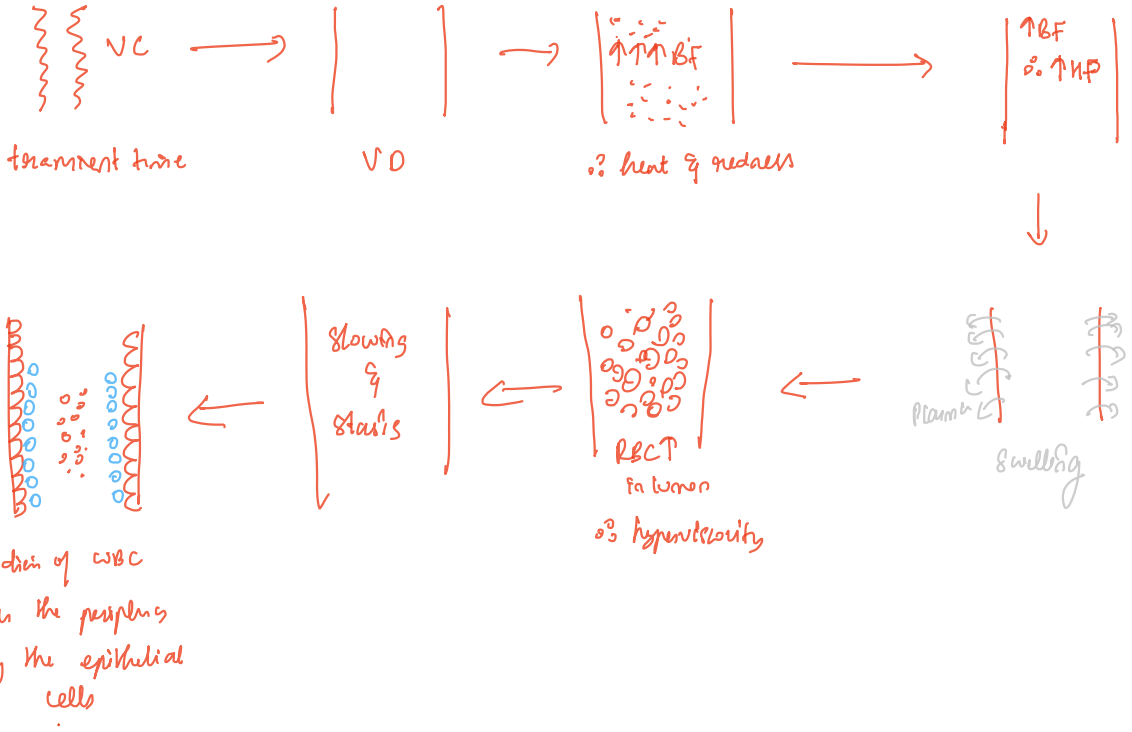
Inflammation

↳ defined as the local response of living mammalian tissues to injury from any agent.

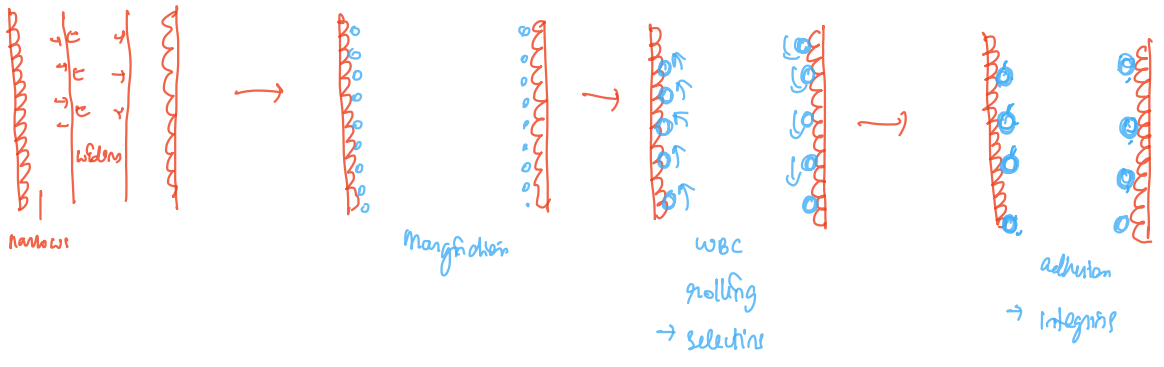
Acute Inflammation
- vascular events
- cellular events
- Chemical mediators.

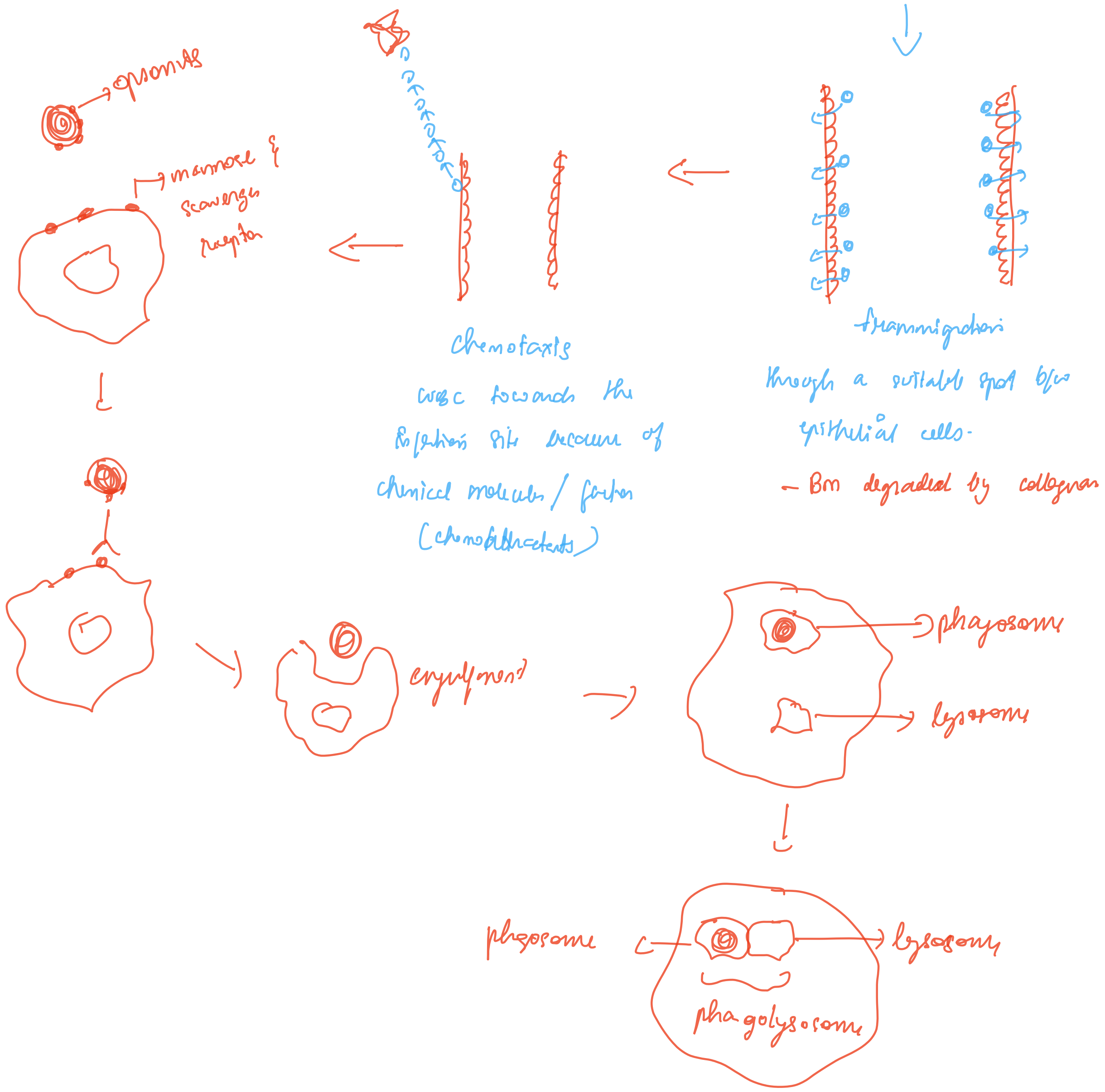
vascular events

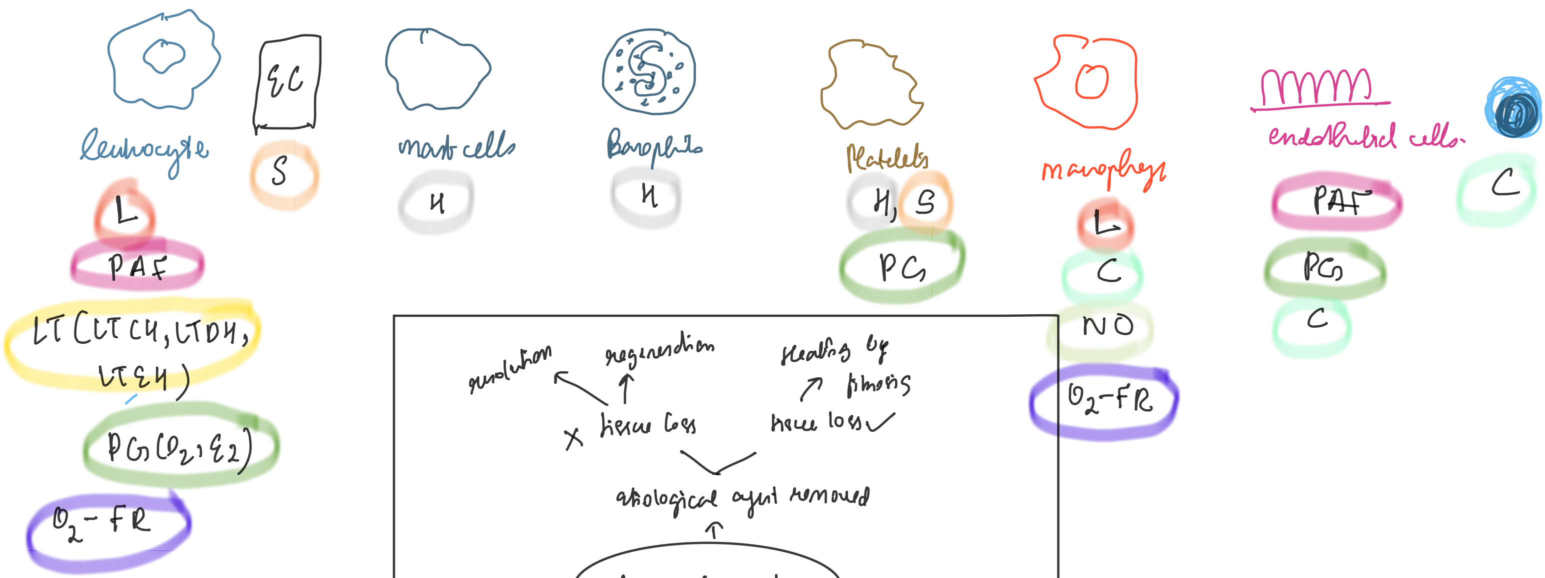
1) vasodilation

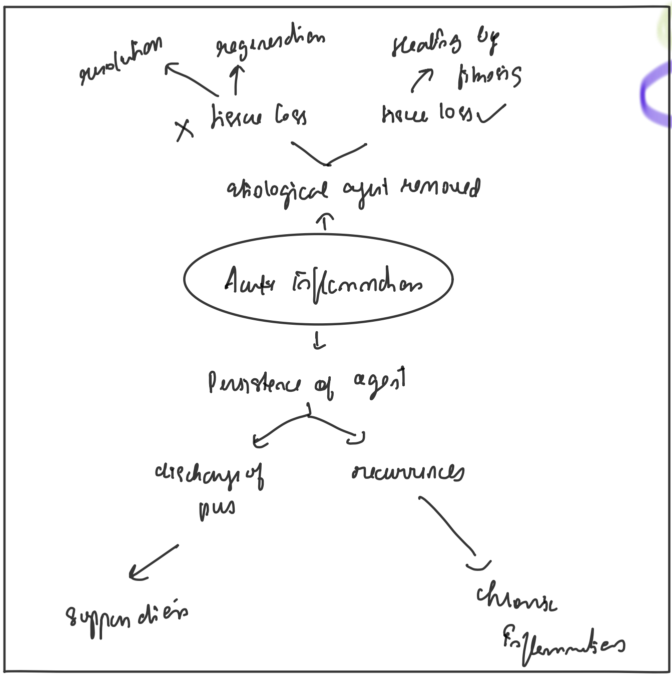


Cellular events




opsonins
mannose & scavenger receptor
chemotaxis
WBC towards the infection site because of chemical molecules / factors (chemoattractants)
transmigration
through a suitable spot b/w epithelial cells.
- BM degraded by collagenase
engulfment
phagosome
lysosome
phagosome
lysosome
phagolysosome


leukocyte
L
PAF
LT (LTC4, LTD4, LTE4)
PG (D_2, E_2)
O_2-FR
EC
S
mast cells
H
Basophils
H
Platelets
H, S
PG
macrophage
L
C
NO
O_2-FR
endothelial cells
PAF
PG
C
C


resolution
regeneration
healing by fibrosis
x tissue loss
tissue loss ✓
etiological agent removed
Acute inflammation
Persistence of agent
discharge of pus
recurrences
suppuration
chronic inflammation

Wound repair

Repair mechanisms

Process of granulation tissue formation &

1) Bleeding phase

- stops after 4-6 hrs
- following a clot formⁿ (VC & platelet aggregation)

2) Inflammatory phase

- After blood clotting → fibrin & fibronectin remains in tissues
  ↓
  acts as substrate for the adhesion of various inflammatory cells.
- Inflammatory response start (vascular, cellular by chemical mediators)
  ↓
  Initially, infiltration by PMNs & fluid exudates within few hours
  ↓
  influx of phagocytic cells (mφ, monocytes)
- Dead & dying cells, necrotic debris, fibrin mesh & clot all cleared off by combination of proteolytic & autolytic enzymes (liberated from neutrophils & dead cells)

3) Proliferation phase

- ✗ generation of repair material that form the initial granulation tissue
- ✗ Begins with 1-2 days after injury.
- ✓ 2 phases:
  * Angiogenesis
  * Fibrogenesis

i) Angiogenesis

- form^{n} of new blood vessels at the site of injury
- By prolif^{n} of endothelial cells from the margins of severed blood vessels
- Initially, proliferating endothelial cells are solid buds but then → lumen → carries blood
- Newly formed BV are leaky, accounting for the edematous appearance of new granulation tissue
- The process of angiogenesis, stimulated with proteolytic destruction of BM factors for ACs
  - * VEGF - vascular endothelial GF
  - * PDGF → Platelet derived GF
  - * Transforming GF-β (TGF-β)
  - * basic fibroblast GF (b-FGF)

ii) Fibrogenesis

- * After angiogenesis, they are surrounded by amorphous ground substance / matrix
- * New fibroblasts appears. These cells are halfway in btw regular fibroblast & Smooth muscle cells. ∴ called as myofibroblasts.
- * Around 5th day, of healing, type-III collagen fibrils start forming
- * myofibroblasts have receptors on their surfaces for a protein called fibronectin, which helps link collagen fibers together.
- * As wound matures, weaker type-III collagen is broken down by an enzyme called collagenase
- * Replaced by stronger type-I collagen, which no. of active fibroblast & new BV decr

4) Remodelling phase

- 2-3 weeks following injury
- Replacement of collagen & its associated extracellular matrix
- type I collagen fibrils that have replaced ones have more cross links & greater tensile strength.
- Results in inactive looking scar ⇒ Scarification

Transfusion rxn

Immunologic

Non-immunologic

1) Hemolytic transfusion rxn

a) Intravascular hemolysis - ABO incobatable

b) Extravascular " - Rh incompatibility.

2) Transfusion - related acute lung injury [TRALI]

- transfusion of donor plasma containing high level of anti-HLA Abs

3) Other allergic

→ Febrile reactions

→ Anaphylactic shock

→ Allergic

⇒ Transfusion related graft v/s host disease.

B) Non-immune transfusion:

* Circulatory overload - pulmonary congestion & acute heart failure

* Massive transfusion.

* Transmission of infection

* Air embolism

* Thrombophlebitis -

* Transfusion hemosiderosis