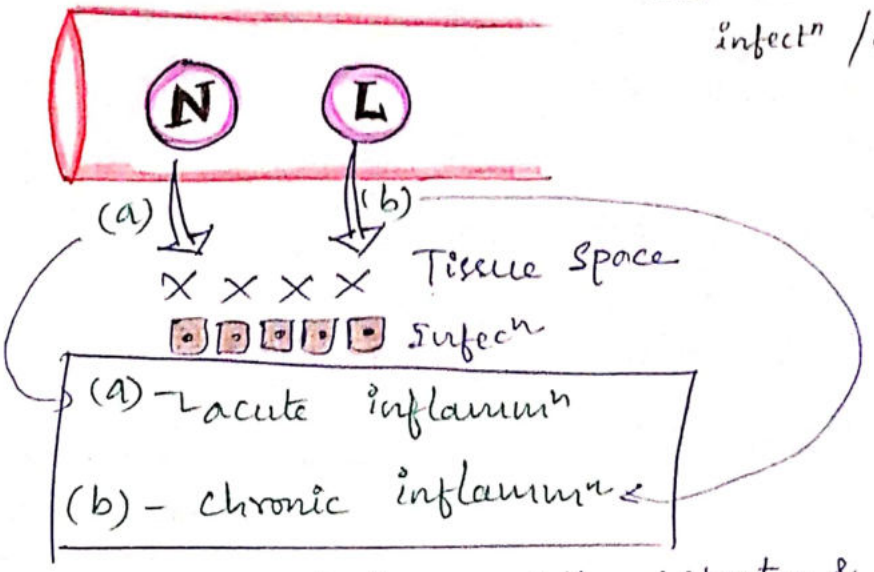
Inflammation → is the response of Vascularized Tissues that allows N, L, molecules of Immunity to leave the blood & enter the site of infect^n / cell death



* Inflamm^n - allows → inflamm. cells (Neutro & lympho).
→ plasma proteins
→ fluid → edema
to exit blood V/s & enter Interstitial Space.

* ② types - Acute (Neutro) & Chronic (lympho-cytes).

I) ~~ACUTE INFLAMMATION~~

(2) Characteristics of Acute Inflammat^n

- edema
  - ⇓
  - due to fluid leakage from blood V/s.
- Neutrophils
  - ⇓
  - Key Inflamm. cells.

Infection
↓
To eliminate pathogen

Tissue Necrosis.
↓
To clear debris.
↓
So that Healing can take place.

Why WBC ↑ in MI?
So that Neutrophils can clear debris of death myocytes

Characteristics of AI

(i) immediate response ⇒ after infect fluid starts building instantly & Neutrophils ↑ c in 24hrs.

(ii) Has limited specificity (due to immediate response)

(iii) part/component of Innate Immunity.

Note: what does innate immunity comprise of?

Comprises of (i) epithelium that covers the body

(ii) Mucous secreted by various cells

(iii) Complement system – (series of inactive protein present in plasma which can be ⊕ to produce immune response)

(iv) Cells.

- Mast cells
- Neutrophils.
- Macrophages.
- Eosino & Basophils.

Receptors.

* present on cells of innate immunity (macrophages & dendritic cells)

* Able to recognize PAMPs (Pathogen Associated Molecular Patterns).

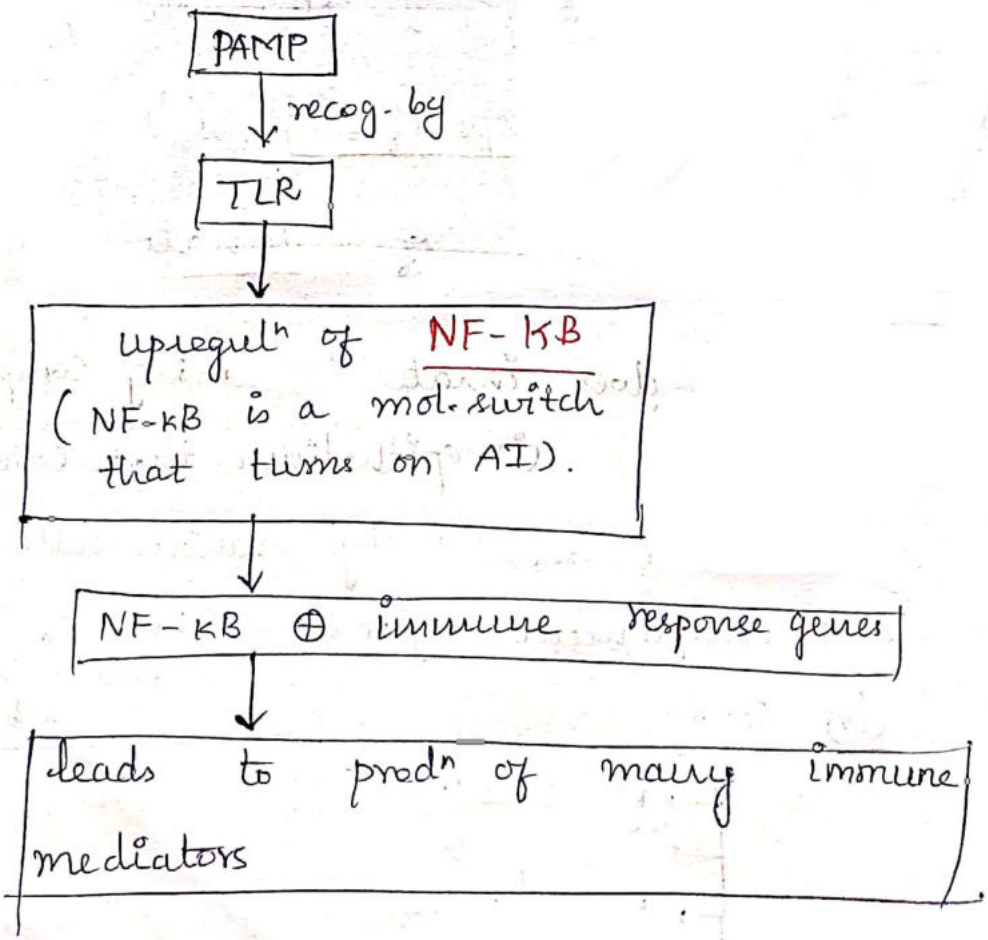
Common for a grp of microbes.

→ recognized by TLRs. → (AI).

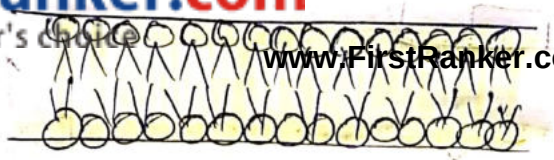
eg: CD14

* TLR of macrophage.

* recognizes the PAMP called LPS. (Lipo-Poly Saccharide –) present on outer memb. of GN bacteria



Note:
* TLRs. are also present in cells of Adaptive immunity
* So TLRs also involved in Chronic Inflamm.



Phospholipid B.L.

P.L.A2

Arachidonic ac

cox. pathways

5 - LOX pathway

PGs are produced

Leukotrienes (LTs) are produced.

* PG I_2, PG D_2, PG E_2 mediate (i) Vasodilation (arterioles).

(ii) ↑ vascular permeability (post. capillary venule)

* PG E_2 also mediates fever & pain.

* LTB_4 attracts & ⊕ Neutrophils.

* LTC_4 | LTD_4 | LTE_4 mediate

(i) Vasoconstricⁿ

(ii) Bronchospasm

(iii) ↑ vascular permeability

SM Contracⁿ

pericyte contracⁿ

Note :

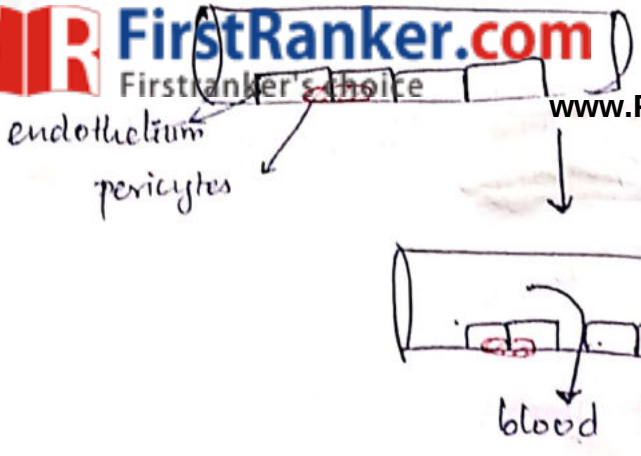
④ chemicals which attracts & ⊕ Neutrophils.

(i) LTB_4

(ii) (C_5a - Complement)

(iii) IL 8

(iv) bacterial products



blood leaks out.

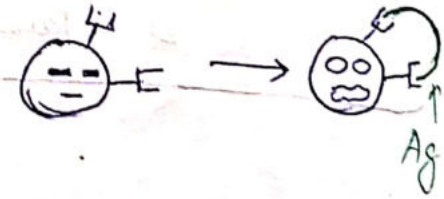
↑ Vascular permeability due to contract of pericyte.

(iii) Mast cells

* widely distributed along conn. tissues.

③ ways of activatn of mast cells :

- (i) Tissue Trauma
- C_3a & C_5a (Complement proteins)
- Cross linking of cell surface IgE by Ag.



② Responses
- Immediate
- Delayed / Late

Immediate Response :

- → Release of preformed Histamine Granules.
- → Mediates Vasodilation of arterioles & ↑ vascular permeability. (at post capillary venule).

Why this happens? in order to maintain AI

How? * Production of Arachidonic a metabolites particularly LTs (LeukoTrienes).

(iv) Complement

* proinflammatory Serum proteins
* which assist/complement inflamm".
* ⊙late as inactive precursors.

③ pathways of ⊕ of Complement

Classical pathway
IgG/IgM ⊕ Ag
⊕ C_1
⊕ Complement

Mnemonic: GM produces Classic Cards

Alternative pathway
Microbial products
⊕
⊕ Complement

Mannose binding lectin pathway (MBL)
MBL + mannose
⊕ Complement

mannose – present in microbes.

RESULT: ③ pathways finally activates the following

(i) C_3 convertase ⊕
$C_3 \longrightarrow C_3a + C_3b$

(ii) C_5 convertase ⊕
$C_5 \longrightarrow C_5a + C_5b$ ⇒ Complexes with C_6 & C_9

Formation of MAC (Memb. Attack Complex).

$C_5b + C_6 + C_9 \longrightarrow$ MAC

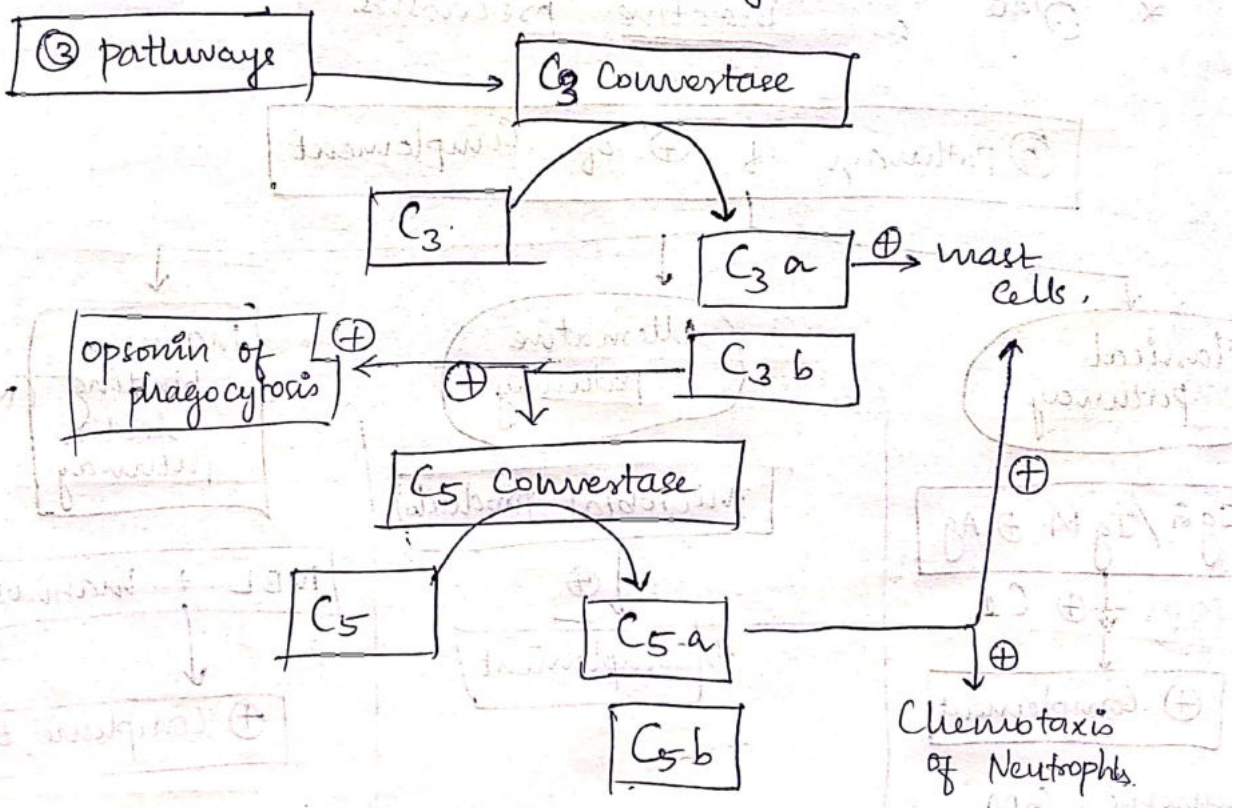
↓

Hole in memb. of pathogen

↓

lysis.

Summary of Complement pathway



- ✓ C_3a & C_5a $\xrightarrow{\oplus}$ mast cell.
- ✓ C_5a → chemotaxis of Neutrophils
- ✓ C_3b → opsonin for phagocytosis.
- ✓ MAC → holes in memb. of bacteria...

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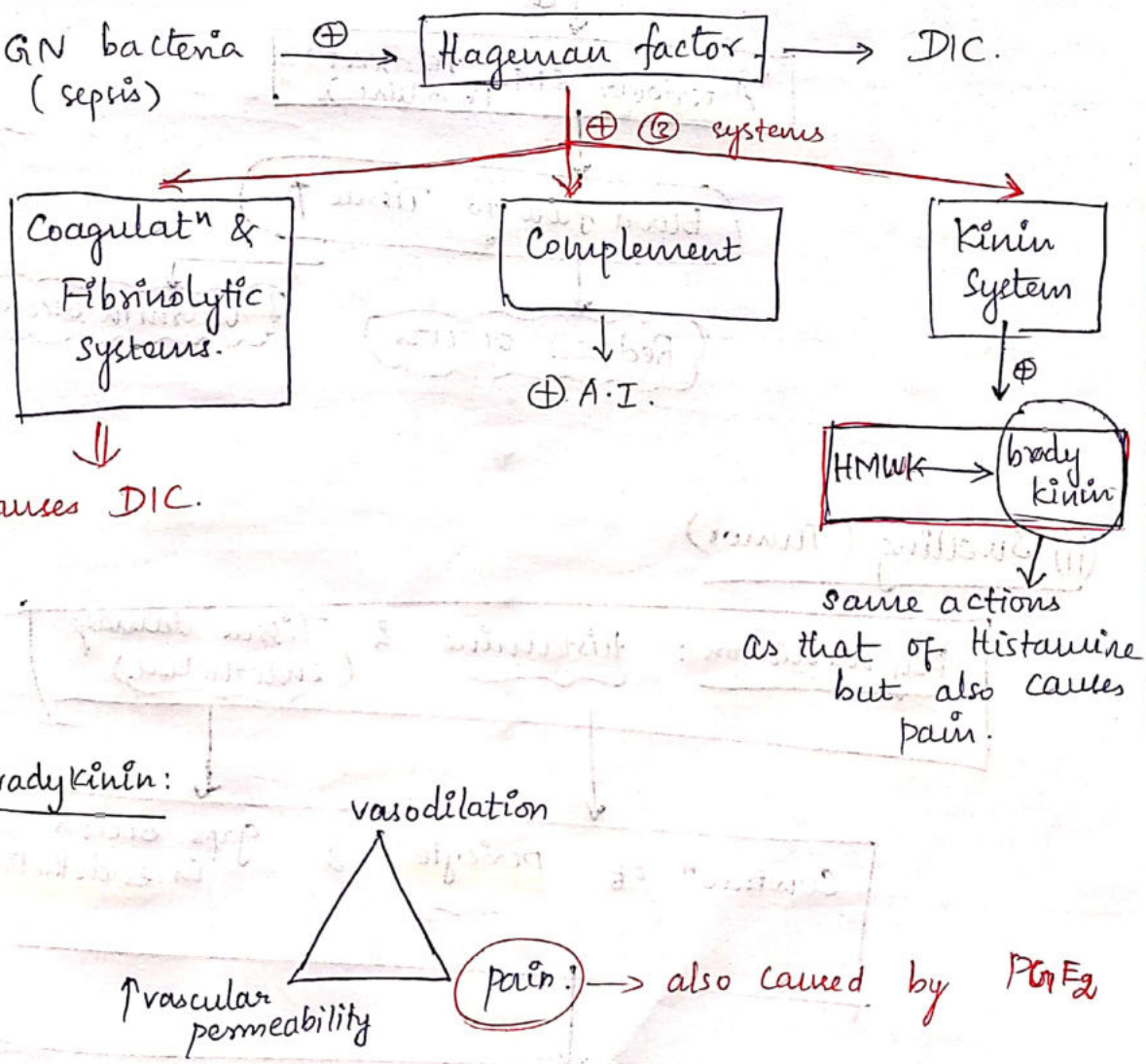
opsonin → usually phagocytosis is random (or) less specific but opsonin can bind to bacteria & make it more tastier for Macrophages.

* It is an inactive proenzyme protein produced in Liver.

* Activated ⊕ by exposure to Subendothelial (or) Tissue Collagen

How Hageman factor ⊕ by Severe GN sepsis Causes DIC.?



Bradykinin:

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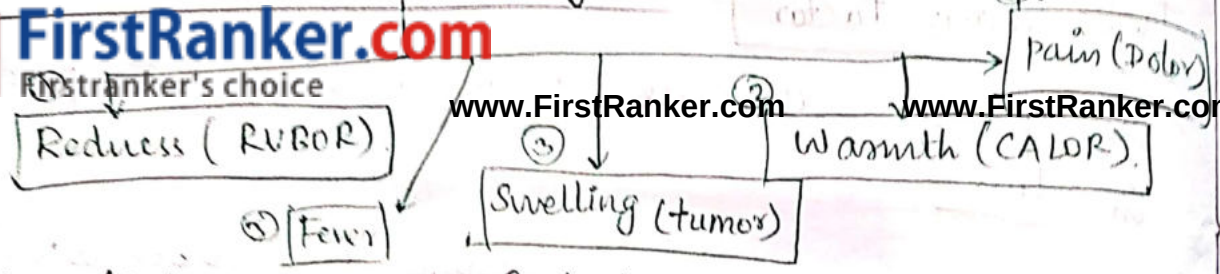
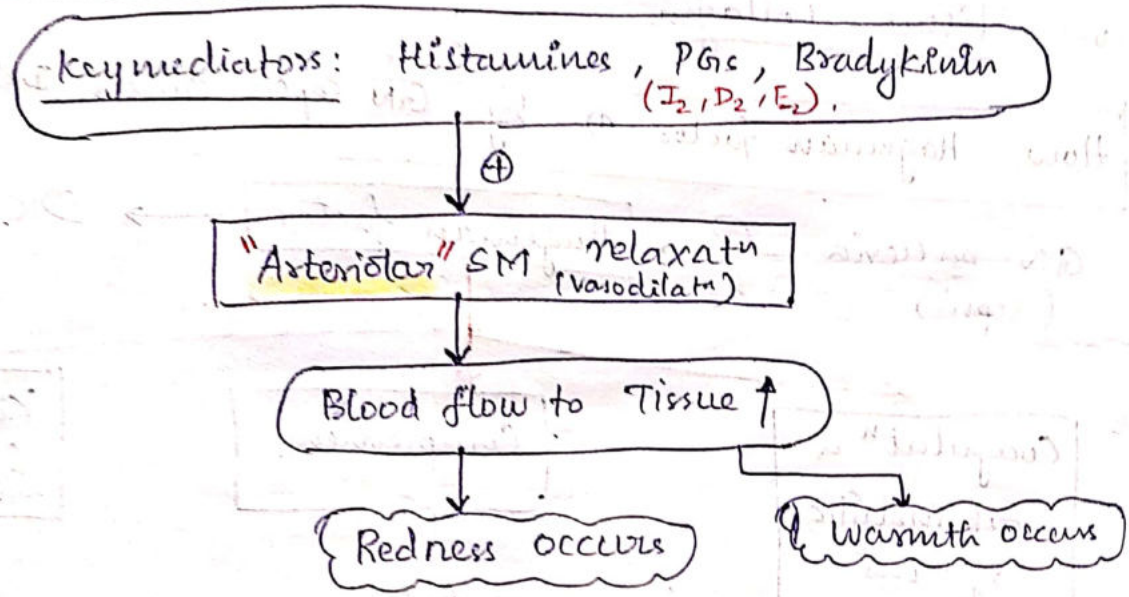
all the (5) mediators of AI

(i) TLR
(ii) Arachidonic ā
(iii) Mast cell
(iv) Complement
(v) ~~Hageman Factor~~

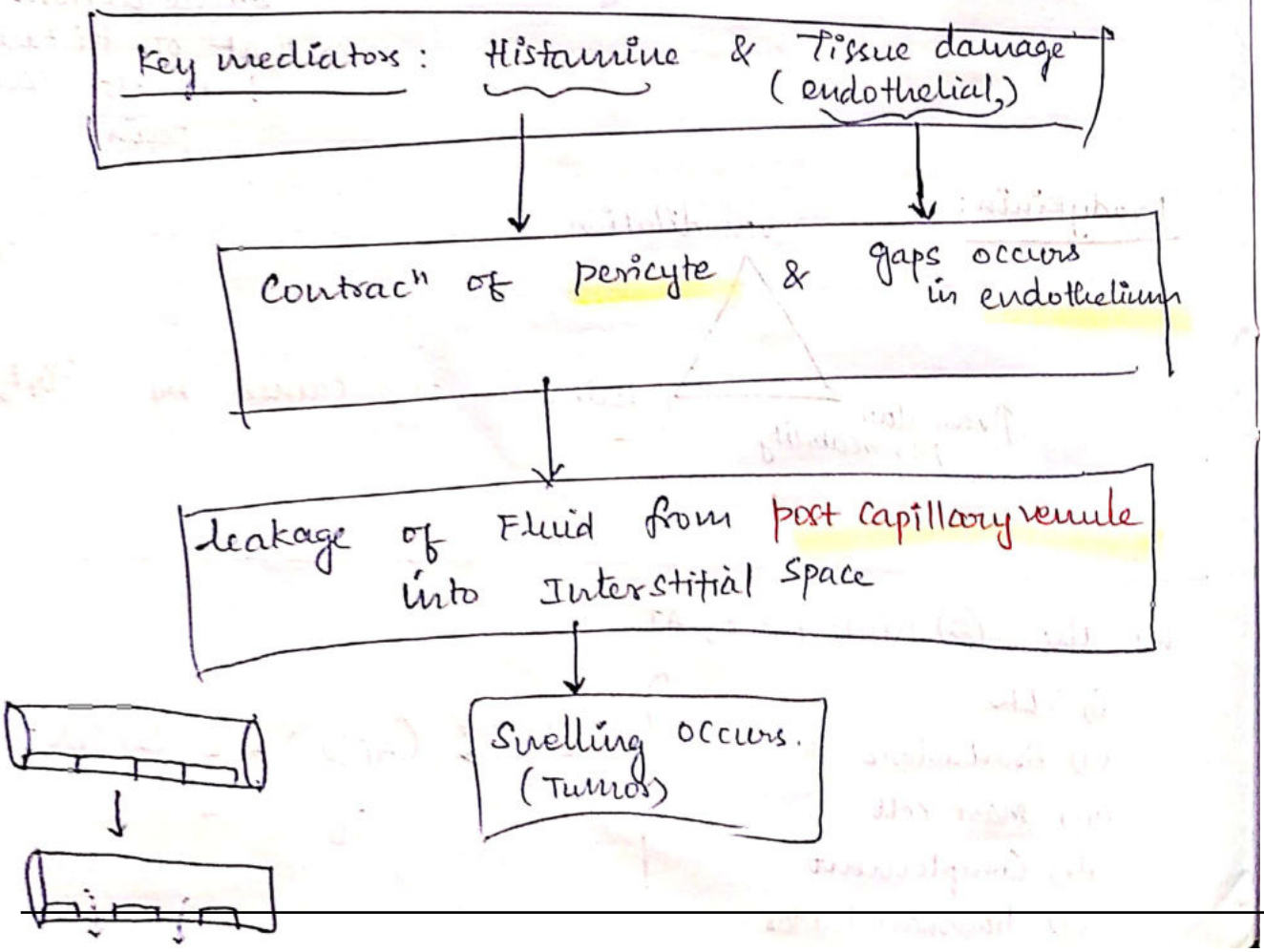
contribute to → CARDINAL SIGNS of AI



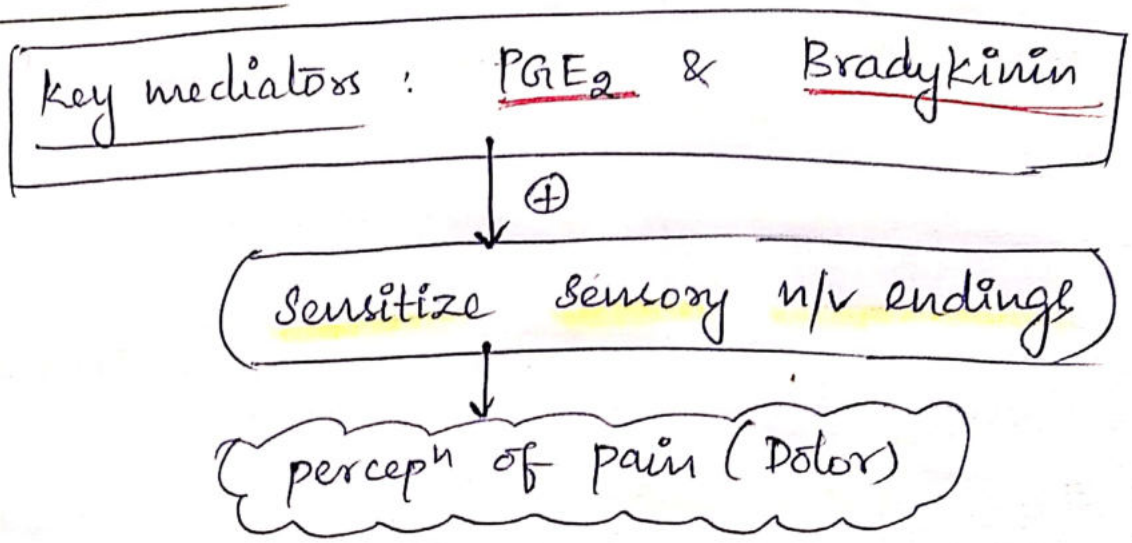
(i) Redness/Rubor: & Warmth (Calor)



(ii) Swelling (Tumor)



(iii) Pain (Dolor)



(iv) Fever :