

## **Periodontology- fourth stage**



# First semester-Defense Mechanisms of the Gingiva Lec-10

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- The gingival tissue is continuously subjected to challenge, influencing the host response type.
- Resistance to these action is provided by:
  - ✓ Sulcular Fluid (Gingival crevicular fluid).
  - ✓ Saliva.
- ✓ Leukocytes in the Dentogingival Area.

## Gingival crevicular fluid

- Gingival crevicular fluid : It's a complex mixture of substance derived from serum, leukocyte, structural cells of the periodontium and oral bacteria.
- In the healthy sulcus, the amount of gingival fluid is very small. During inflammation, however, the gingival fluid flow increases, and its composition starts to resemble that of an inflammatory exudate.
- The main route of the gingival fluid diffusion is through the basement membrane, through the relatively wide intercellular spaces of the junctional epithelium, and then into the sulcus.



# GCF: Transudate or exudate?

 Brill (1950) confirmed the presence of GCF in humans and considered it as "transudate.

 However, others demonstrated that GCF is an inflammatory exudate rather than a continuous transudate. In strictly normal gingiva, little or no fluid can be collected.



# **Function of GCF**

(1) Cleanse material from the sulcus.

(2) Contain plasma proteins that may improve adhesion of the epithelium to the tooth.

(3) Possess antimicrobial properties.

(4) Exert antibody activity to defend the gingiva.



## **Methods of Collection**



## (1) Absorbing paper strips

 The absorbing paper strips are placed within the sulcus (intrasulcular method) or at its entrance (extrasulcular method)





Intrasulcular method

Extrasulcular method

- Placement of the filter paper strip in relation to the sulcus or pocket is important. The Brill technique involves inserting it into the pocket until resistance is encountered (Fig.A). This method produces some degree of irritation of the sulcular epithelium that by itself can trigger the flow of fluid.
- To minimize this irritation, Löe and Holm-Pedersen placed the filter paper strip just at or over the pocket entrance (Fig. B–C). In this way, fluid that seeps out is picked up by the strip, but the sulcular epithelium is not in contact with the paper.



Extrasulcular methods

#### (2) Twisted threads

✓ Weinstein and colleagues used preweighed twisted threads. The threads were placed in the gingival crevice around the tooth, and the amount of fluid collected was estimated by weighing the sample thread.



## > Micropipette

The use of micropipettes permits the collection of fluid by capillarity. Capillary tubes of standardized length and diameter are placed in the pocket, and their contents are later centrifuged and analyzed.



## > Crevicular washings

✓ Crevicular washings can be used to study GCF from clinically normal gingiva.

A. One method involves the use of an appliance that consists of a hard acrylic plate that covers the maxilla, with soft borders and a groove that follows the gingival margins.

- It is connected to four collection tubes.
- Washings are obtained by rinsing the crevicular areas from one side to the other with the use of a peristaltic pump

B. A modification of the this method involves the use of two injection needles that have been fitted one within the other.

- During sampling, the inside (ejection) needle is at the bottom of the pocket and the outside (collecting) needle is at the gingival margin.
- The collection needle is drained into a sample tube via continuous suction



## **Methods of Measurement**

• The amount of GCF collected on a paper strip can be studied in multiple ways:

## 1. Staining method

- Accessing the area of filter paper wetted by GCF sample.
- This can be made more visible by staining with Ninhydrin to produce a purple color in the area where gingival fluid is accumulated.

## 2. An electronic method (Periotron).

 The wetness of the paper strip affects the flow of an electric current and provides a digital readout.

TableTranslation of Periotron values to clinicalconditions and Gingival Index with which they maybe asd			
Periotron reading	Level of gingival inflammation	Gingival Index	
0-20	healthy	0	
21-40 41-80	mild	2	
81-200	severe	3	



## **3.Weighting the strips.**

Strip is weighed before and after collecting the GCF sample.

## **Composition of GCF**

#### Cellular Elements

- Bacteria
- Desquamated epithelial cells
- Leukocytes

#### Electrolytes

- Potassium
- Sodium
- Calcium
- Fluoride

## Organic Compounds

- Carbohydrates
- proteins
- Lipid

## Metabolic and bacterial products

- Lactic acid
- Urea
- Hydroxyproline
- Endotoxins
- Cytotoxic substances
- Hydrogen sulfide
- Antibacterial factors

#### Enzyme

- Acid phosphatase
- Hyaluronidases
- Alkaline phosphatase
- Cytokines
- Collagenase

## **Clinical Significance of GCF**

The amount of GCF is greater when inflammation is present, and it is sometimes proportional to the severity of inflammation.

## □ Factors that influence the amount of GCF are:

## > Circadian Periodicity

 There is a gradual increase in the amount of GCF from 6 a.m. to 10 p.m. and a decrease thereafter.

## Sex Hormones

- Female sex hormones increase GCF flow, probably because they enhance vascular permeability.
- Pregnancy, ovulation, and hormonal contraceptives all increase GCF production.

#### Mechanical Stimulation

 Chewing and vigorous gingival brushing stimulate the flow of GCF. Even minor stimuli represented by intrasulcular placement of paper strips increases the production of fluid.

#### > Smoking

 Smoking produces an immediate transient but marked increase in GCF flow but, in the long term, a decrease of salivary and GCF flow.

## Periodontal Therapy

• There is an increase in GCF production during the healing period after periodontal surgery.

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#### > Periodontal Therapy

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## **Drugs in Gingival Crevicular Fluid**

 Drugs that are excreted through the GCF may be used advantageously in periodontal therapy.

Bader and Goldhaber demonstrated that intravenously administered tetracyclines in dogs are excreted through the GCF; this finding triggered extensive research that showed a concentration of tetracyclines in GCF as compared with serum. Metronidazole is another antibiotic that has been detected in human GCF.



- $\checkmark$  Saliva has protective functions and maintains the oral tissues in a physiologic state.
- ✓ Saliva exerts a major influence on plaque by mechanically cleansing the exposed oral surfaces, buffering acids produced by bacteria, and modulating bacterial activity with immune mediators.
- ✓ Saliva is now considered a main biologic fluid for diagnosis of human health and diseases. Systemic and local disease markers are available through saliva.

## **Role of Saliva in Oral Health**

## I. Antibacterial Factors

- Saliva carries inorganic and organic factors that influence bacteria and their products in the oral environment.
- Inorganic factors include ions and gases, bicarbonate, sodium, potassium, phosphates, calcium, fluorides, ammonium, and carbon dioxide.
- Organic factors include lysozyme, lactoferrin, myeloperoxidase, lactoperoxidase, defensins, peptides, and agglutinins such as glycoproteins, mucins, β2-macroglobulins, fibronectins, and antibodies.

A. Lysozyme is a hydrolytic enzyme that cleaves the linkage between structural components of the glycopeptide muramic acid–containing region of the cell wall of certain bacteria in vitro.

**B.** Lactoperoxidase-thiocyanate system in saliva has been shown to be bactericidal to some strains of Lactobacillus and Streptococcus by preventing the accumulation of lysine and glutamic acid, both of which are essential for bacterial growth.

C. Myeloperoxidase, it is bactericidal for Actinobacillus.

**D.** Salivary Antibodies: Saliva contain IgG,IgM and IgA antibodies. Although immunoglobulins G (IgG) and M (IgM) are present, the preponderant immunoglobulin found in saliva is immunoglobulin A (IgA), It has been shown that IgA antibodies present in parotid saliva can inhibit the attachment of oral Streptococcus species to epithelial cells.

## **II.** Salivary Buffers and Coagulation Factors

- The primary effect action of buffers has been investigated with relationship to dental caries. In saliva, the most important buffer is the bicarbonate-carbonic acid system.
- ✓ Saliva also contains coagulation factors (i.e., factors VIII, IX, and X; plasma thromboplastin) that hasten blood coagulation and protect wounds from bacterial invasion.

## **III. Leukocytes**

✓ Novel investigation presents the positive correlation between rate of PMN migration and the severity of gingival inflammation, and it is therefore a reliable index for the assessment of gingivitis.

Function	Salivary Components	Probable Mechanism
Lubrication	Glycoproteins, mucoids	Coating similar to gastric mucin
Physical protection	Glycoproteins, mucoids	Coating similar to gastric mucin
Cleansing	Physical flow	Clearance of debris and bacteria
Buffering	Bicarbonate and phosphate	Antacids
Tooth integrity maintenance	Minerals	Maturation, remineralization
	Glycoprotein pellicle	Mechanical protection
Antibacterial action	Immunoglobulin A	Control of bacterial colonization
	Lysozyme	Breaking of bacterial cell walls
	Lactoperoxidase	Oxidation of susceptible bacteria

## Leukocytes in the Dentogingival Area

- Leukocytes have been found in clinically healthy gingival sulci in humans and experimental animals. The leukocytes found are predominantly PMNs. The main port of entry of leukocytes into the oral cavity is the gingival sulcus.
- Leukocytes are present in sulci even when histologic sections of adjacent tissue are free of inflammatory infiltrate.
- Differential counts of leukocytes from clinically healthy human gingival sulci have shown 91.2% to 91.5% PMNs and 8.5% to 8.8% mononuclear cells.

- Mononuclear cells were identified as
- ✓ 58% B lymphocytes,
- ✓ 24% T lymphocytes
- ✓ 18% mononuclear phagocytes.
- The ratio of T lymphocytes to B lymphocytes was found to be reversed from the normal ratio of about 3:1 found in peripheral blood to about 1:3 in GCF.
- The majority of these cells are viable and have phagocytic and killing capacity. Therefore leukocytes constitute a major protective mechanism against the extension of plaque into the gingival sulcus.



 Newman and Carranza's Clinical Periodontology, THIRTEENTH EDITION.

