# Gingival Inflammation

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- One of the characteristics of living tissues is its ability to react to injury/ irritant

- ***Inflammation*** is defined as an observable alteration in tissues associated with changes in vascular permeability and dilation, often with the infiltration of leukocytes into affected tissues.
- These changes result in the Erythema, Edema, Heat, Pain, and loss of function which are the ***cardinal signs*** of inflammation

# *PERIODONTIUM*

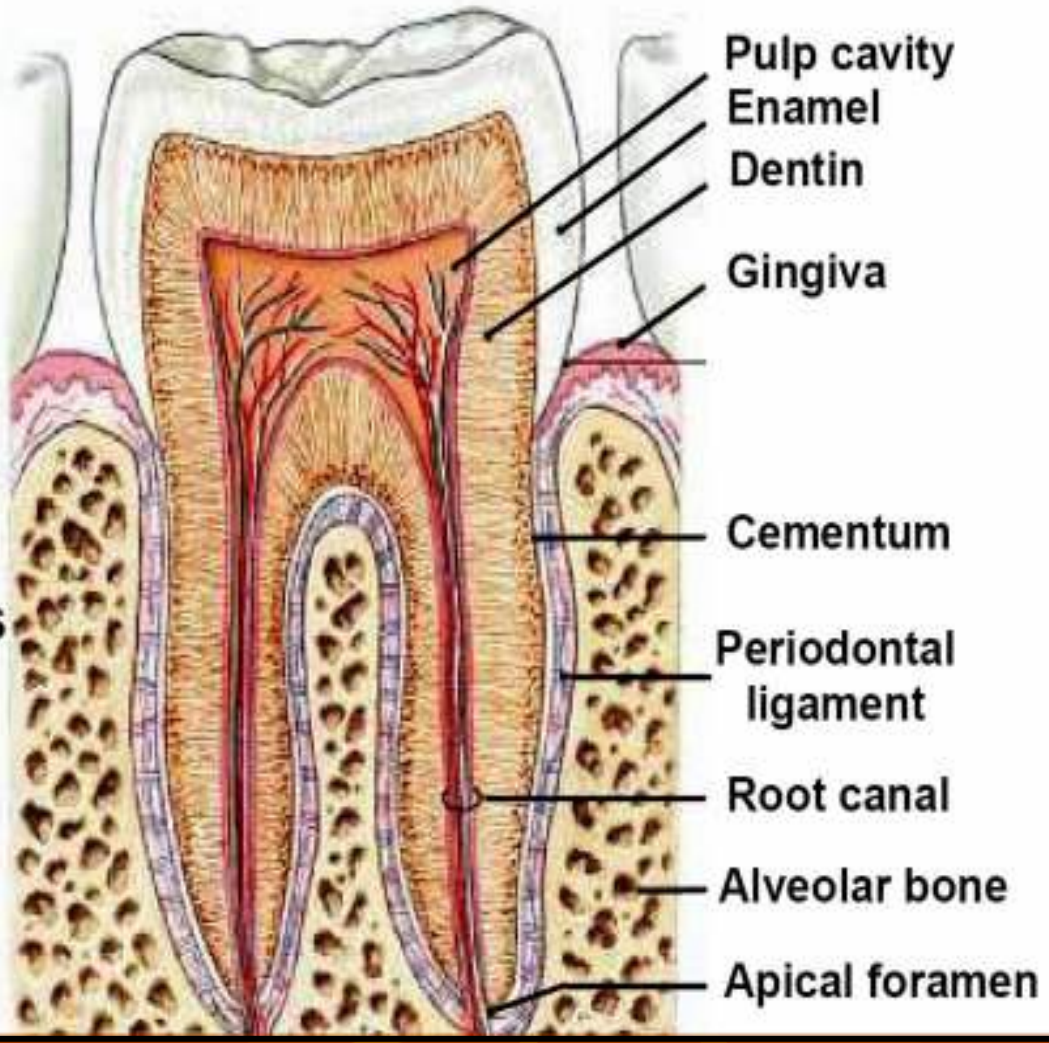
Cementum

PDL

Alveolar bone

Sharpey's fibers

Attachment organ



- The body responds to an infection by developing an inflammatory response.
- The gingiva is no exception, and inflammation in the gingiva is termed ***gingivitis***.
- Despite extensive research, we still cannot distinguish definitively between normal gingival tissue and the initial stage of gingivitis.

# Histologic characteristics of Healthy Gingiva

- Normal junctional epithelium
- Few phagocytosing polymorphonuclear cells from the subepithelial vasculature in the junctional epithelium
- Inflammatory cells consisting predominantly of T cells, with very few B cells or plasma cells
- Minimal exudate from the sulcus

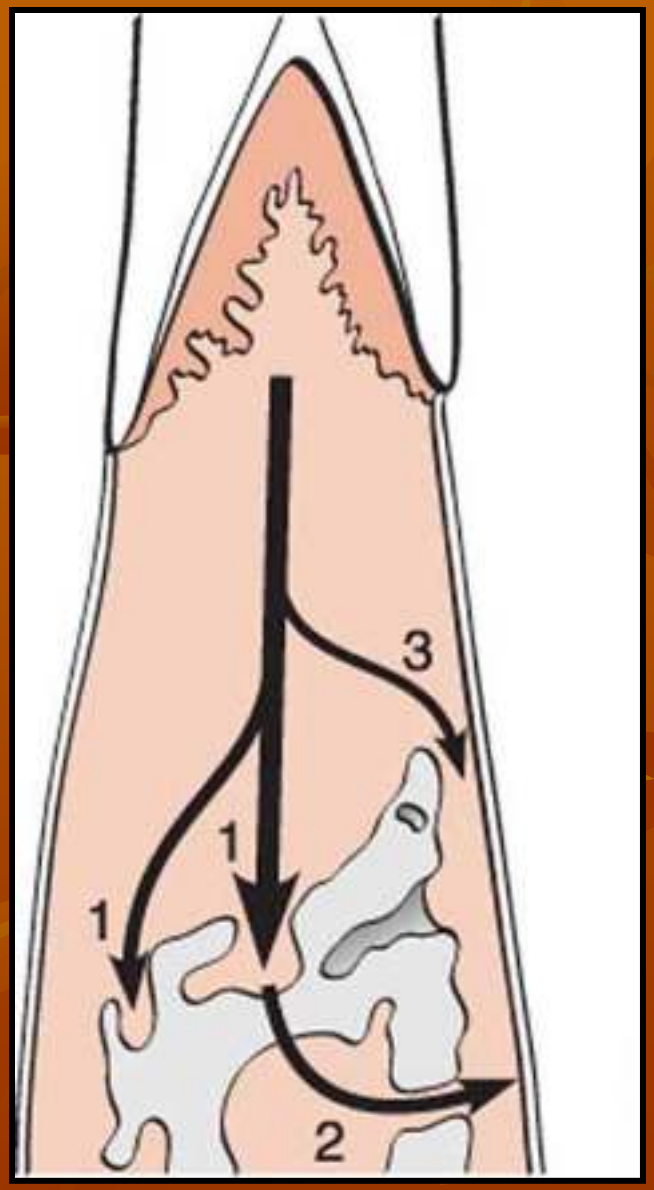
- Under normal conditions, therefore, a constant stream of neutrophils is migrating from the vessels of the gingival plexus through the junctional epithelium, to the gingival margin, and into the gingival sulcus and oral cavity.
- In the absence of clinical signs of inflammation, approximately ***30,000 PMNs*** migrate *per* minute through the junctional epithelia of all human teeth into the oral cavity ***(Schiött and Löe, 1970)***

# Pristine Gingiva

- Its normal gingiva that is free from significant accumulation of inflammatory cells (histologically)
- Super healthy gingiva
- Can be produced in experimental animals

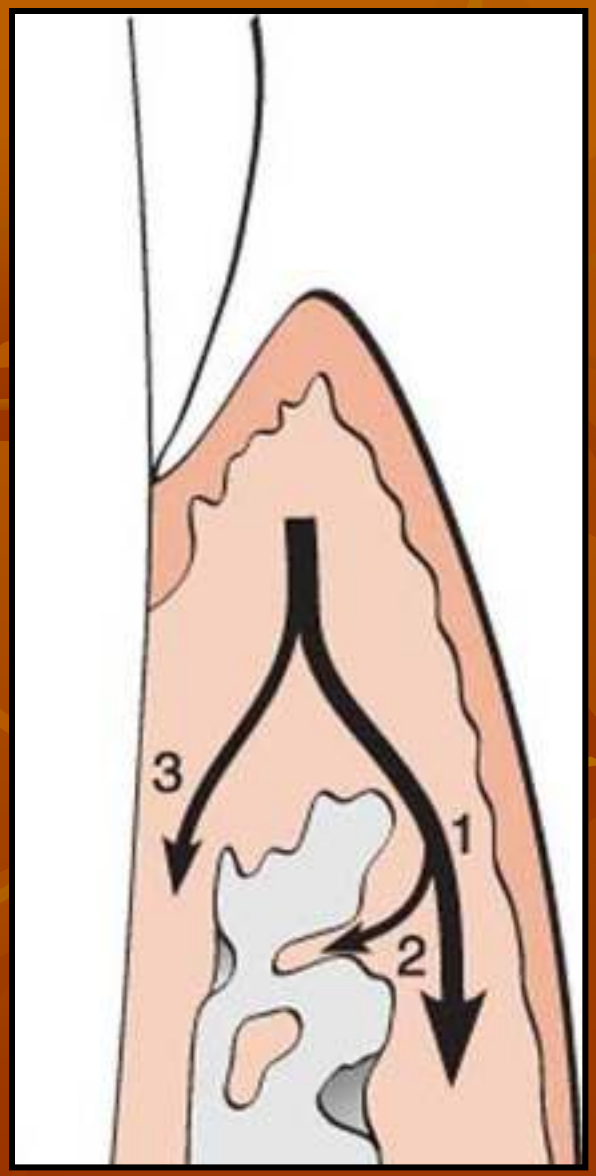
# Pathways of Inflammation from Gingiva into the supporting Periodontal tissues

- Gingival inflammation extends along the collagen fiber bundles and follows the course of the blood vessels through the loosely arranged tissues around them into the alveolar bone.




***Interproximally,***

1. From the gingiva into the bone
2. From the bone into the periodontal ligament
3. From gingiva into the periodontal ligament




***Facially and Lingually***

1. From gingiva along the outer periosteum
2. From the periosteum into the bone
3. From gingiva into the periodontal ligament

***Periodontitis is always preceded by gingivitis, but not all gingivitis progresses to periodontitis***

Gingival inflammation has two components:

*Acute* inflammatory component,

- with vasodilation,
- edema, and
- polymorphonuclear infiltration

*Chronic* inflammatory component,

- with B and T lymphocytes and
- capillary proliferation forming a granulomatous response.

With inflammation the gingival tissue manifests changes in color, size, shape, consistency and tendency for bleeding from gingival sulcus.

| | Normal gingiva | Gingivitis |
|---|---|---|
| Color | Pale pink (melanin pigmentation common in certain groups) | Reddish/bluish red |
| Size | Papillary gingiva fills interdental spaces; marginal gingiva forms knife edge with tooth surface; sulcus depth ≤ 3 mm | Swelling both coronally and bucco/lingually; false pocket formation |
| Shape | Scalloped – troughs in marginal areas rise to peaks in interdental areas | Edema which blunts the marginal and papillary tissues leads to loss of knife edge adaptation. Marginal swelling leads to less accentuated scalloping |
| Consistency | Firm | Soft; pressure induced pitting due to edema |
| Tendency to bleed | No bleeding to normal probing | Bleeding on probing |

- The sequence of events cumulating in clinically apparent gingivitis was categorized by ***Page & Schroeder*** into
    1. Initial,
    2. Early,
    3. Established,
    4. Advanced/ stage of periodontal breakdown ***(Lindhe)***

- The initial and early phases were thought to reflect the histopathology of clinically ***"acute"*** or early stages of gingivitis, while the established lesion reflected the histopathology of ***"chronic"*** gingivitis.
- Progression of gingivitis to periodontitis was reflected in the histopathology of the advanced lesion.

## TABLE 21-1

Stages of Gingivitis

| Stage | Time (Days) | Blood Vessels | Junctional and Sulcular Epithelia | Predominant Immune Cells | Collagen | Clinical Findings |
|---|---|---|---|---|---|---|
| I. Initial lesion | 2-4 | Vascular dilation<br>Vasculitis | Infiltration by PMNs | PMNs | Perivascular loss | Gingival fluid flow |
| II. Early lesion | 4-7 | Vascular proliferation | Same as stage I<br>Rete pegs<br>Atrophic areas | Lymphocytes | Increased loss around infiltrate | Erythema<br>Bleeding on probing |
| III. Established lesion | 14-21 | Same as stage II, plus blood stasis | Same as stage II but more advanced | Plasma cells | Continued loss | Changes in color, size, texture, etc. |

PMNs, Polymorphonuclear leukocytes (neutrophils).

# Stage I Gingivitis: The Initial Lesion

- Microbes– activation of resident leukocytes– subsequent stimulation of endothelial cells--
- Vascular changes- dilated capillaries & increased blood flow
- ***Subclinical gingivitis-*** initial response to plaque is not apparent clinically

- Microscopically- features of acute inflammation in CT beneath JE– widening of capillaries – adherence of neutrophils to vessel wall– PMNs leave capillaries– seen more in CT, JE & gingival sulcus– exudation of fluid from gingival sulcus & extravascular proteins
- Subtle changes in JE & CT– CT is altered, exudation & deposition of fibrin– lymphocytes begin to accumulate

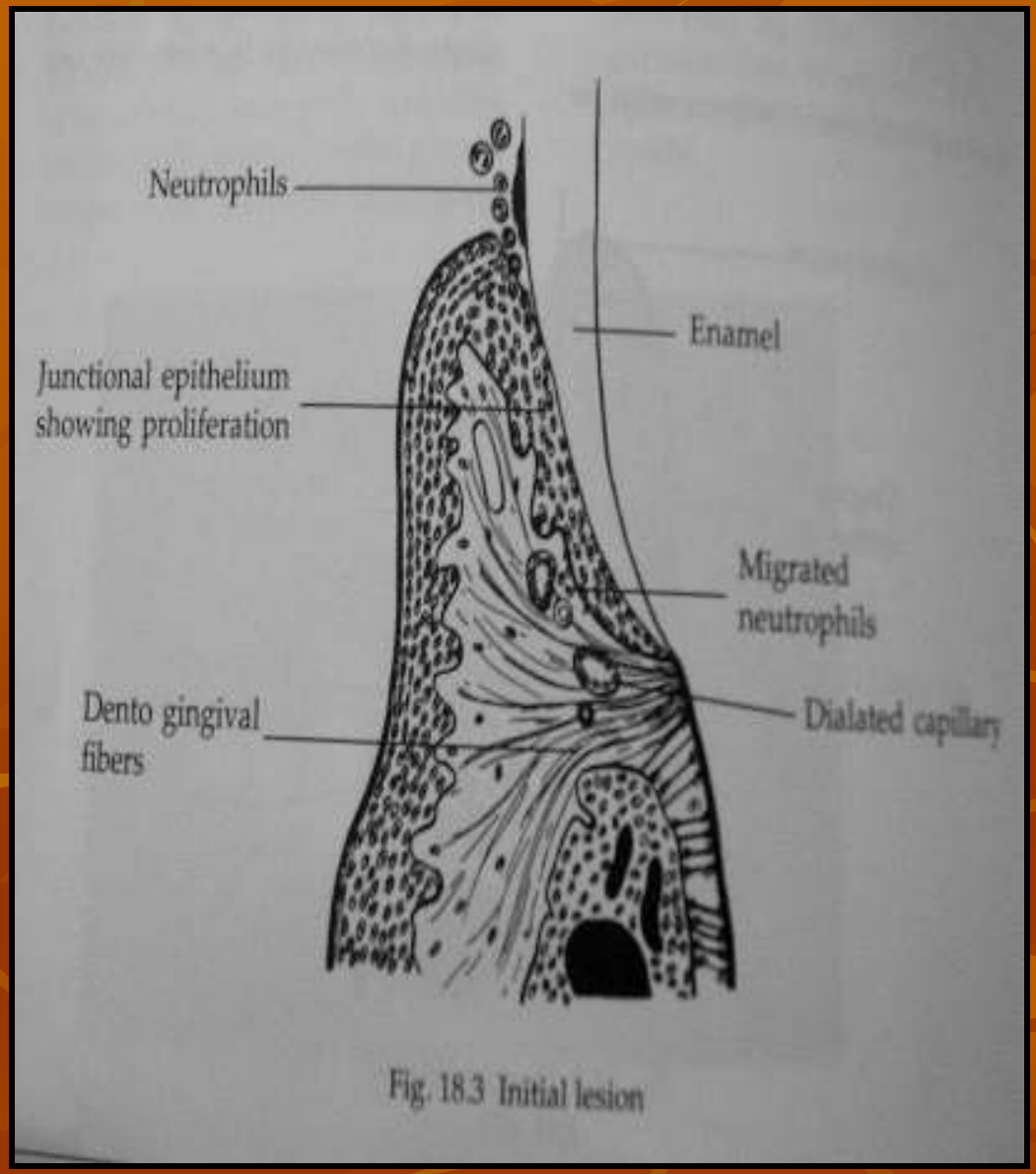



Fig. 18.3 Initial lesion

# Stage II Gingivitis: The Early Lesion

- The early lesion evolves from the initial lesion within about 1 week after the beginning of plaque accumulation.
- Proliferation of capillaries & increased formation of capillary loops between rete pegs– clinical signs of erythema.
- Bleeding on probing
- Gingival fluid flow and the no. of transmigrating leukocytes reach their maximum between 6 and 12 days after the onset of clinical gingivitis

- Collagen destruction, 70% of collagen destroyed around cellular infiltrate
- Fibers affected- ***circular & dentogingival***
- PMNs found in epith & emerge in sulcus– attracted to bacteria– phagocytosis– release lysosomes
- Microscopically- leukocyte infiltration in CT beneath JE, consisting mainly of lymphocytes ***(75% T cells)*** but also some migrating neutrophils, macrophages, plasma cells & mast cells– (all changes in the initial lesion intensify)
- Neutrophils infiltrate JE & sulcus– JE shows development of rete pegs/ridges.

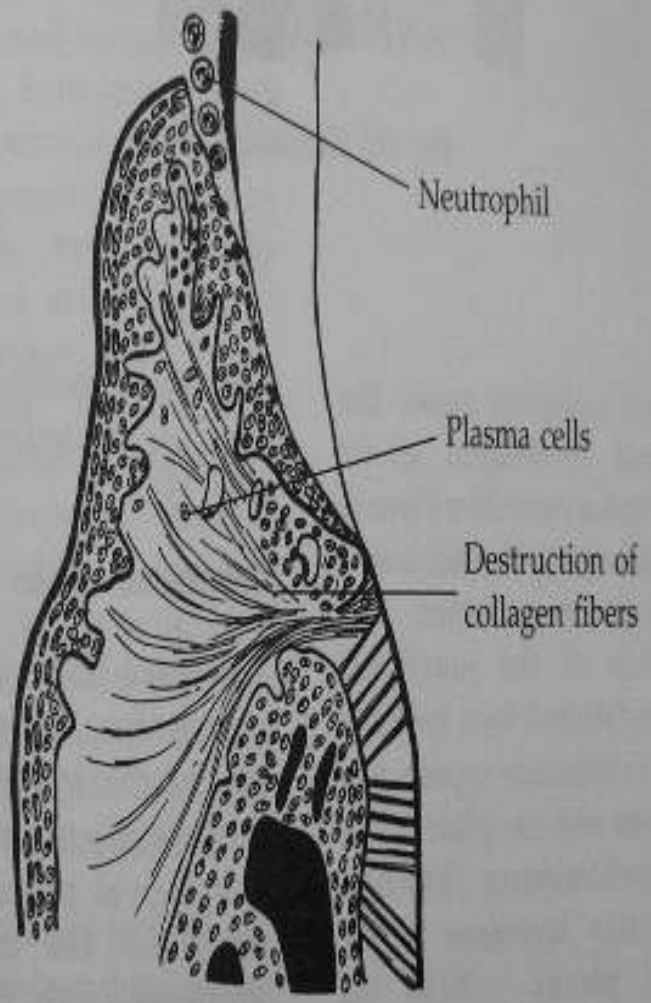



Fig. 18.4 Early lesion

# Stage III Gingivitis: The Established Lesion

- Characterized by predominance of ***plasma cells*** and B lymphocytes with the creation of small gingival pocket lined with a pocket epithelium.
- B cells produce predominantly IgG1 and IgG3
- Vessels engorged & congested– impaired venous return– sluggish blood flow– ***localized gingival anoxemia***– bluish hue on reddened gingiva
- Increased collagenolytic activity by collagenase (bacteria & PMNs)
- Established lesion- moderately to severely inflamed gingiva.

Microscopically- increased plasma cells– invade CT, around blood vessels & between collagen bundles.

JE- wide intercellular spaces filled with granular cellular debris, including lysosomes that contain acid hydrolases that destroys tissue

JE rete pegs protrude into connective tissue & basal lamina destroyed in some areas.

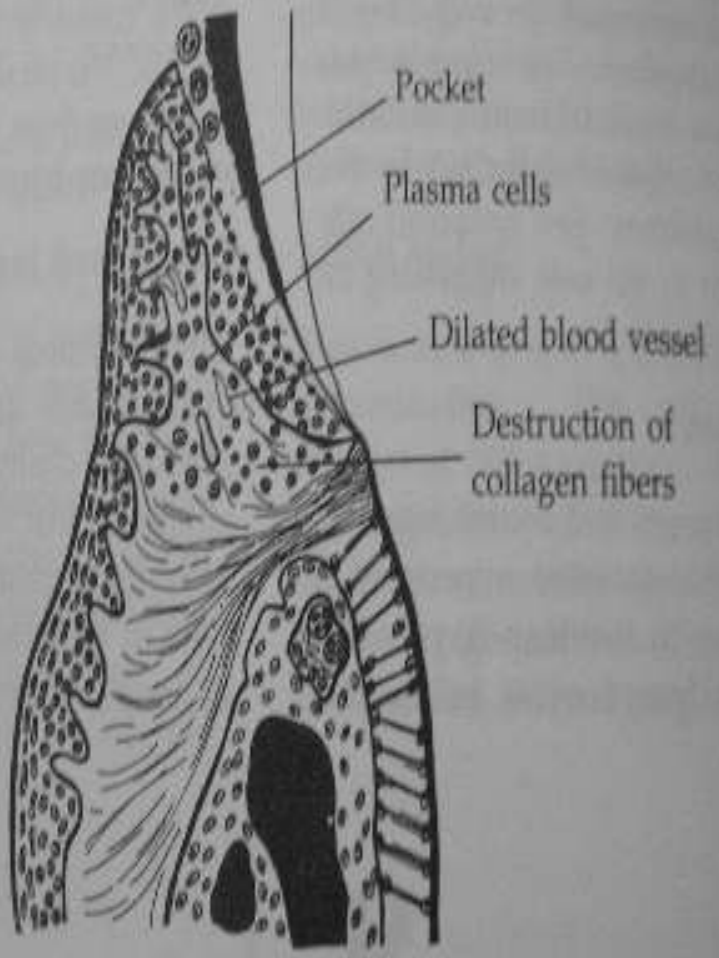



Fig. 18.5 Established lesion

2 types-

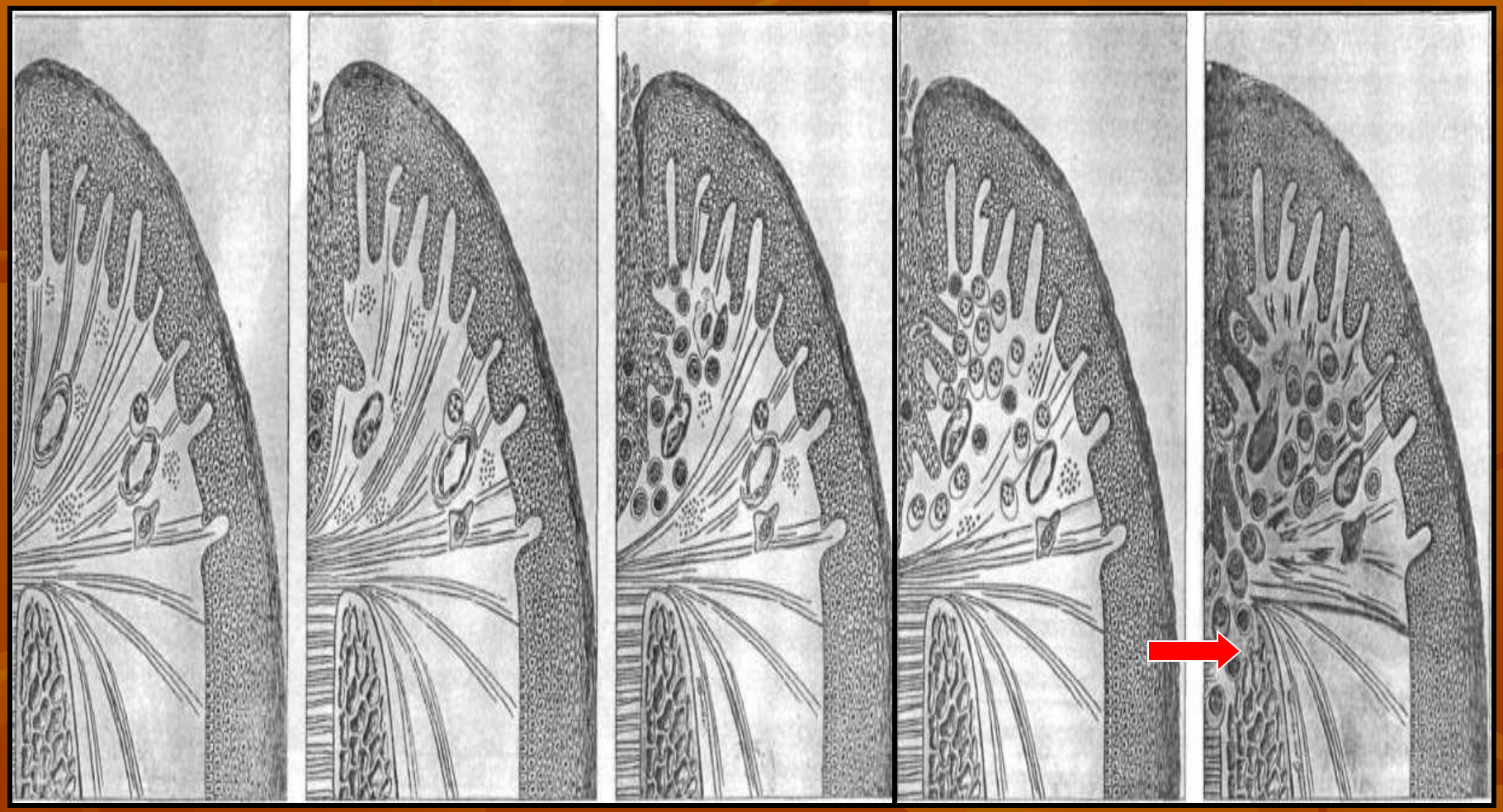
1. Remain stable & don't progress for months/yrs
2. More active, converts to progressively destructive lesions

Established lesion is reversible– sequence of events after perio therapy are reverse to that of gingivitis

As it reverses plasma cells decrease in no. & lymphocytes population increases proportionately

# Stage IV Gingivitis: The Advanced Lesion

- Extension of the lesion into alveolar bone characterizes a fourth stage known as the ***advanced lesion*** or ***phase of periodontal breakdown***.
- Microscopically, there is
  - fibrosis of the gingiva and
  - widespread manifestations of inflammatory and immunopathologic tissue damage.
- Plasma cells continue to dominate the connective tissues, and neutrophils continue to dominate the junctional epithelium and gingival crevice.



- Gingivitis will progress to periodontitis only in individuals who are susceptible.
- However, whether periodontitis can occur without a precursor of gingivitis is not known at this time

- Inflammation
- Histologic characteristics of Healthy Gingiva
- Pristine gingiva
- Pathways of Inflammation from Gingiva into the supporting Periodontal tissues
- Stages of Gingivitis

# THANK YOU

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