
# General Pathology

## Inflammation

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# What is Inflammation?

Inflammation is the **body's protective response** to injury, infection, or harmful stimuli. Its goal is to:

- **Eliminate** the cause of injury,
- **Remove** damaged cells,
- **Initiate** healing.

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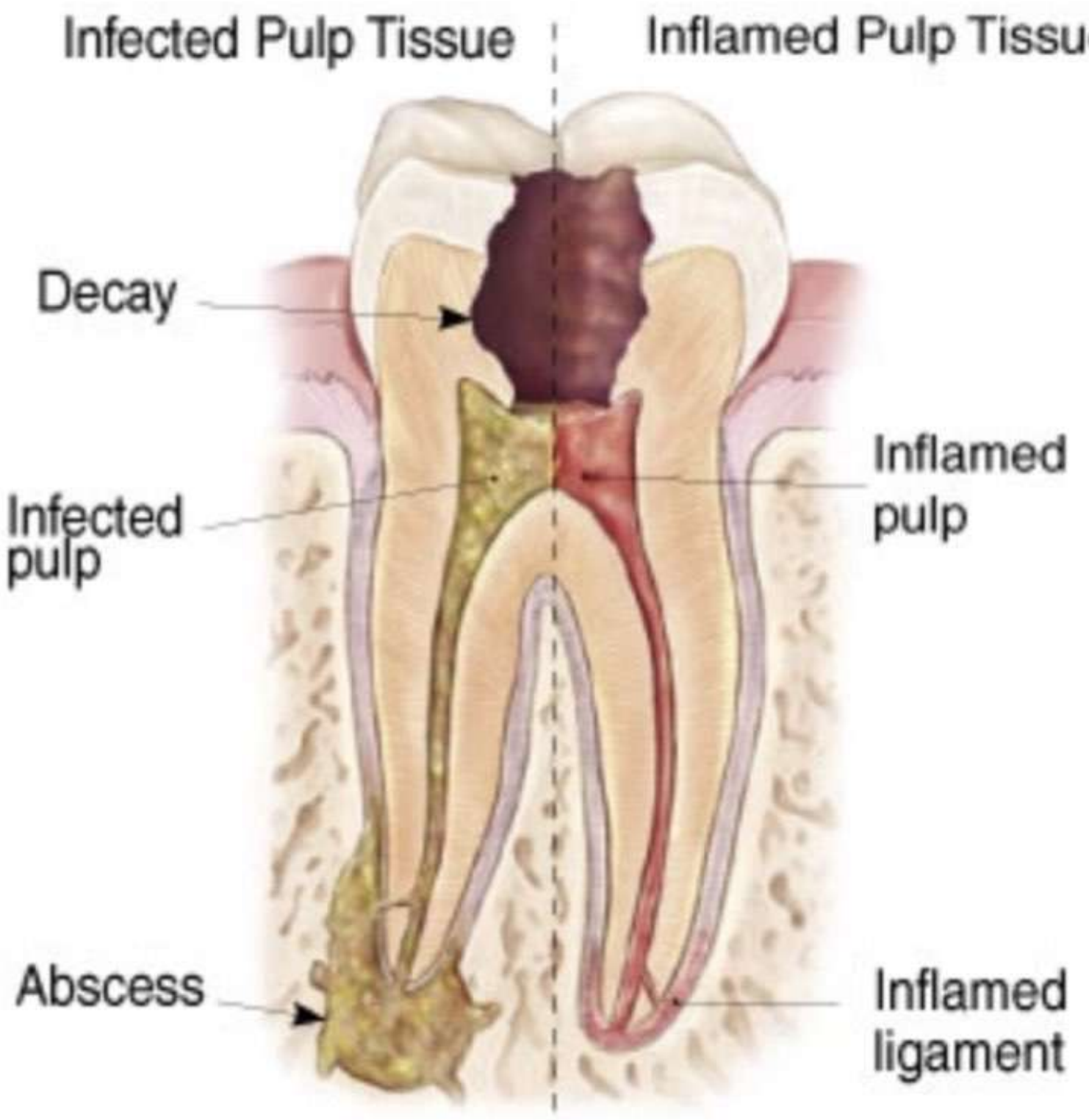


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**Signs of inflammation** include:

- Redness (*rubor*),
- Heat (*calor*),
- Swelling (*tumor*),
- Pain (*dolor*),
- Loss of function (*functio laesa*).

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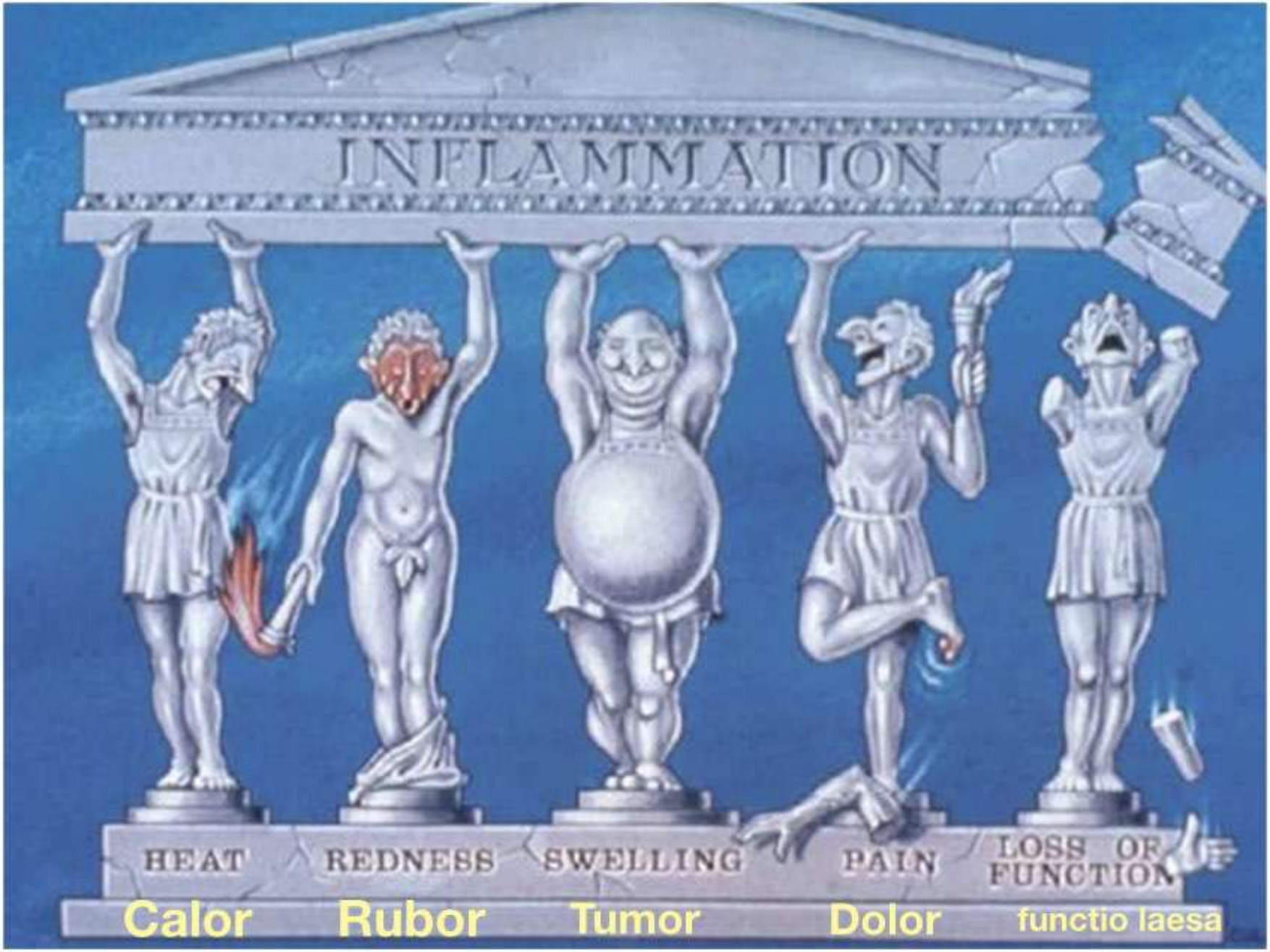


Cardinal signs of inflammation

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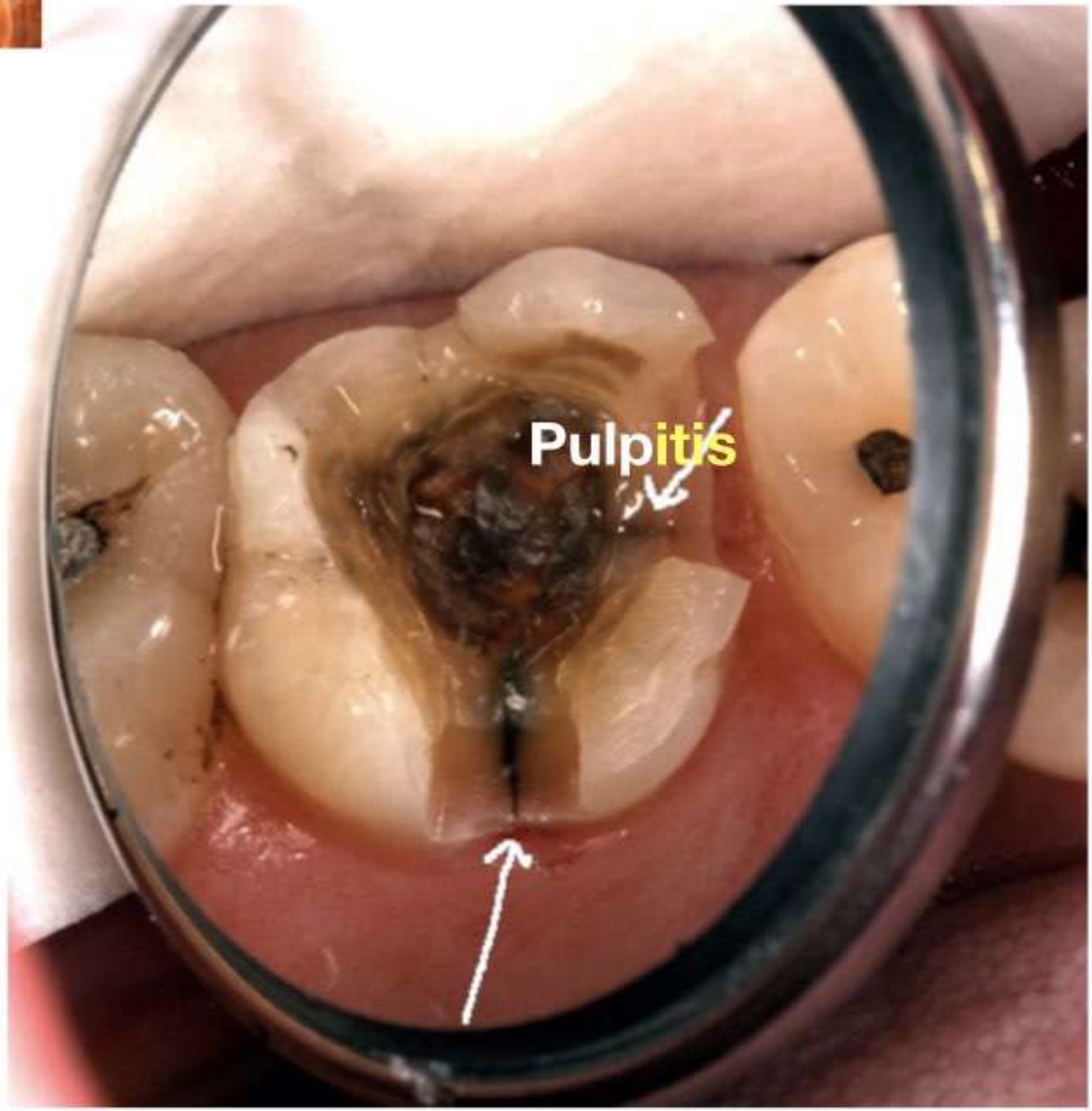
# Inflammation of the pulp _*itis*

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# Inflammation of the gum (gingiva) *itis*


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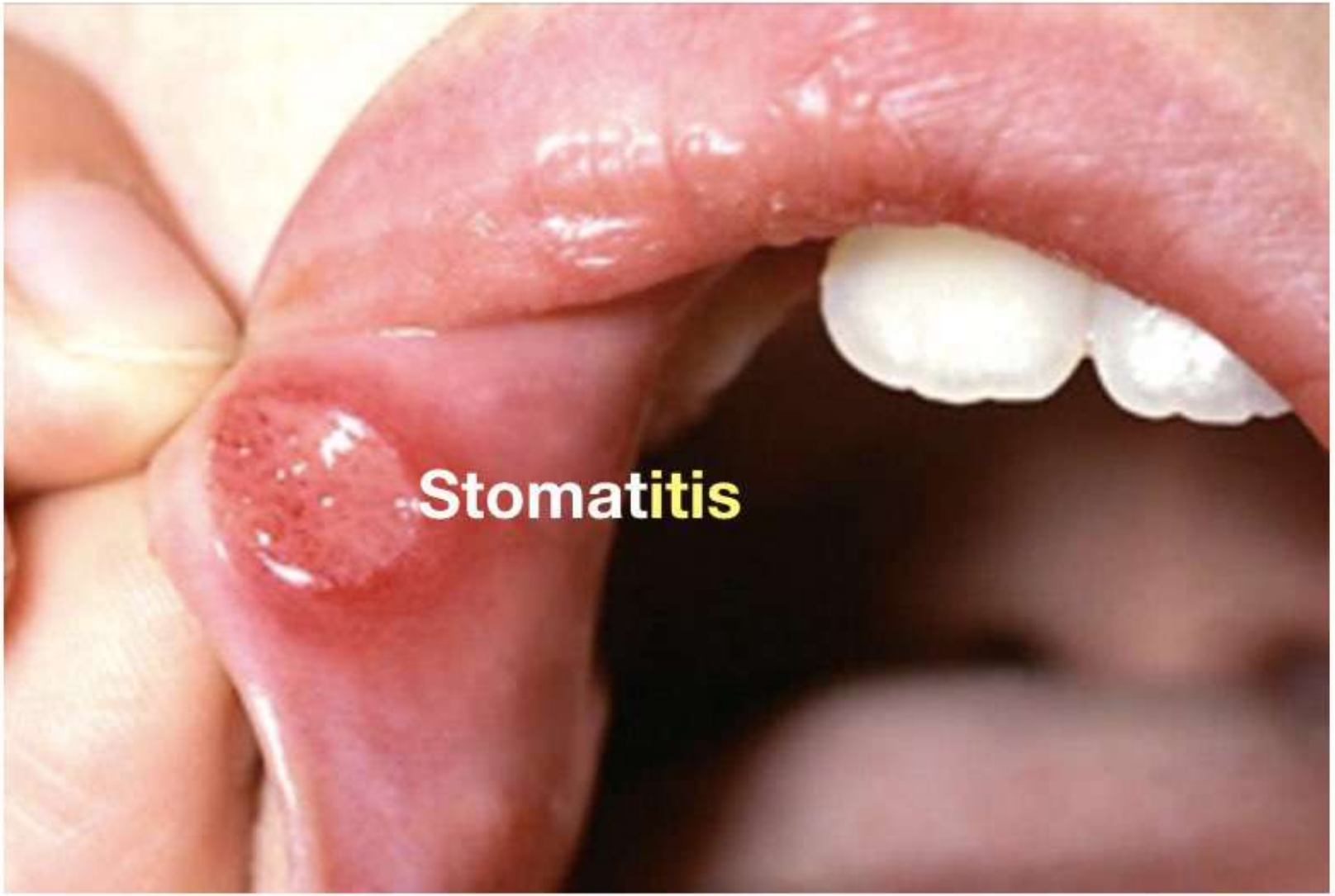
# Inflammation of the mouth (stoma) _itis

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# Inflammation of the tongue (glossa)_*itis*


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# Local Effects

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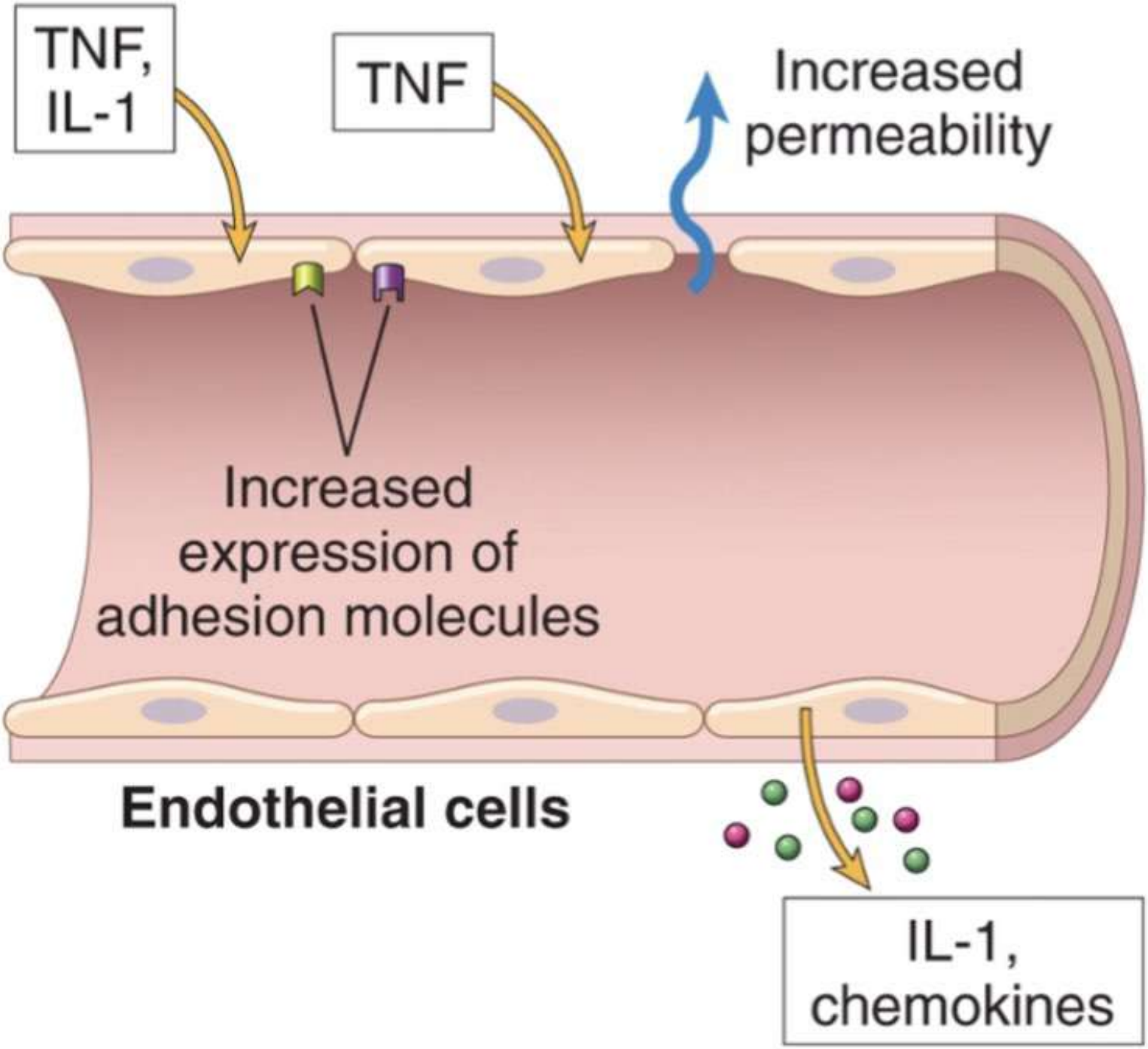


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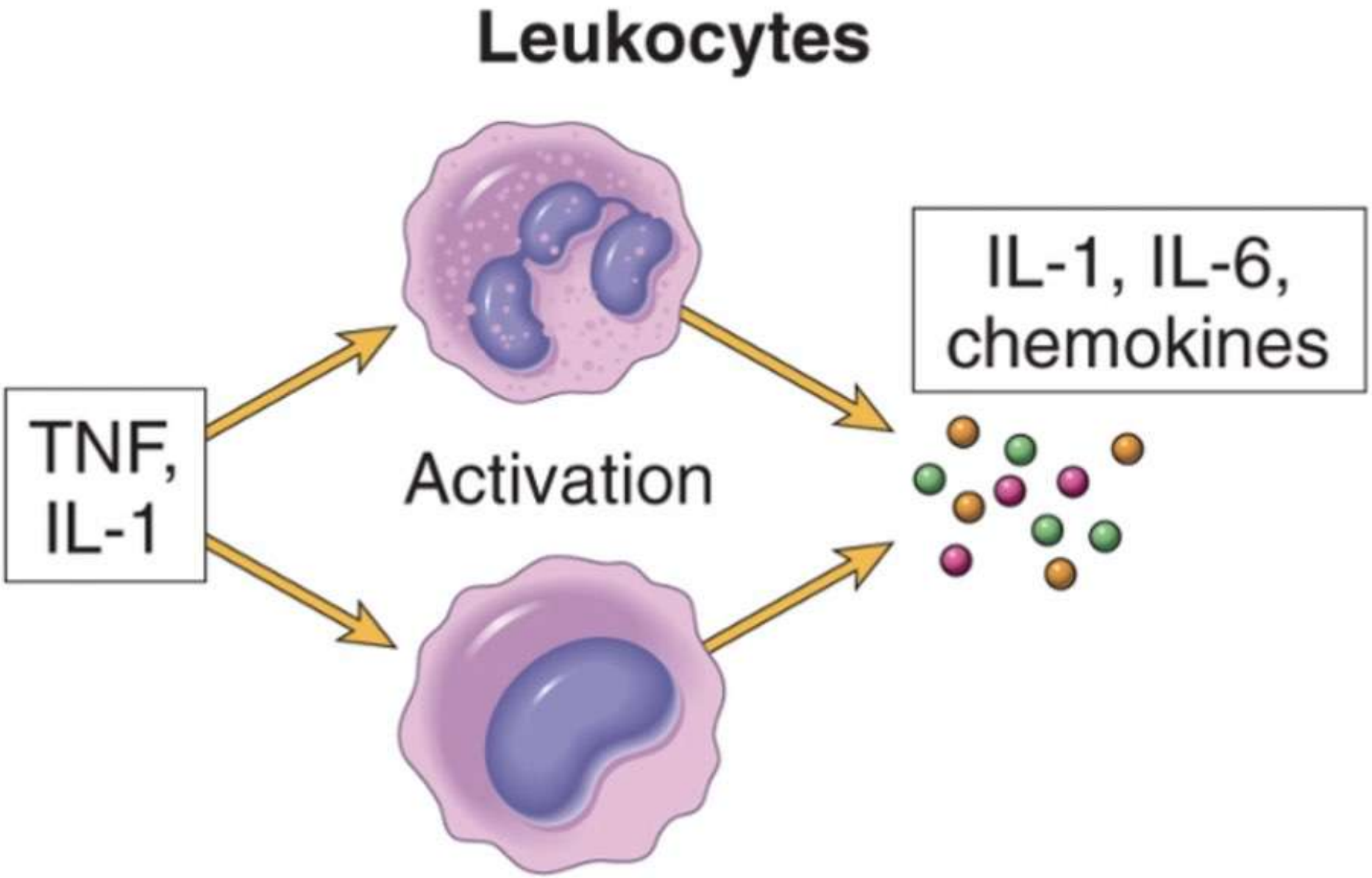


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# Systemic Effects

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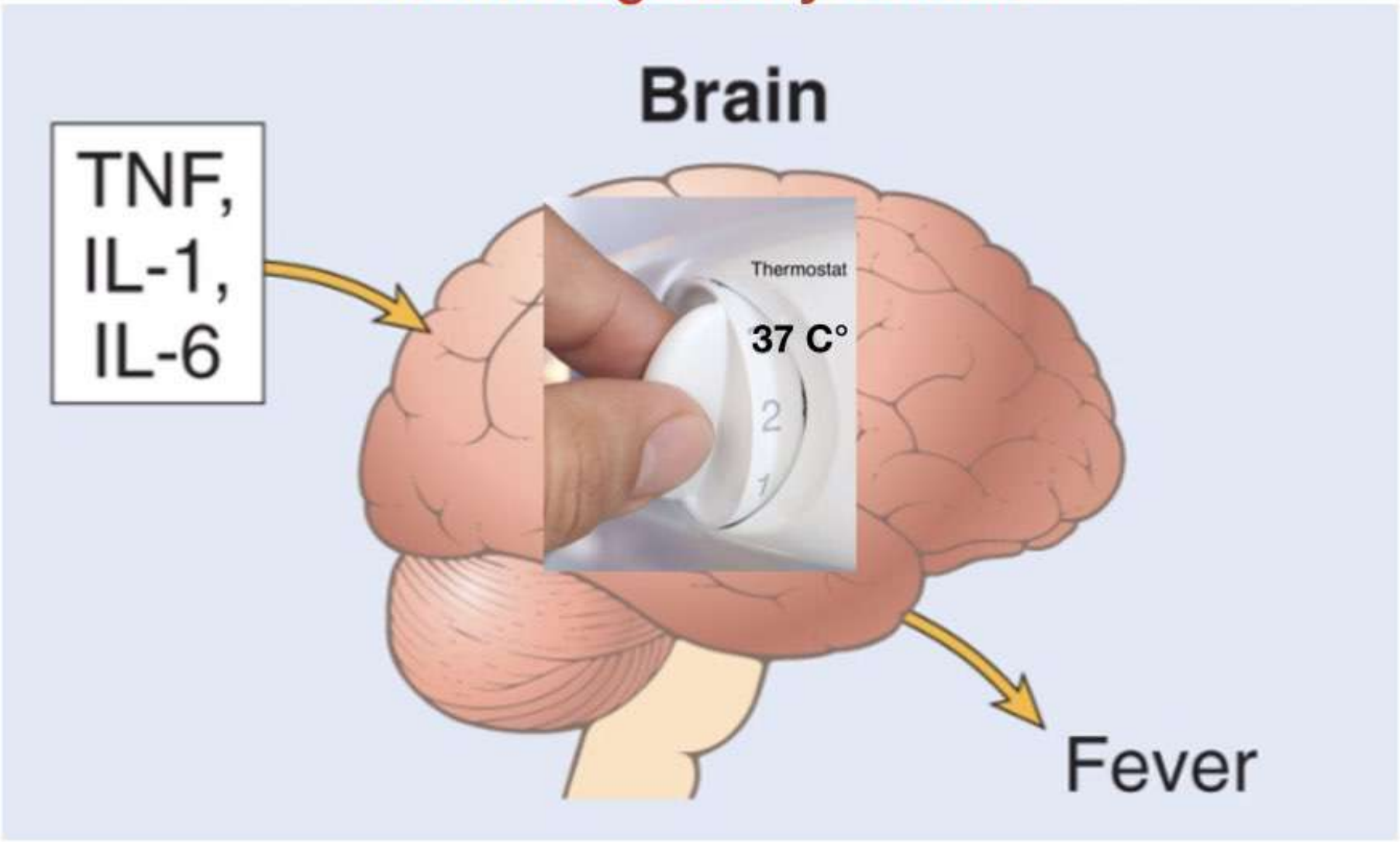
# Thermoregulatory centers

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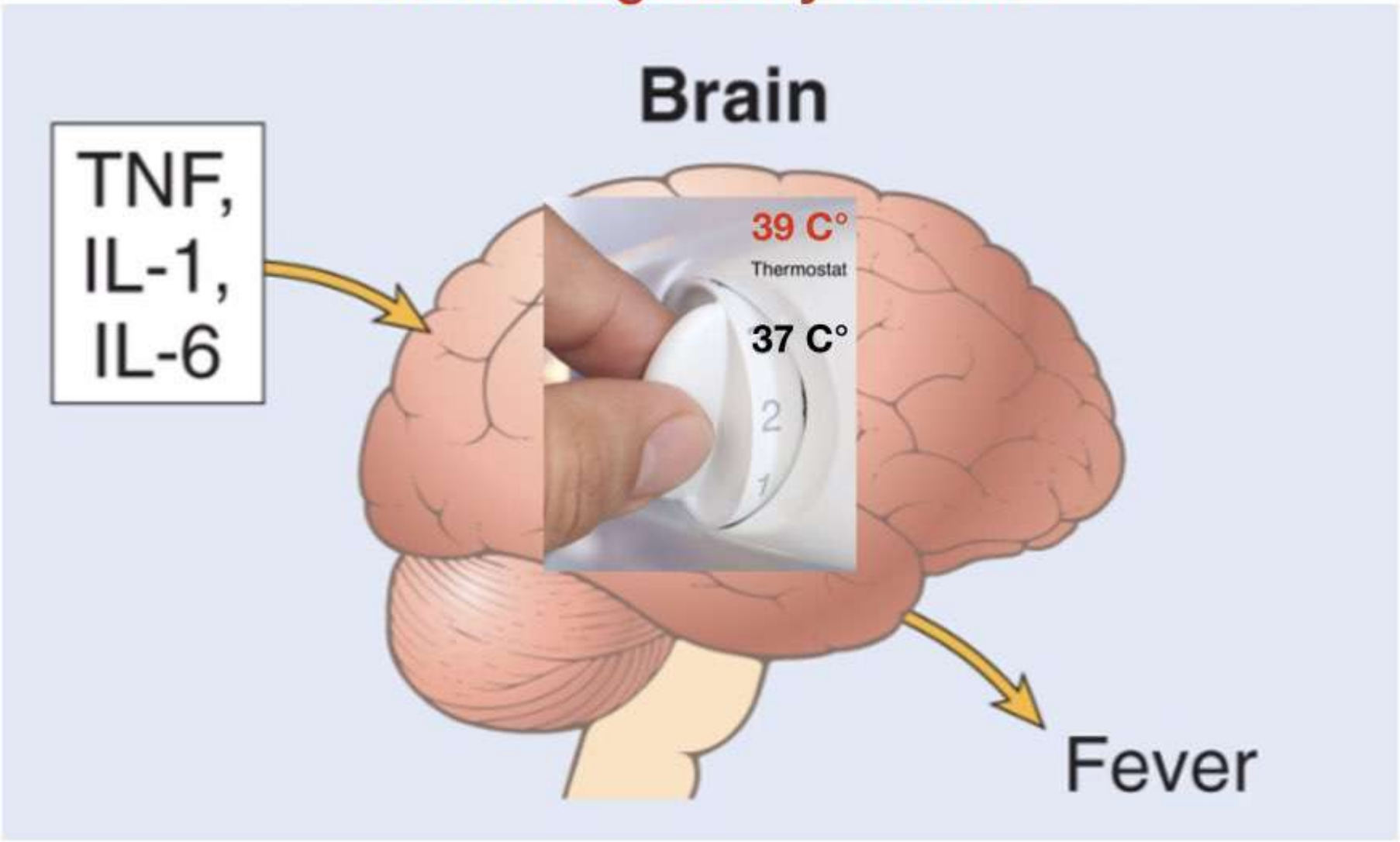
# Thermoregulatory centers

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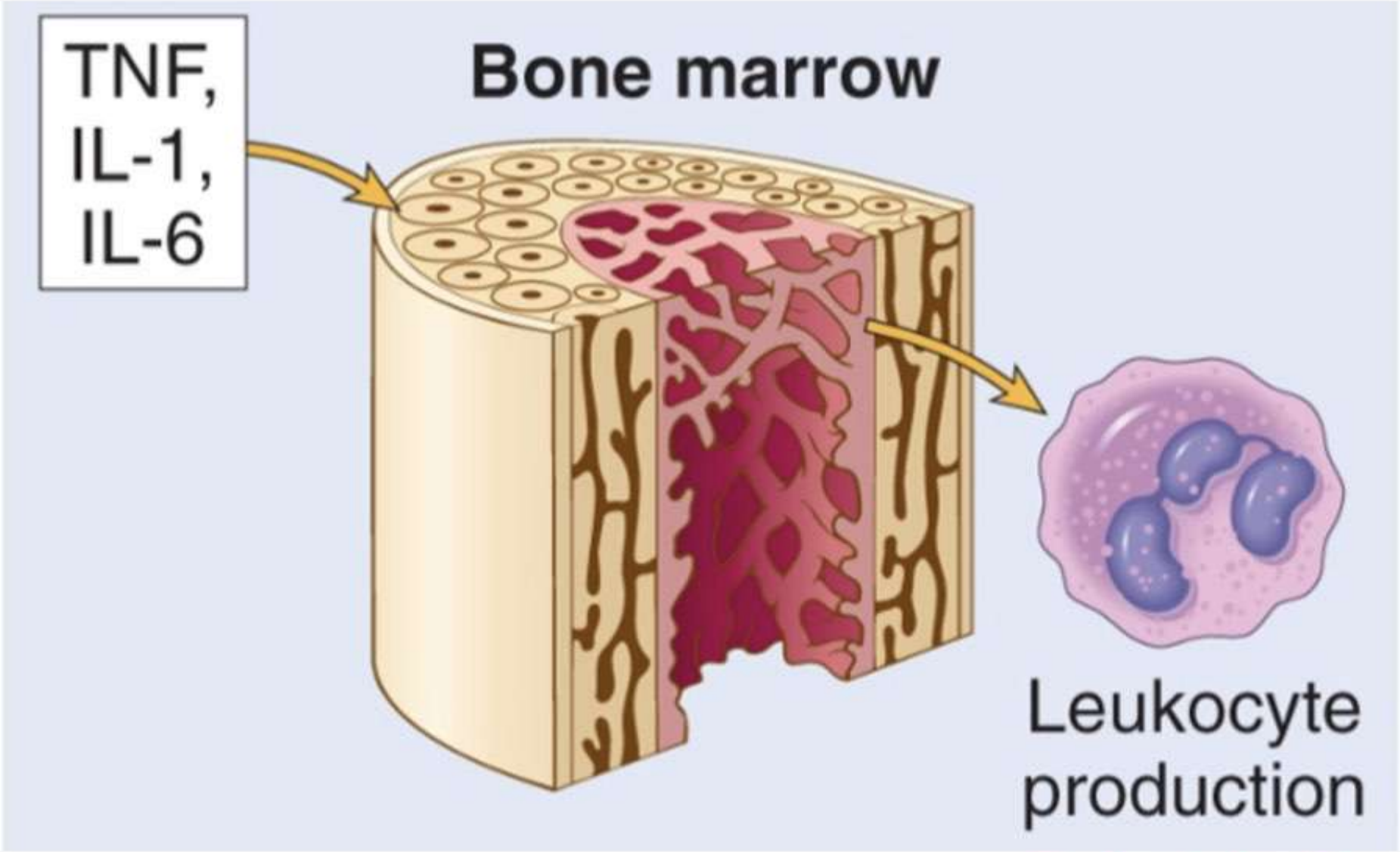


## Leukocytosis

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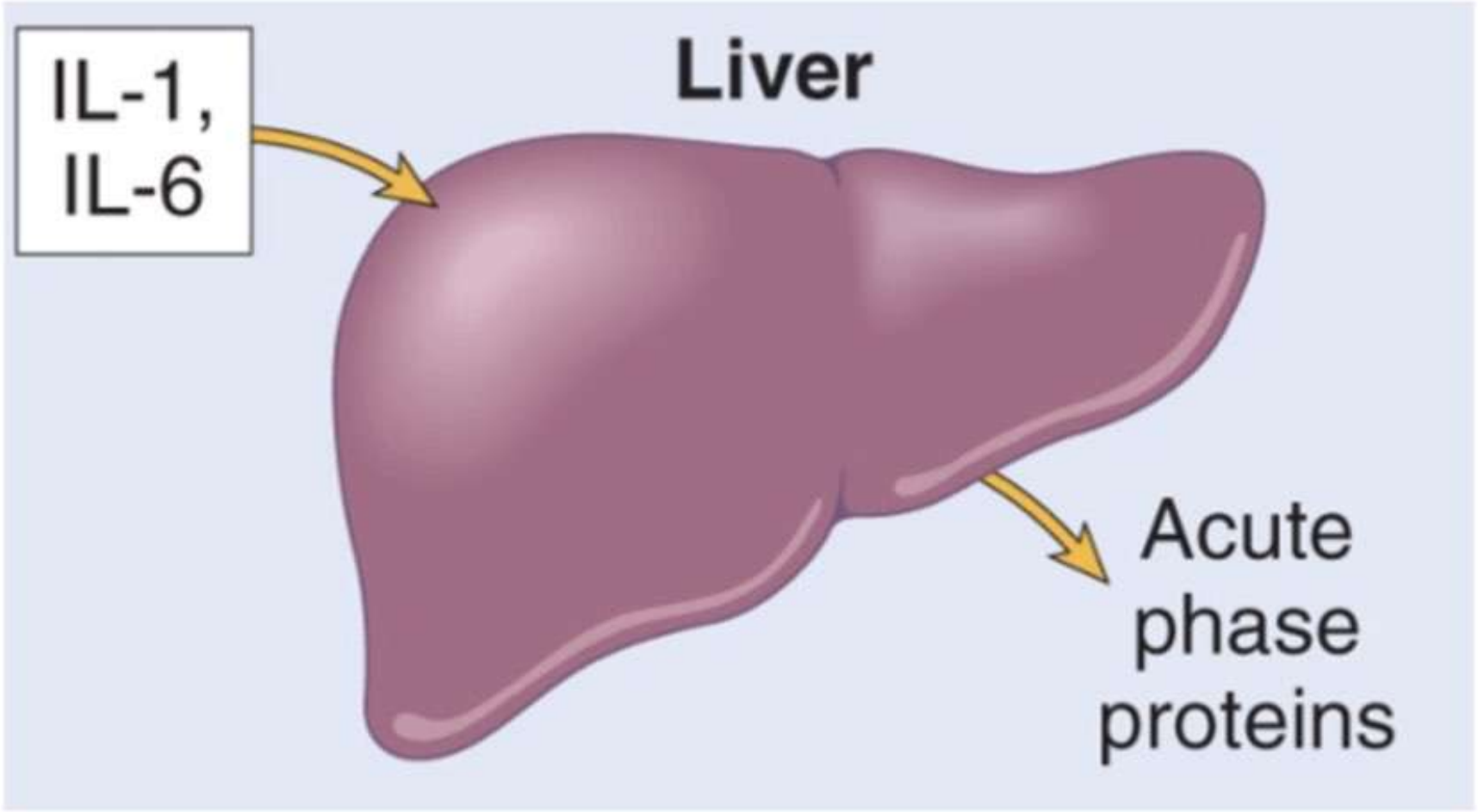


## C-Reactive Protein

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# Side Effects

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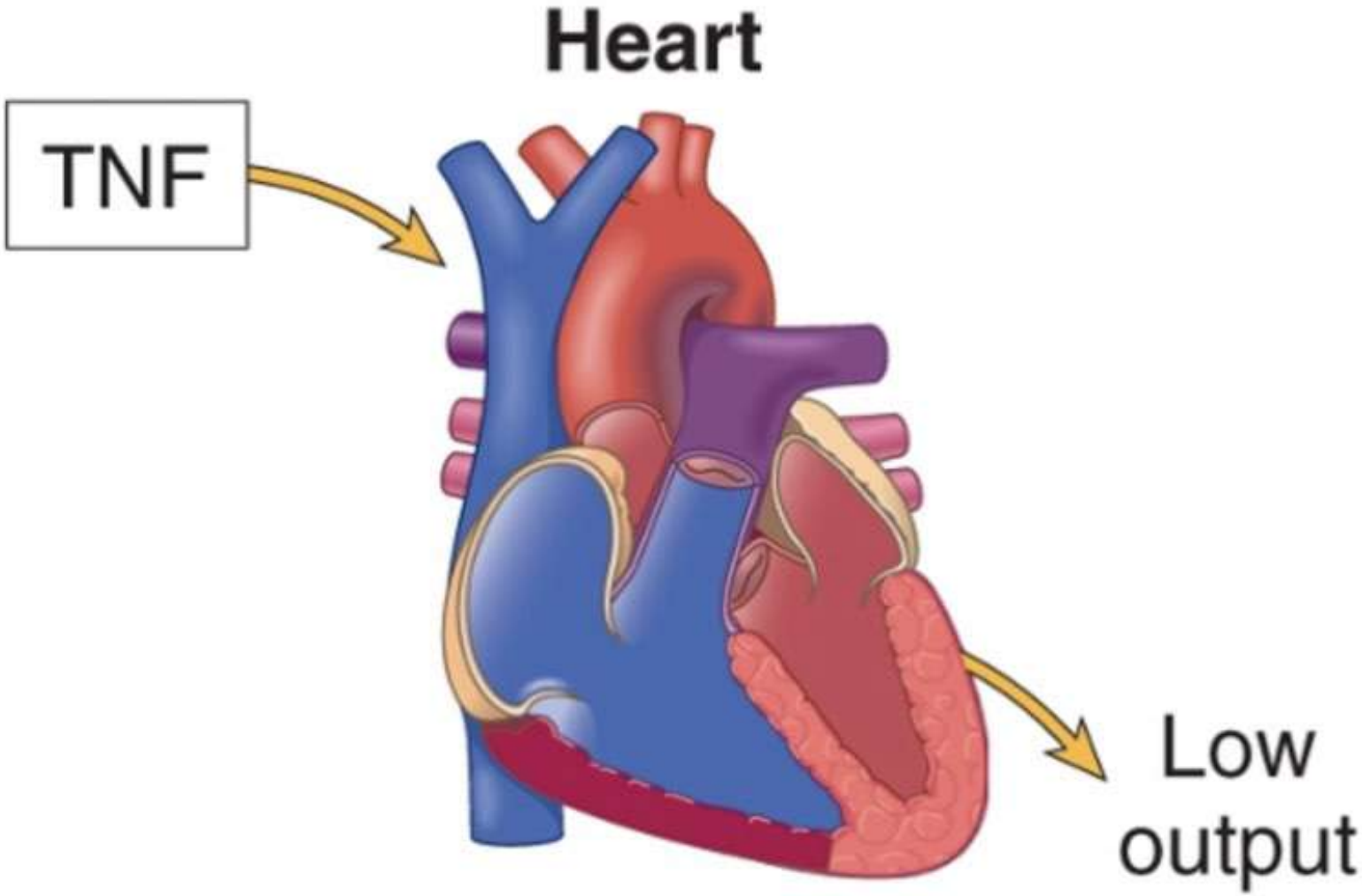


**Septic shock**

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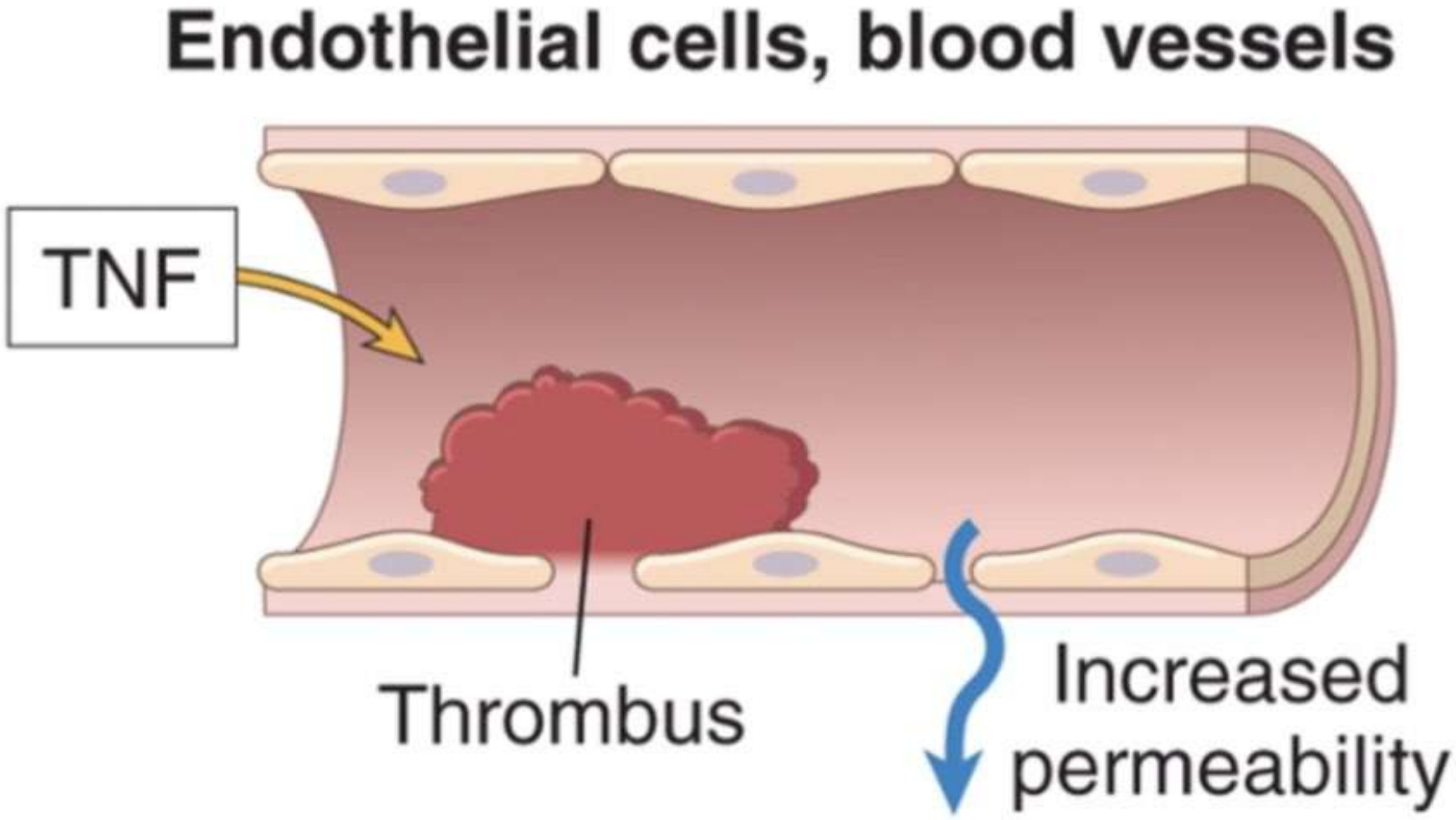


## Disseminated Intravascular Coagulation

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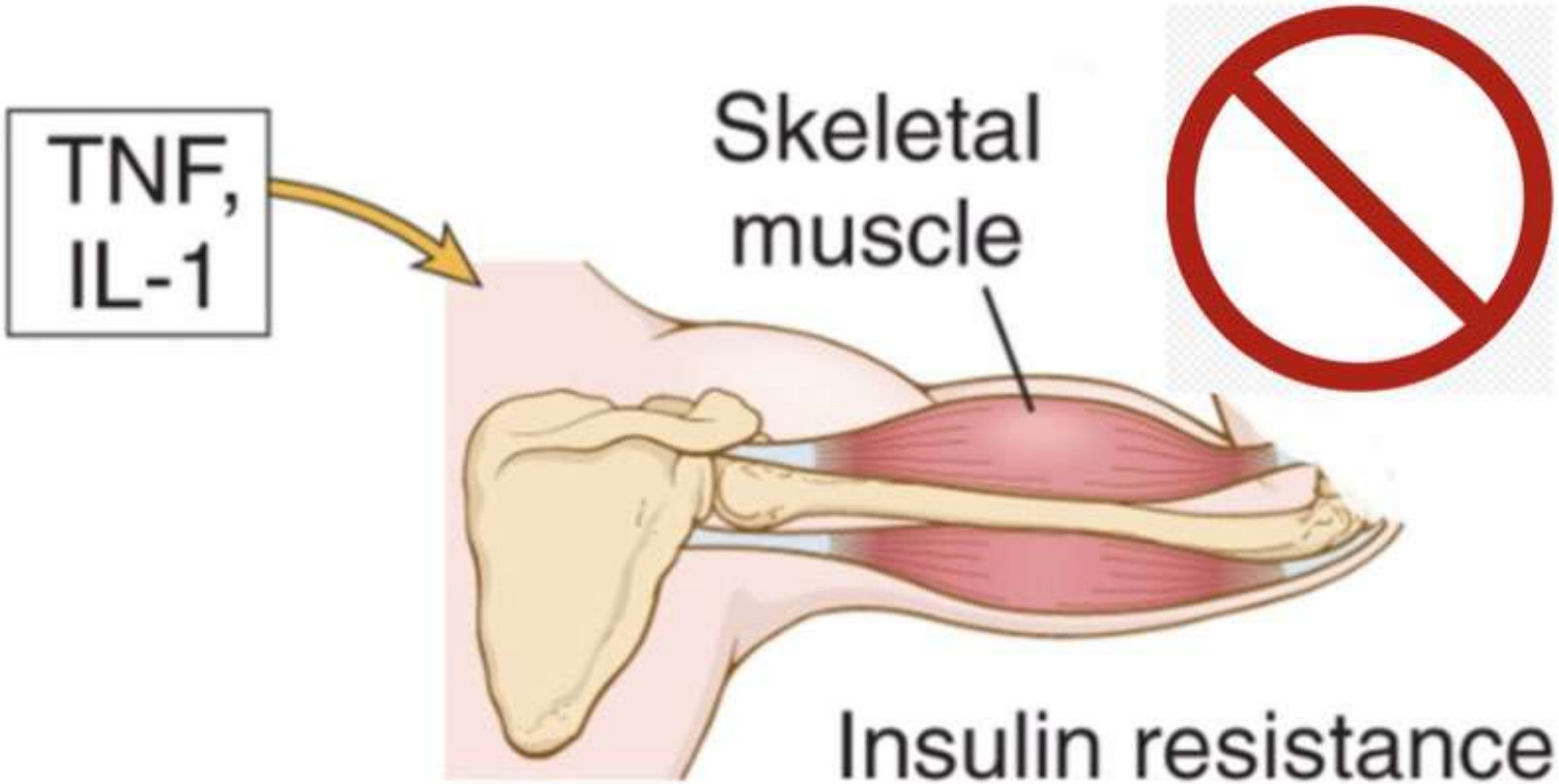
# Multiple tissues




**Malaise, Tiredness and loss of power.**

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# Types of Inflammation

| Feature | Acute | Chronic |
|---|---|---|
| Onset | Fast: minutes or hours | Slow: days |
| Cellular infiltrate | Mainly neutrophils | Monocytes/macrophage and lymphocytes |
| Tissue injury, fibrosis | Usually mild and self-limited | Often severe and progressive |
| Local and systemic signs | Prominent | Less prominent; may be subtle |

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# Acute Inflammation

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# Acute Inflammation

Acute inflammation is **rapid and short-lived** (minutes to days). It is typically beneficial.

**Key features:**

- Triggered by infections, trauma, burns, etc.
- Involves **vasodilation**, increased permeability, and leukocyte (mainly **neutrophil**) infiltration.
- Results in **edema** and localized symptoms.

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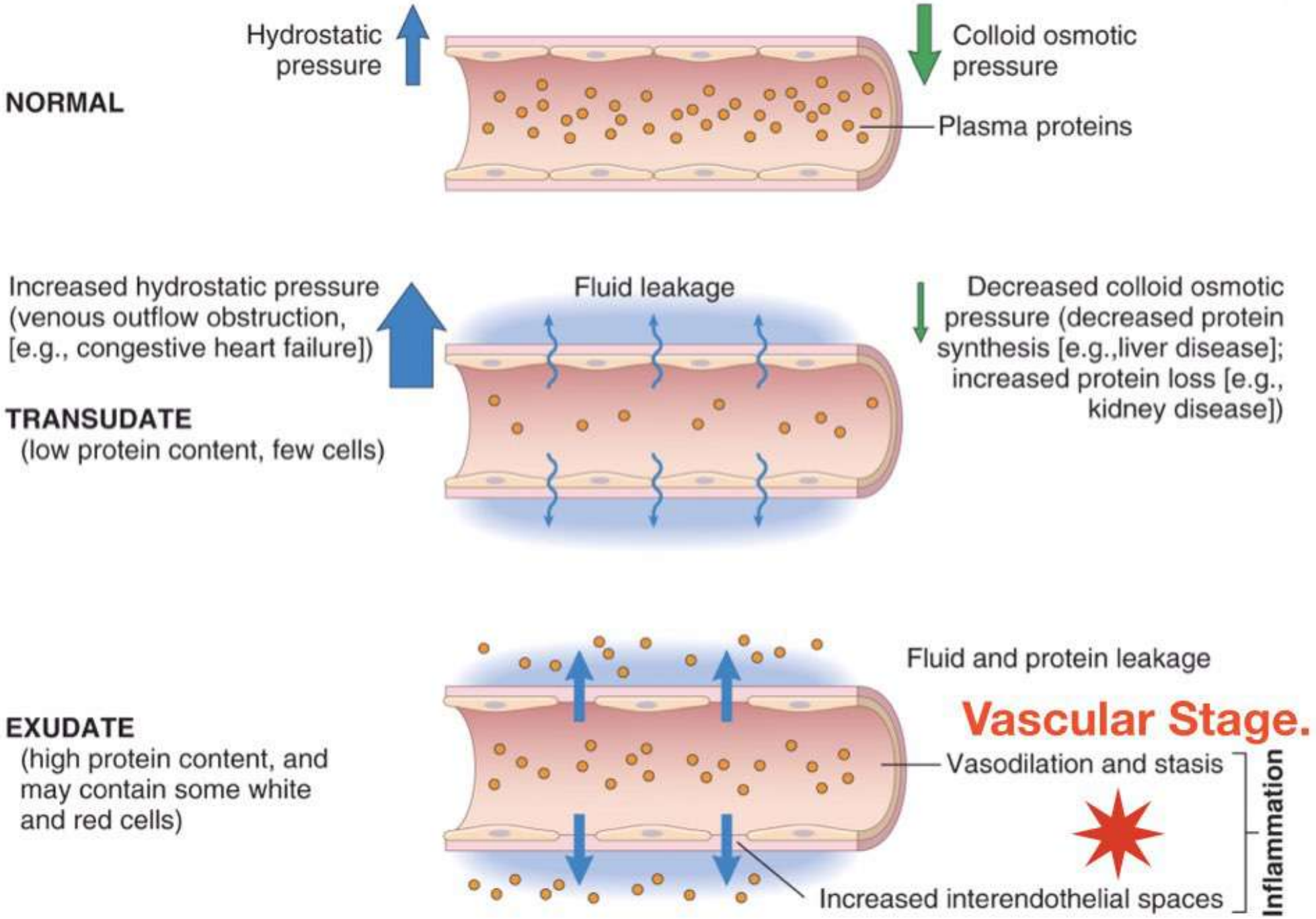


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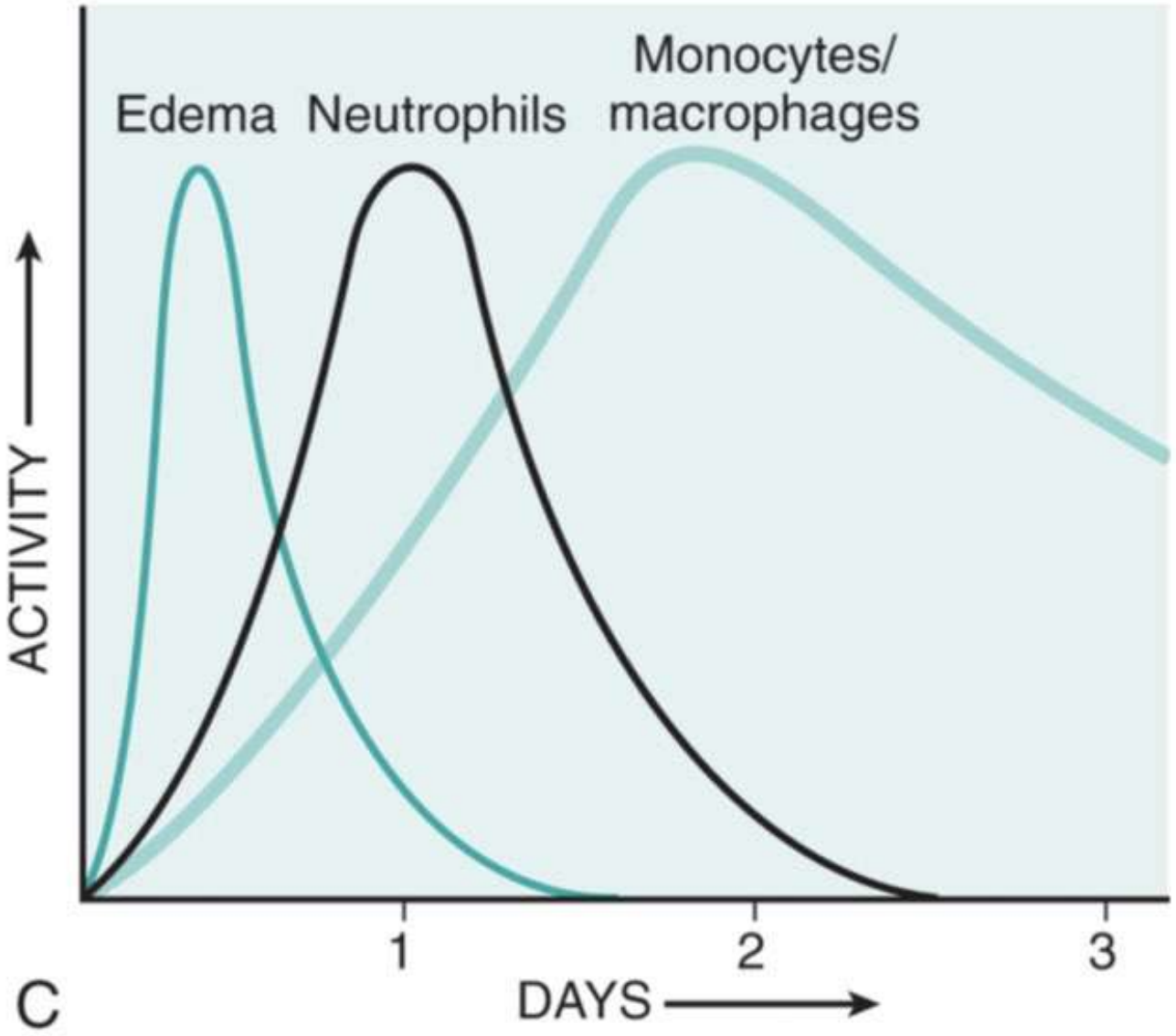
# Nature of leukocyte infiltrates in inflammatory reactions

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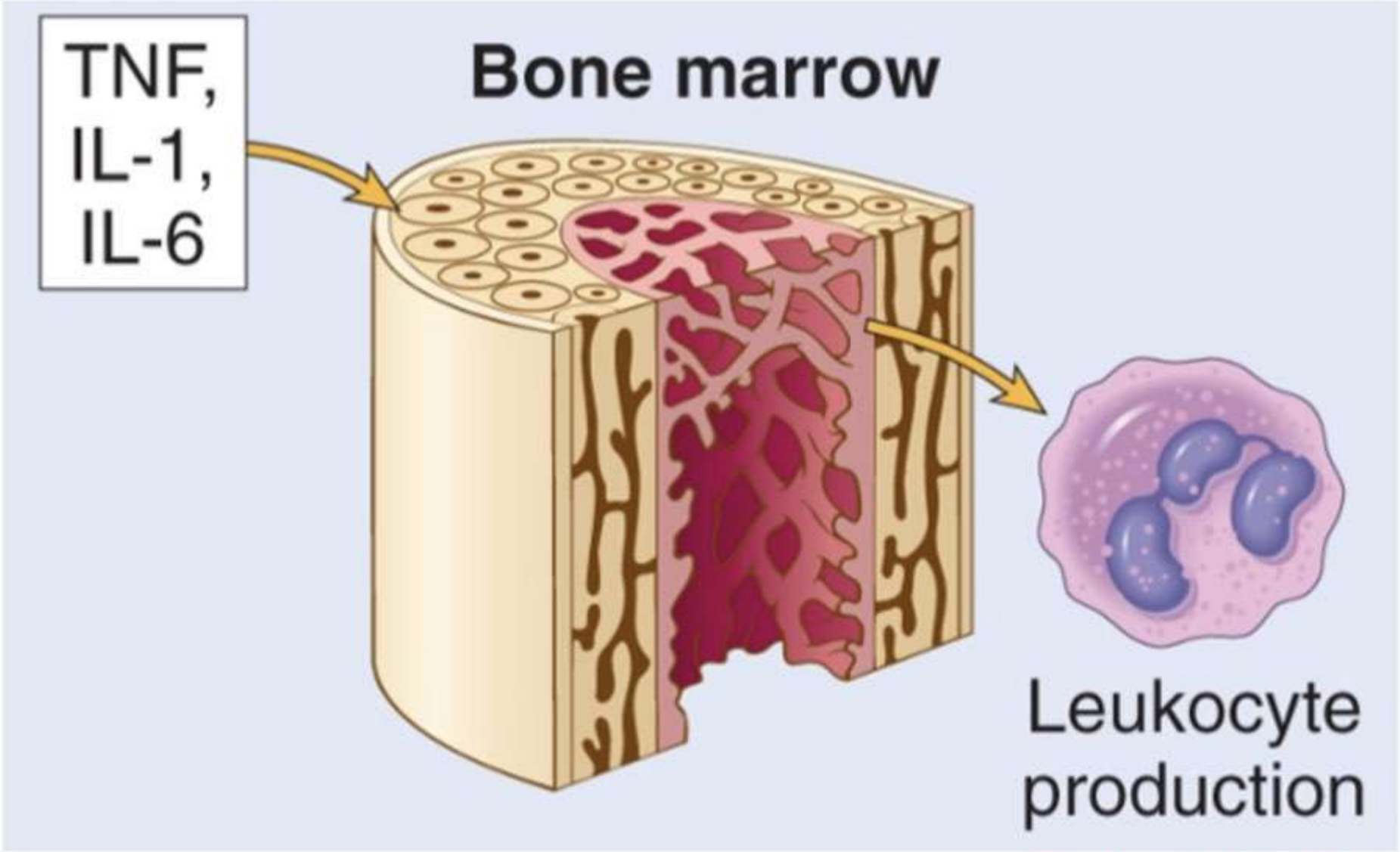
# Cellular Stage.




## Leukocytosis

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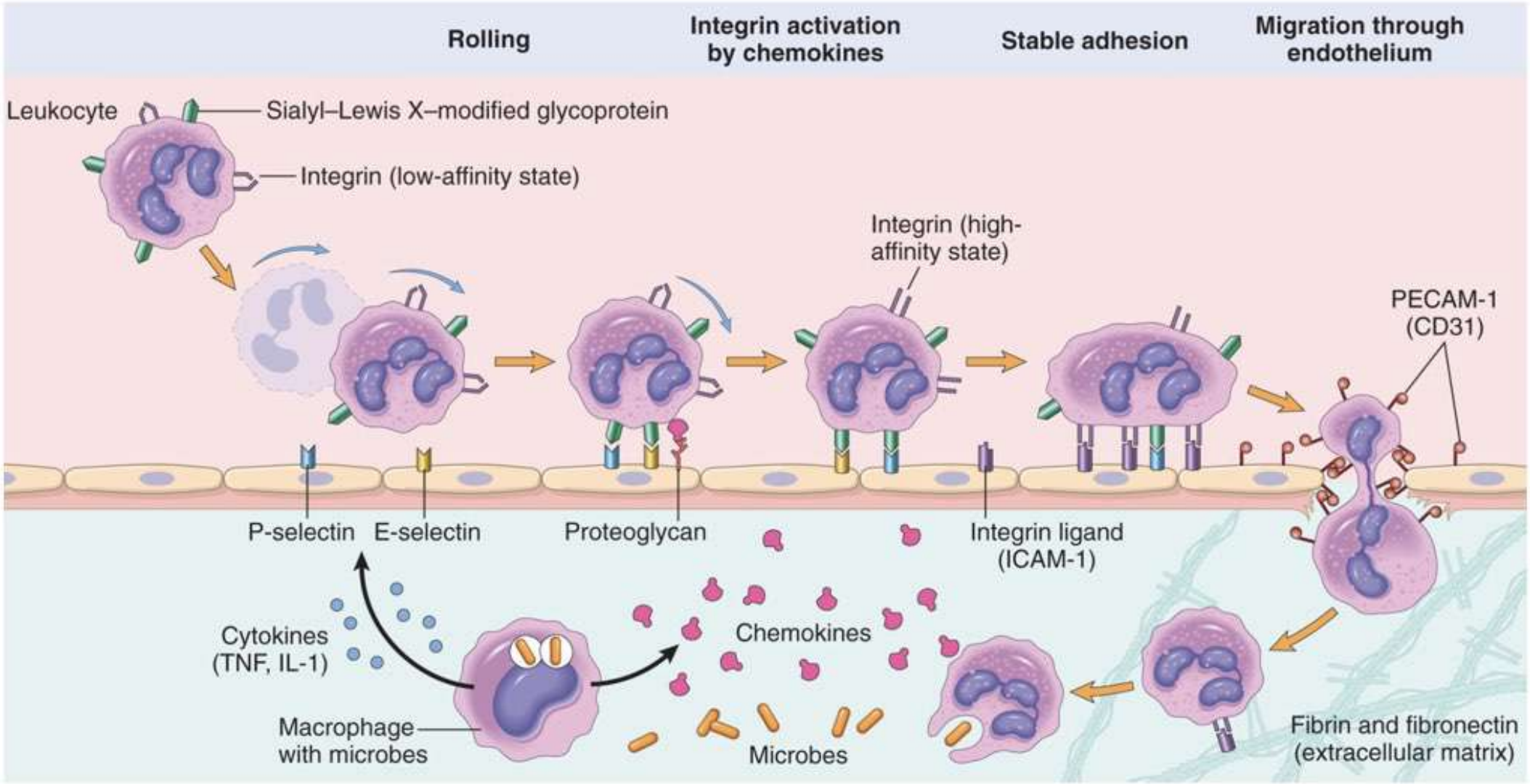
# Cellular Stage.





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# Inflammatory Mediators

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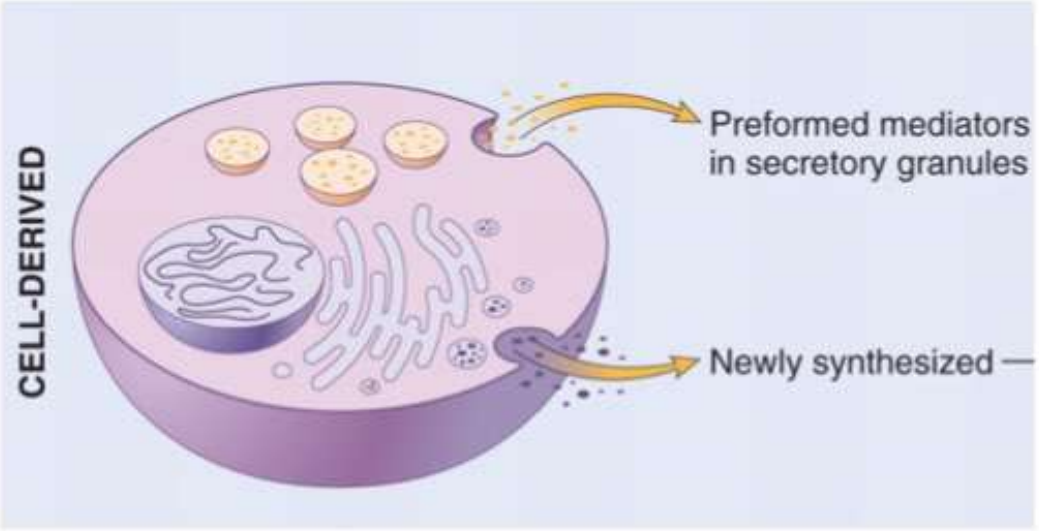
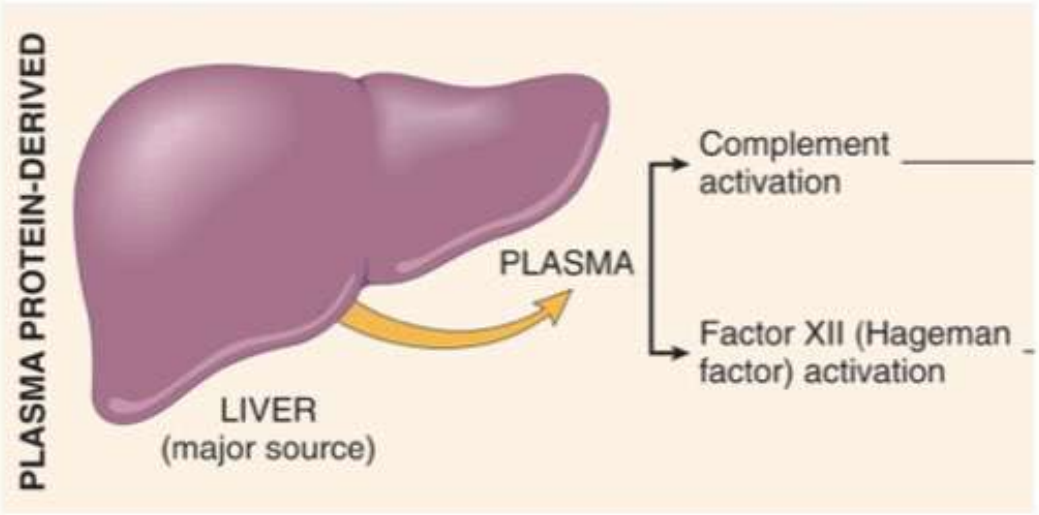
# Chemical Mediators of Inflammation

Chemical mediators regulate every step of inflammation. They can be:

- **Plasma-derived** (e.g., complement, kinins),
- **Cell-derived** (e.g., cytokines, histamine, prostaglandins).

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## Important mediators include:

| Mediator | Source | Effect |
|---|---|---|
| **Histamine** | Mast cells | Vasodilation, ↑ permeability |
| **Prostaglandins** | Leukocytes | Pain, fever, vasodilation |
| **TNF-α, IL-1** | Macrophages | Fever, leukocyte recruitment |
| **Bradykinin** | Plasma proteins | Pain, vasodilation |
| **Complement (C3a, C5a)** | Liver/plasma | Chemotaxis, cell lysis |

These mediators act **quickly** but are tightly regulated to **prevent** excess tissue damage.

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# Fate of Acute Inflammation (**Outcomes**):

- **Resolution**,
- **Abscess (pus)** formation,
- Progression to **chronic** inflammation.

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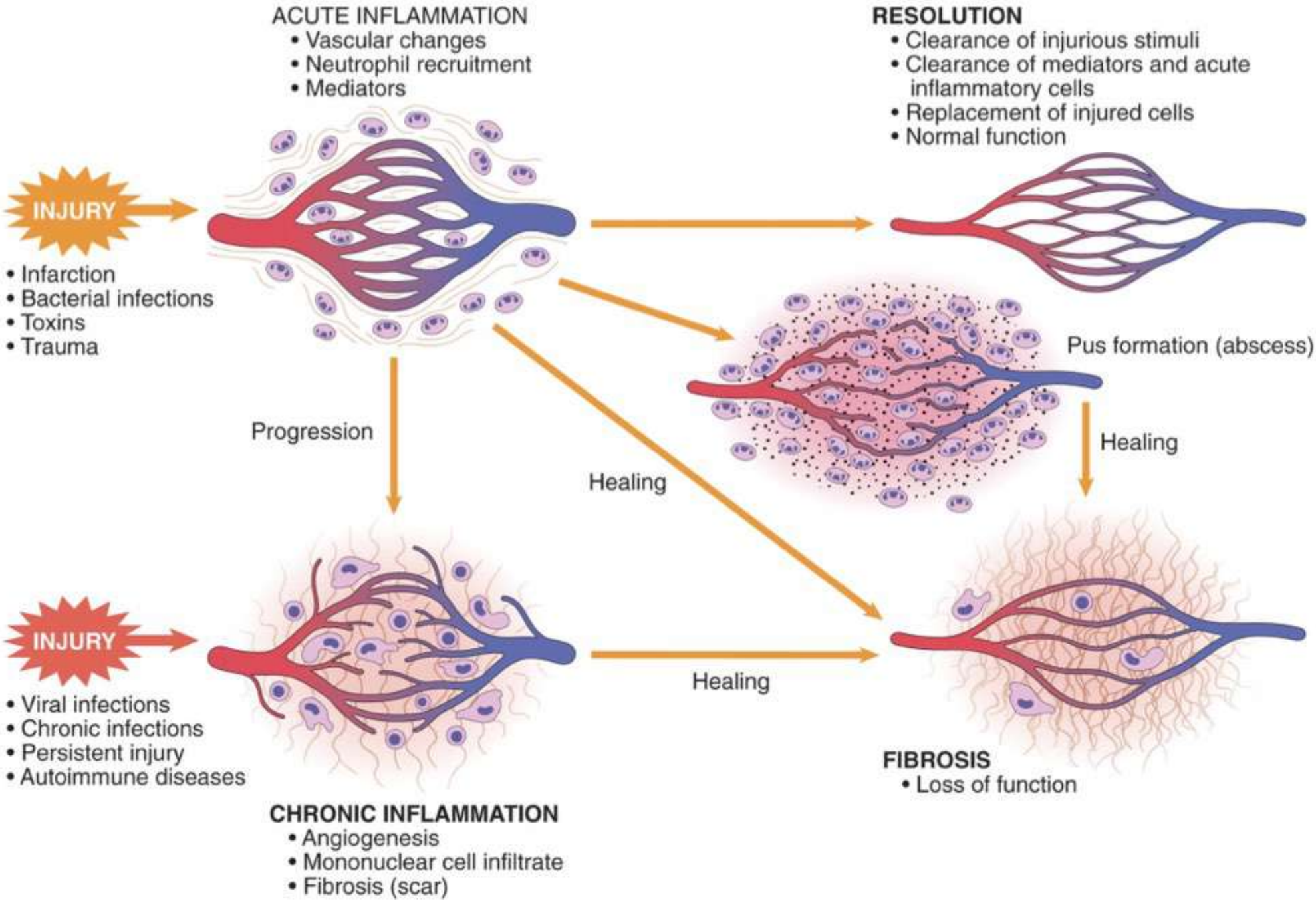


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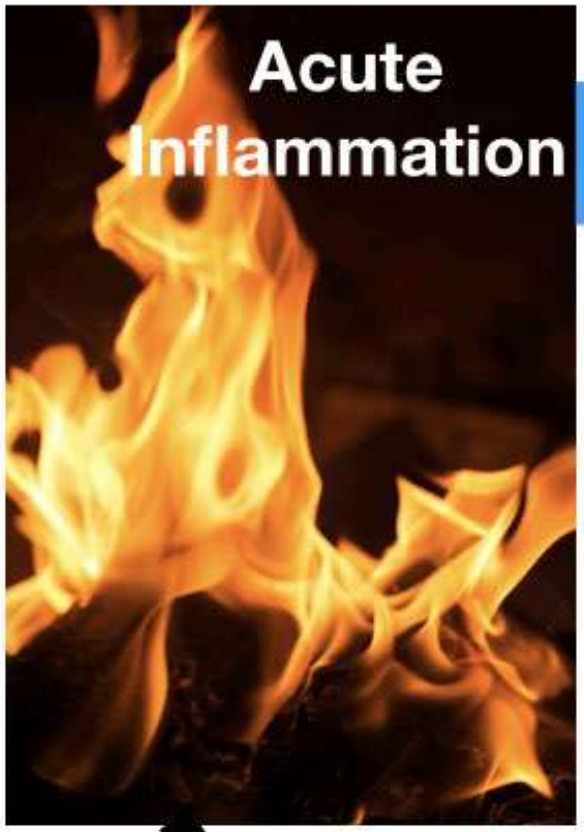


Resolution

Abscess



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# Necrotising stomatitis (NOMA)

is a severe oral infection, starting in the gingiva as acute necrotising ulcerative gingivitis and extending onto and destroying part of the jaw or face.

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# Chronic Pathology (Chronic Inflammation)

Chronic inflammation is **prolonged**, often lasting weeks, months, or even years.

**Causes include:**

- **Persistent** infections (e.g., TB),
- **Autoimmune** diseases (e.g., rheumatoid arthritis),
- Prolonged **exposure** to toxins.

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# Features of chronic inflammation:

- Infiltration by **mononuclear cells** (macrophages, lymphocytes),
- **Tissue destruction** and **fibrosis** (scar formation), and **angiogenesis** (new blood vessels).
- Can lead to **organ dysfunction** and **chronic disease**.

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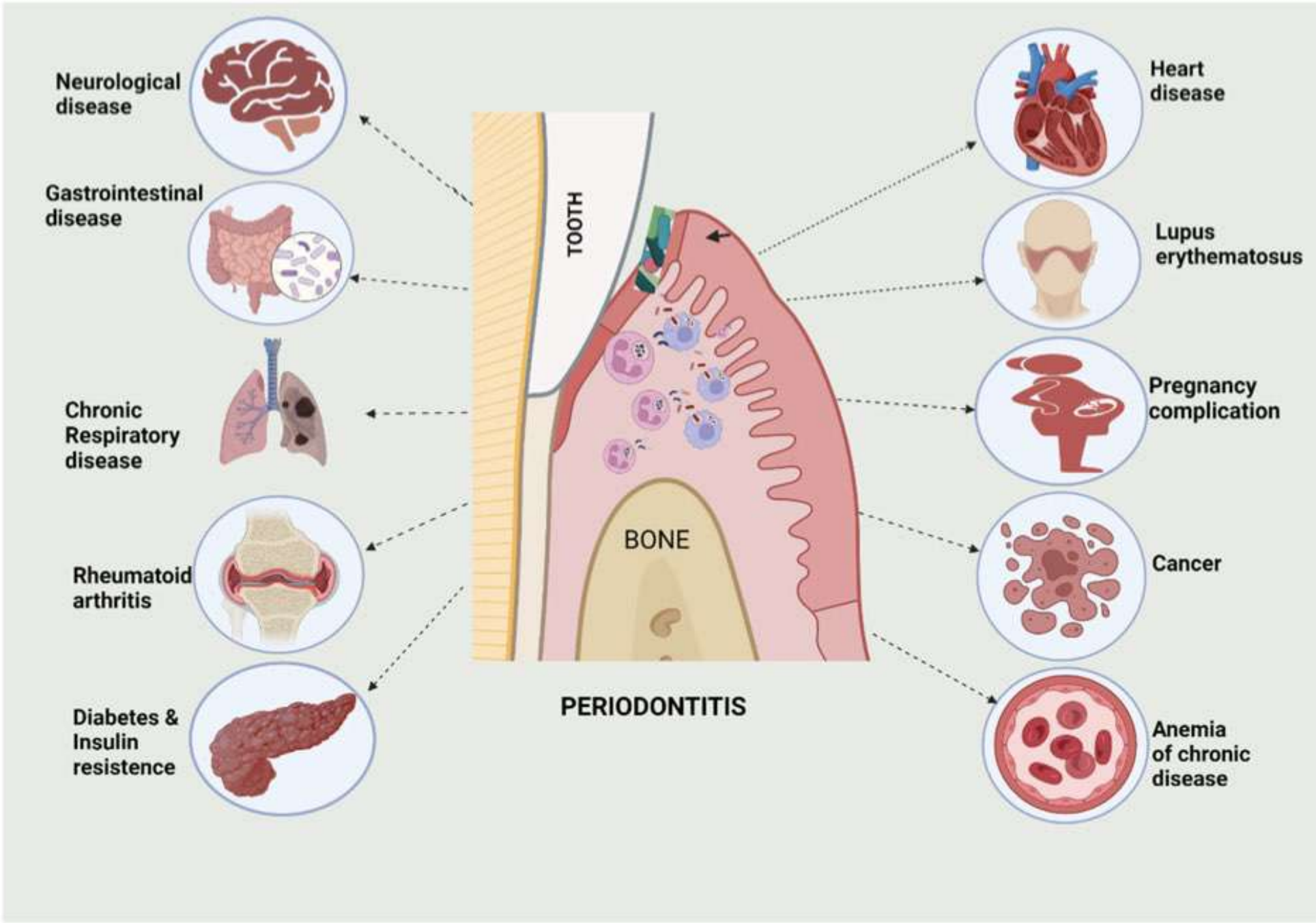


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# Summary

- **Inflammation** is essential but must be controlled.
- **Acute inflammation** is protective and self-limiting.
- **Chronic inflammation** contributes to many diseases.
- **Chemical mediators** orchestrate the entire process.

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