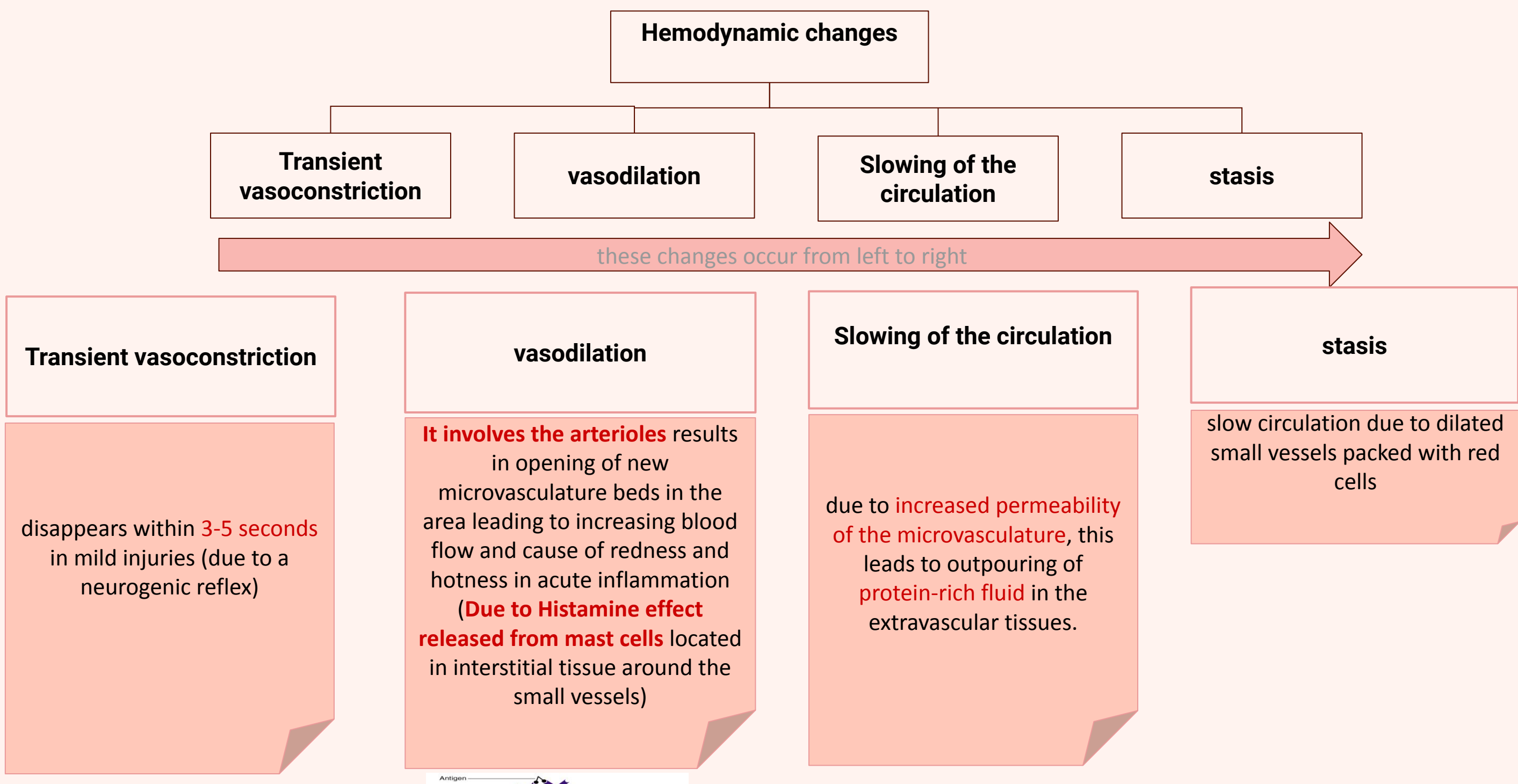
You after 7 hours of non-stop lectures and No lunch break

“itis” as a suffix means inflammation (like dermatitis or vasculitis)

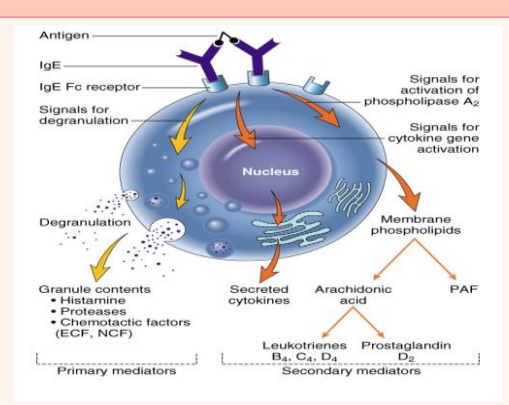
EXTRA INFO

endothelium = simple squamous epithelium of blood vessels

Events of acute inflammation (hemodynamic changes & Increased vascular permeability)



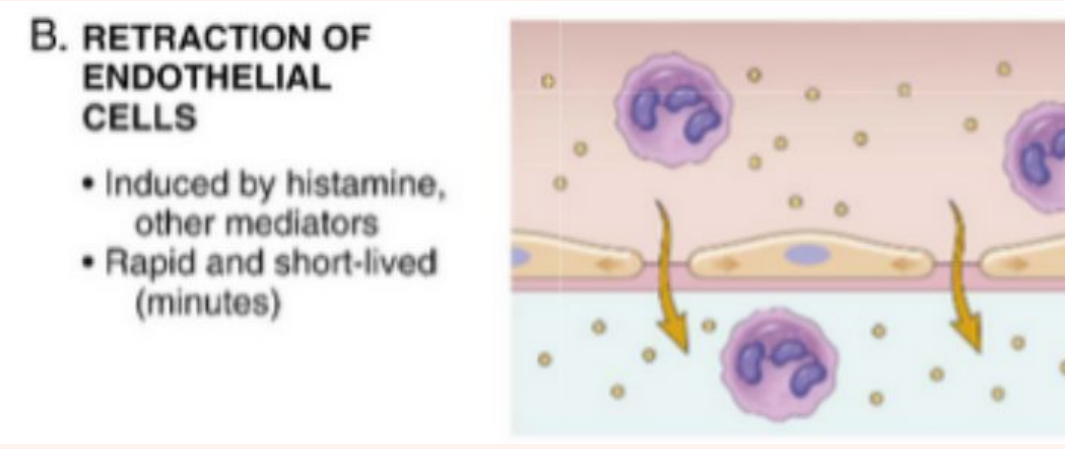
EXTRA INFO
Transient = temporary



EXTRA INFO
This explains why exudate happens in Inflammation

Increased vascular permeability (most important)

- **A hallmark of acute inflammation** (escape of protein rich fluid)
- induced by **histamine, kinins**, and other mediators (**bradykinin**)
- It affects small & medium size **venules**, through gaps between endothelial cells
- It results in **swelling** which occurs as a **cardinal sign of inflammation**



EXTRA INFO
the picture above happens in normal inflammation, but it's special to venules, while a direct injury to the endothelium would make any blood vessel more permeable, but that's abnormal

Normal capillary fluid exchange and exchange during inflammation

Edema: excess of fluid in the interstitial or serous cavities (which could be **exudate** or **transudate**)

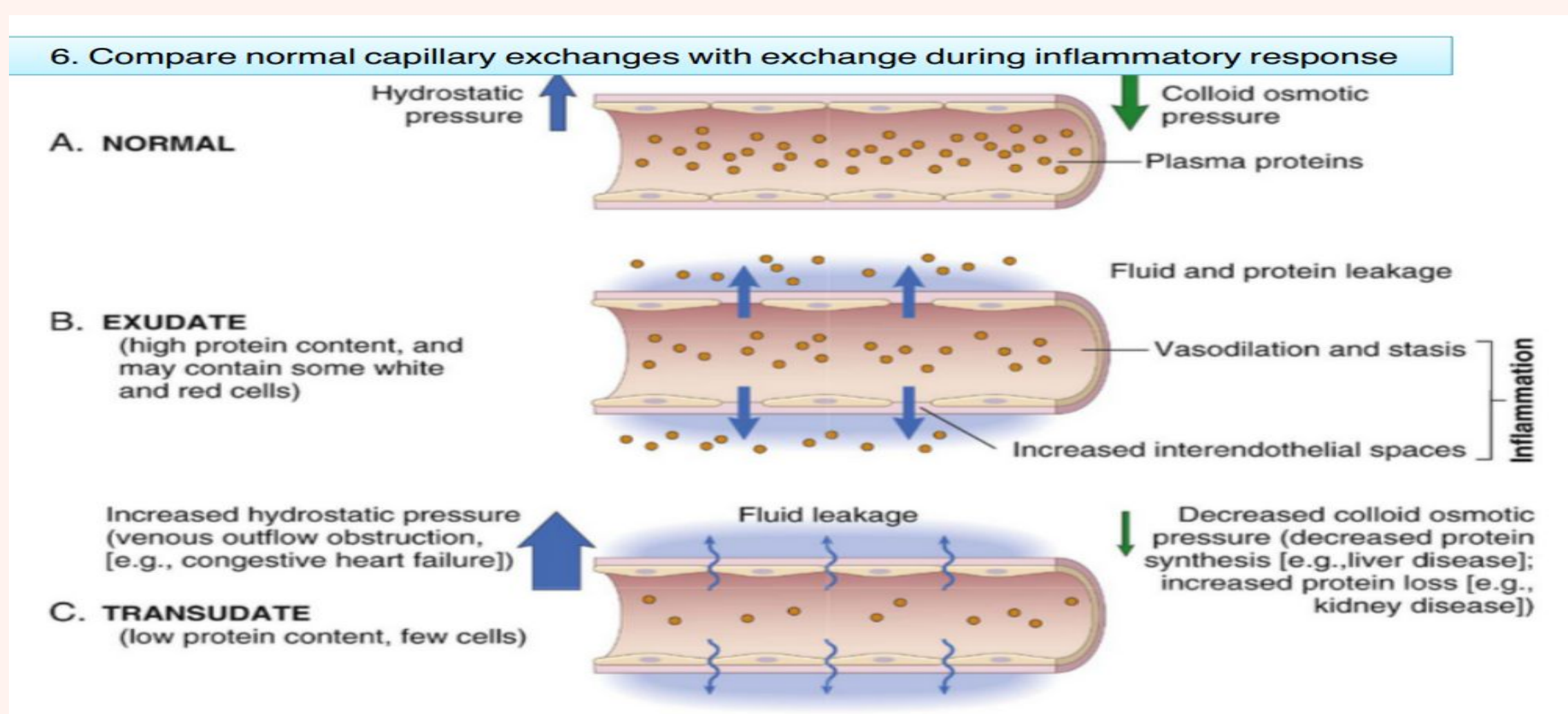
Edema

Transudate

is a fluid with **low protein content** and a specific gravity of less than 1.012. It is essentially an ultrafiltrate of blood plasma that results from **osmotic or hydrostatic imbalance** across the vessel wall **without an increase in vascular permeability**.

Exudate

An **inflammatory** extravascular fluid that has a **high protein concentration**, cellular debris, and a specific gravity above 1.020. It implies **significant alteration in the normal permeability** of small blood vessels in the area of injury.



EXTRA INFO

These diagrams are important to understand the idea behind normal and abnormal fluid movement throughout the body.

Increased blood volume leads to increased local hydrostatic pressure leading to transudation of protein-poor fluid into the extravascular space. (which is why we have edema in CHF {Congestive Heart Failure})

1- which on of the following is NOT an ancient cardinal sign of inflammation?

A) Tumor	B) Calor	C) Dolor	D) Molor
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2- Escape of a protein-rich fluid is hallmark of which acute inflammation event?

A) Hemodynamic changes	B) Increased vascular permeability	C) emigration of the leukocytes from the microcirculation	D) Transient vasoconstriction of artenolos
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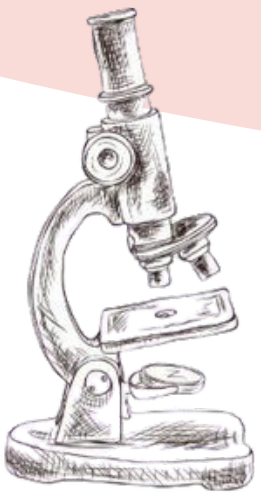
3- An inflammatory extravascular fluid that has a high protein concentration, cellular debris. and a specific gravity above 1.020?

A) Edema	B) Transudate	C) Exudate	D) none of the above
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4- A 41-year-old woman complains of excessive menstrual bleeding and pelvic pain of 4 months. She uses an intrauterine device for contraception. Endometrial biopsy reveals an excess of plasma cells and macrophages within the stroma. The presence of these cells and scattered lymphoid follicles within the endometrial stroma is evidence of which of the following conditions?

A) Acute inflammation	B) Chronic inflammation	C) Granulation Tissue	D) Granulomatous inflammation
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5- Transudate has			
A)high protein	B)low protein	C)Fibers	D)high gravity specificity
6- How long is transient vasoconstriction?			
A)3-4 hours	B)1-5 minutes	C)3-5 seconds	D) 3-5 years
7- pain is mediated by			
A)histamine	B)bradykinin	C)vasodilation	D)vasoconstriction
8- what is the reason for redness and warmth in inflammations			
A)vasodilation	B)vasoconstriction	C)PGE2	D)bradykinin
9- what is a sign of inflammation			
A)shivering	B)vasoconstriction	C)hypokalemia	D)loss of function



PATHOLOGY TEAM 444

PATHOLOGY TEAMWORK

MED 444

Ritaj Alsubaie

LEADER

Manar Alqahtani

LEADER

Abdulaziz Nasser

LEADER



Shaden Alotaibi



Rahaf Al turki



Layal Alkhalifah



Norah Alnoشان



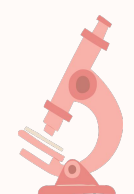
Noor Altalag



Aram Alzahrani



Nisreen Alotaibi



Lana Alfouzan



Seeta bin aqeel



Lujain Darraj



Hessa Alamer



Sahar Alfallaj



Nora Albahily



Sadeem Alotaibi



Abdulmalik Aldafs



Abdumohsen Alrahaimi



Ibrahim Abdallah



Ibrahim Al Bin Ali



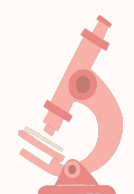
Lubna Alamri



Fahad Albalawi



Jana Alrumaihi



Hmood Alsehali



Osama Alotaibi



Ziyad BuKhari



Abdullah Alzoom



Khalid Alkanhal



Mazen Alzahrani



Rakan Alarifi



Nora Albahily



pathology.444ksu@gmail.com