

## V.E. INFLAMMATION & REPAIR

### E.a. INFLAMMATORY RESPONSE TP VE-1

#### E.a.A. FUNCTIONS

- 1) Neutralize or destroy pathogen/antigen
- 2) Contain the damage
- 3) Alert individual to problem
- 4) Prepare injured area for healing

#### E.a.B. EF'S (T 2.1, p 38)

- 1) Ischemic tissue damage
  - 2) Trauma
  - 3) Infection
  - 4) Immune response
  - 5) Vascular lesions
- } => injured capillaries & other cellular injury => inflammatory mediators => vasodilation => <inflammation>

### E.a. ACUTE INFLAMMATION

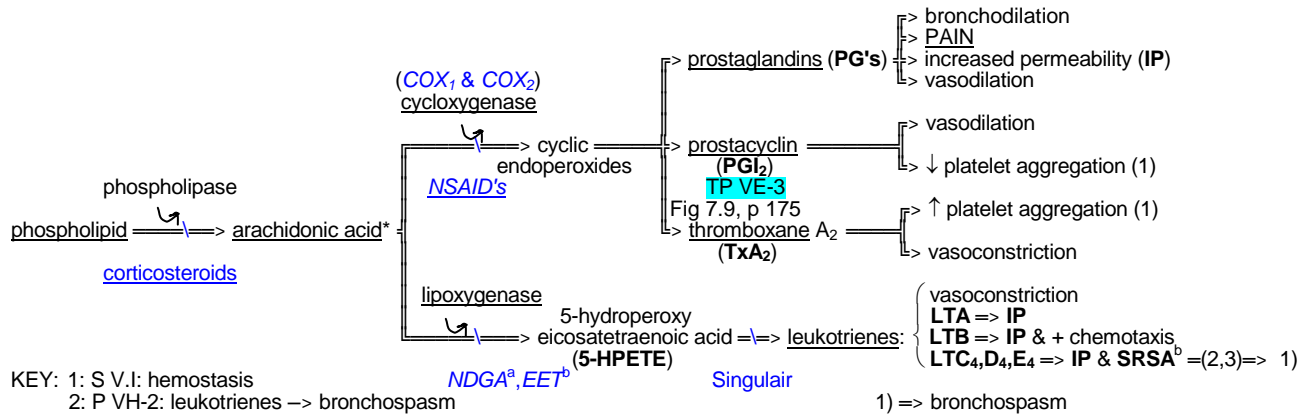
#### E.b.A. CARDINAL SIGNS & SYMPTOMS

- 1) Swelling (*tumor*)
- 2) Heat (*calor*) {L heat}
- 3) Arrested mobility ~ *functio laesa* (loss of function)
- 4) Redness (*ruber*)
- 5) Pain (*dolor*) {L sorrow}

#### E.b.B. INFLAMMATORY MEDIATORS [substances that cause inflammation] (see CPs VE-2 & VE-3 for details)

##### E.b.B.a. CELL-DERIVED MEDIATORS

- 1) Histamine--released by platelets, basophils (mast cells) by degranulation; causes increased permeability & vasodilation
- 2) Leukotrienes, prostacyclin, thromboxane & prostaglandins--derived from cell membrane of injured cells & degranulation of mast cells, microphages (**PMNL**) & macrophages (monocytes) (Fs 2.14, p 39 & 2.15, p 40)



#### TP VE-2

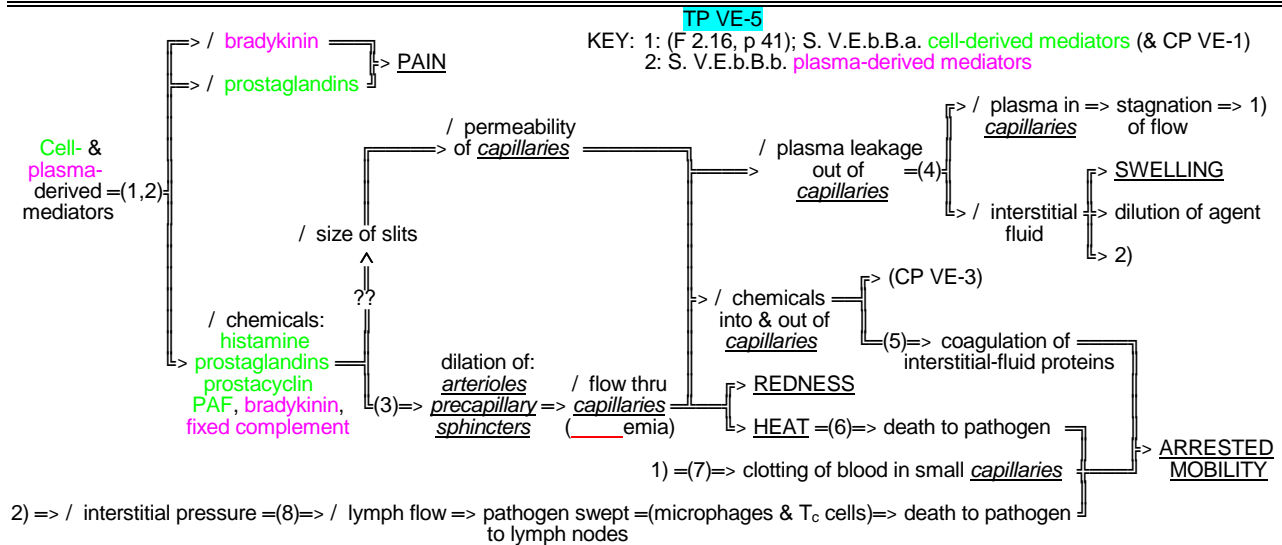
Critical Path VE-1. Path from Phospholipids in the Cell Membrane to Prostaglandins & Leukotrienes (source unknown)

- 3) Platelet activating factor (PAF)--released from mast cells by degranulation & other cells (membrane)
  - a. Platelet aggregation
  - b. + chemotaxis of eosinophils & neutrophils (18, p 347)
  - c. Bronchospasm (contraction of smooth muscles on bronchioles)
- 4) Other products from microphages, macrophages, other leukocyte products & destroyed cells
  - a. Enzymes (kininogenase, phospholipase A<sub>2</sub>)
  - b. Lysosomal enzymes (CF IIIA-7): cell swelling; (CP IIIA-9): cell rupture
  - c. K<sup>+</sup> (CP IIIA-9)
  - d. Cytokines (S V.F & S V.G.)

### E.b.B.b. PLASMA-DERIVED MEDIATORS (plasma proteins)

- 1) Kinins (bradykinin)
- 2) Fixed Complement (CP VG-3: complement cascade)
- 3) Clotting factors (S V.I: hemostasis)
- 4) Fibrinolytic factors (S V.I: hemostasis)

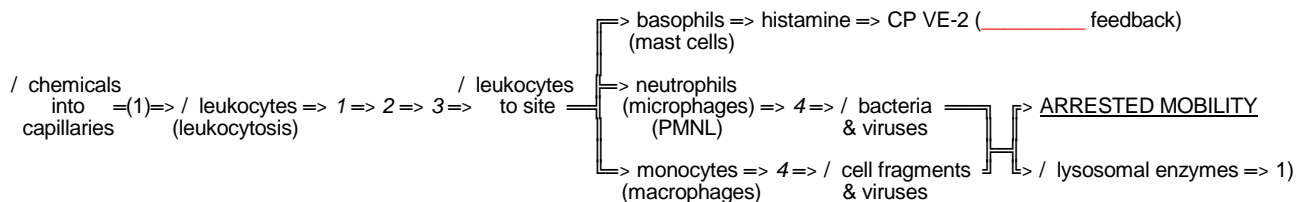
### E.b.C. VASCULAR & LEUKOCYTIC RESPONSES



- KEY: 3: F VD-2: arteriovenous shunt & precapillary sphincter  
4: CF IVA-2: / interstitial plasma proteins -> / interstitial osmotic pressure  
5: CP IIIA-9: etiology of coagulation necrosis as a result of hypoxia  
6: CP VD-4: purpose of fever  
7: S. V.I: hemostasis  
8: F IVA-3: action of lymph capillaries

#### TP VE-4

Critical Path VE-2. Vascular Responses to Cellular Injury, Their Relationship to the Cardinal Signs of Inflammation & Their Importance



- KEY: 1: S V.F.b.B.a.: I L-1,3 & LCSF -> leukocytosis  
1 = margination-pavementing (F 2.8, p 33), (11, p 87) **TP VE-7**  
2 = + chemotaxis  
3 = diapedesis-emigration-transmigration (F 2.10, p 34)  
4 = phagocytosis (use lysosomal enzymes to destroy bacteria)

1) = (2) => tissue damage => S.V.E.d. (repair)

KEY: 2: CF IIIA-7: role of released lysosomal enzymes in cellular swelling of adjacent cells

#### TP VE-6

Critical Path VE-3. Leukocytic Responses to Cellular Injury, Their Relationship to a "Cardinal Sign" of Inflammation & Their Importance

### E.b.D. INFLAMMATORY EXUDATES [cells, proteins, & other solid materials gradually discharged from inflamed tissue]

#### E.b.D.a. SEROUS

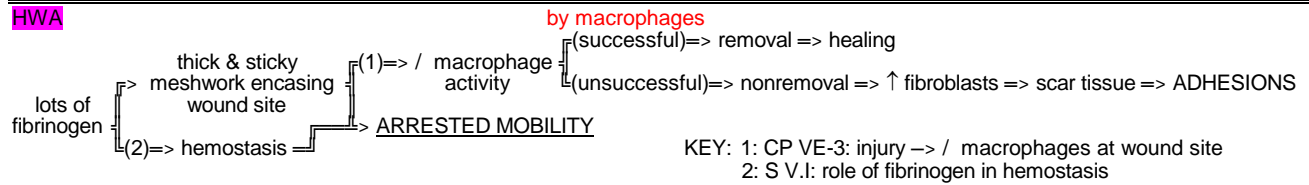
1<sup>st</sup> one to occur [mostly plasma, w/small proteins (no cells)], includes catarrhal inflammation: {inflammation of mucous membrane}  
CI -X-> ↑ mucus; -> ↑ serum)

#### E.b.D.b. HEMORRHAGIC~SANGUINOUS [involves RBCs]

E.b.D.c. **PURULENT** [pus: PMNL & other WBCs, proteins, & tissue debris]  
(band cells -> neutrophils -> microphages~PMNL)

### E.b.D.d. FIBRINOUS

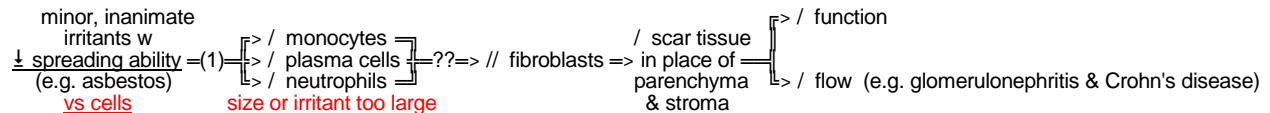
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Path VE-4. Consequences of Fibrinous Exudate

### E.b.D.e. MEMBRANOUS [fibrinopurulent exudate coating mucous surfaces]

### E.c. CHRONIC INFLAMMATION [lasts weeks, to yrs]



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Path VE-5. The Pathophysiology of Chronic Inflammation KEY: 1: CPF VE-1&2: tissue damage → / permeability

### E.d. TISSUE HEALING & REPAIR

#### E.d.A. REGENERATION OF PARENCHYMAL TISSUE [return to normal histological appearance & function] (Table 4.1, p 63)

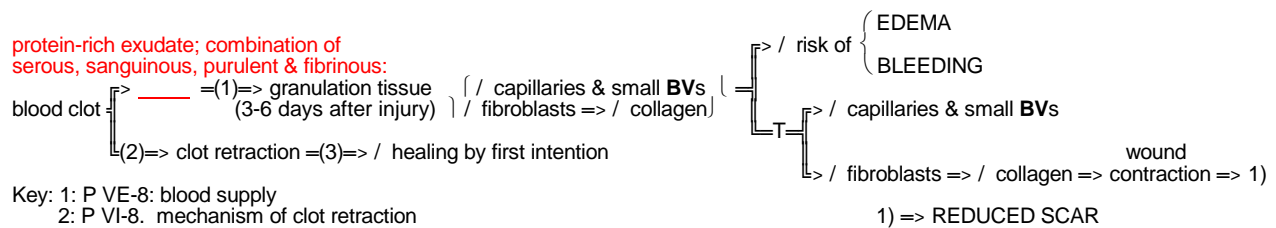
##### E.d.A.a. LABILE CELLS [continue to ÷ throughout life]: easily regenerated (≈ stem cells in F IIIC-2)

##### E.d.A.b. STABLE CELLS [once differentiated, will not ÷ unless damage occurs]: usually easily regenerated (≈ partially differentiated cells in F IIIC-2)

##### E.d.A.c. FIXED CELLS [once differentiated, centrosome is lost, ∴ no new cell division]: not regenerated (≈ fully differentiated cells in F IIIC-2)

### E.d.B. STROMA REPAIR

#### E.d.B.a. GENERAL (F 4.9, p 69)



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Path VE-6. General Process of Connective Tissue Repair

### E.d.B.b. MEMBRANES

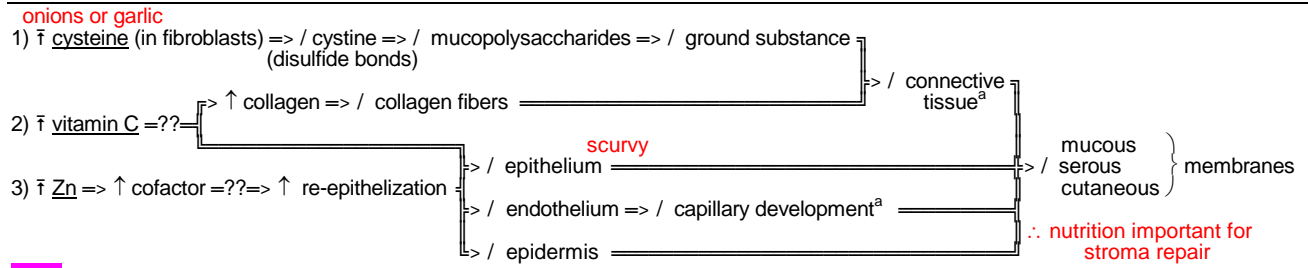
serous & mucous membranes → fibrous exudate → (P VE-4) → adhesion of membranes (12, p 107)

### E.d.C. FACTORS THAT AFFECT WOUND HEALING

#### E.d.C.a. AGE: ↑ age → / time to heal

#### E.d.C.b. INFECTION: ↑ infection → / time to heal

### E.d.C.c. NUTRITION:

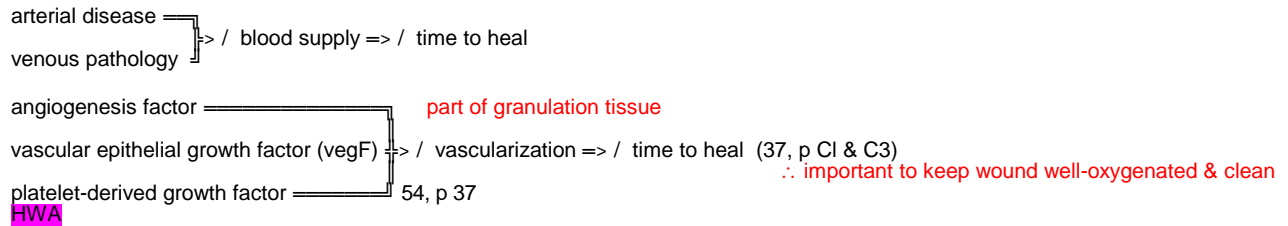


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Path VE-7. Some Aspects of Nutrition & Healing

KEY; <sup>a</sup>connective tissue & capillaries make stroma

### E.d.C.d. BLOOD SUPPLY:



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in the case of chronic or persistent inflammation, these may be needed to stop inflammation

Path VE-8. Some Aspects of Blood Supply & Healing

### E.d.C.e. HORMONAL INFLUENCES:

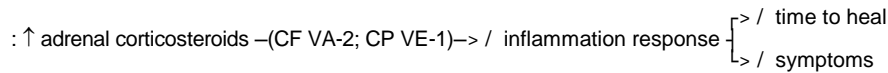


Figure VE-1. Role of Adrenal Corticosteroids in Healing normally very minimal-usually limited to 1<sup>st</sup> 2 days after wound

### E.d.C.f. WOUND SEPARATION~TISSUE LOSS: ↑ separation of edges → / time to heal

healing by First Intention or Second Intention

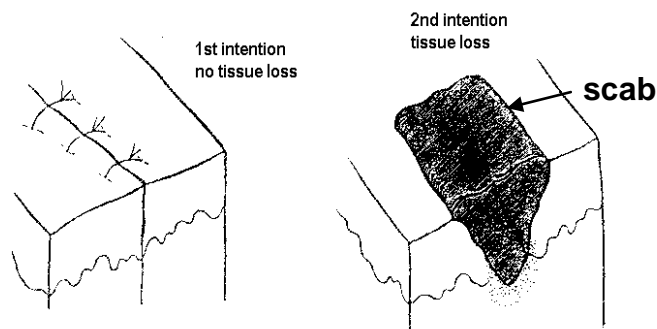


Figure VE-2. Healing by First or Second Intention (4, p 175)

- 1) First Intention [little tissue loss] → / scar tissue → / time to heal
- 2) Second Intention [much tissue loss] → (↑ granulation tissue –(P VE-6 & -8)–> / scar tissue → / time to heal

### E.d.C.g. PRESENCE OF FOREIGN BODIES: e.g. sutures → / time to heal, ∴ remove as soon as possible