

## Oral microbial flora and oral, maxillary and facial infections

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Oral, maxillary and facial anatomical sites are colonized by specific microorganisms. The study of infections located in these territories, as well as of those expanding to other sites cannot be performed without an ecological approach on microbial populations, starting during the first 2-3 hours of life of the newborn.

In the oral and nasal cavities and in the pharynx there is a continuous development of a high number of microbial species.

In the oral biotype there are favorable conditions: nutritive substances, appropriate temperatures, moisture, pH, rH, etc. An estimation on the available oral surface for bacterial growth gave about  $4 \times 10^{28}/\mu^2$ . If we imagine a single layer of  $0.5 \mu$  diameter cocci on this surface, we would get a total number of  $16 \times 10^{28}$  bacterial cells. In the oral cavity about 300 bacterial species have been described, 50 of which have been investigated during the last years. Certainly, there are many other organisms about which there is little, if any, data available.

Estimations have been made that the oral microbiocenosis, as compared to other human microbiocenosis, is characterized by the highest degree of complexity.

We may stress upon the anatomical and physiological continuity of the three ecotypes: the pharynx, including the tonsils, the oral cavity, including the sustaining structures of teeth and the dental plaque - which has been individualized following studies which have established its participation in the pathogeny of dental caries and periodontal disease.

Air, liquids, foods and physiological and pathological secretions cross the border between the three ecotops down the respiratory and digestive tracts, which become loaded with aerosols, dusts, food debris, various microorganisms, etc. - elements which alter the physiologic status of the epithelia. The abundance, diversity and dis-

tribution of microbial species in the oropharynx are directly related to repeated inflammations of the throat and tonsils (over 90% viral infections) and to chemical, termic and mechanical trauma of the oral mucosa, gingival tissues and of the dental sustaining structures. The anatomical communication of this area with the parotid and maxillary glands, with the middle ear, facial and mastoidian sinuses lead to the presence of similar bacterial species involved in contiguous infectious processes (otitis, sinusitis, mastoiditis, etc).

The three ecotopes receive infectious agents, offer multiplication conditions and eliminate them in various ways.

Quantitative measurements of oral microorganisms (oral microbiocenosis) have an orientative value, mainly due to the unequal distribution of microbes in the oral cavity.

The dental areas on which the self-cleaning mechanism does not work - the dental plaque, the gingival groves, the lingual dorsal surface, the oropharynx - have the highest microbial load. Saliva, which is virtually sterile in the moment of excretion, becomes loaded with crevicular fluid, desquamated epithelial cells, leukocytes, various metabolites, enzymes, immunoglobulines, minerals, food debris, etc. The resulting oral fluid does not offer a representative picture for the oral biocenosis, its microbial load being lower than in the above-mentioned areas. Streptococci have been found in 300 times higher numbers in the bacterial plaque than in the oral fluid. Beta-hemolytic streptococci, which are present in the pharynx in pathological conditions, may only exceptionally be found in the oral fluid.

The adherence and colonization by resident microorganisms, as well as by transient ones, which are brought in together with foods, water, inspired air, etc., are limited by the intervention

of physical, chemical and biological mechanisms such as:

- the unidirectional flow of the oral fluid (20-40 irrigations/hour) and of the consumed liquids;
- the composition of foods, the mastication process, the movements of the food bolus and the deglutition process (up to 9 reflex deglutitions/minute during one meal);
- the oral mucus which prevents the adherence and the penetration of bacteria and viruses in the epithelial cells;
- the epithelial desquamation which carries away the attached microbial clusters;
- various antimicrobial substances (lysozyme, immunoglobulins, enzymes - inhibiting factors for some bacteria, toxic radicals, etc.);
- leukocytes (normally up to 4 million/l oral fluid);
- local pH and rH variations;
- microbial antagonism phenomena.

The process of constitution and organization of the oral microbiocenosis as well as of other microbiocenosis are an expression of symbiotic relations between resident microorganisms and human hosts. Microorganisms receive nourishments, multiplication capacities and, to some extent, protection conditions. On the other hand, the human host benefits of microbial activities such as the stimulation of the immune system, prevention of microbial colonization with pathogenic bacteria, the local production of biologically active substances, etc. This symbiotic relationship, with undetermined duration, is established in the presence of disturbing factors such as antibiotic drugs, in conditions that vary from one individual to the other.

The conflict between microorganisms and the human host do not involve only pathogenic bacteria. Many resident bacteria may act as pathogens, overwhelming the resistance mechanisms of the human host and leading to infections.

In the oro-maxillary and facial area the conflict between microorganism and the host may have the following manifestations:

1. General, systemic, specific infections in which a resident microorganism of the oral flora (e.g. *Treponema* spp.) may accidentally reach in the area and causes a local lesion, but, as a general rule, oral lesions become apparent in the secondary phase of the general infection;
2. Nonspecific, metastatic infections in which occasionally pathogenic microorganisms

belonging to the normal oral flora are involved. The site of infection is a distant one (e.g. the endocardium) and the oral cavity is free of symptoms or local lesions (e.g. bacterial endocarditis with oral source). Other bacteria occasionally pathogenic, belonging to the normal oral resident flora: *Actinomyces* spp., *Arahnia* spp., *Nocardia* spp., may cause specific infections in the oral, maxillary and facial areas, which may expand to other tissues and organs;

3. Nonspecific infections, clinically and pathogenetically characteristic for the area, usually produced by resident, occasionally pathogenic bacteria and, less frequently, by exogenous pathogenic agents accidentally brought in by foreign bodies in traumatic wounds;

4. Major diseases - dental caries and periodontal disease - produced by microorganisms which belong to the normal flora and which add up their pathogenic effects, integrating into a unique ecosystem of the human organism - the dental bacterial plaque. The role of the ecosystem in the attack on the hard dental tissues and on the periodontal sustaining structures does not represent the aim of the present paper;

5. Microbial activities with defavorable effects on the human host, determined by various resident microorganisms:

a. halitosis („halitus“ = breath): in case of lacking hygiene, treponemas, fusiform bacilli, anaerobic gram-negative bacteria and other germs from the normal oral flora multiply in the epi- and interdental deposits, causing the degradation of some aminoacids (cysteine, cystine, methionine), biogenic amines and sulphurous compounds which are expired;

b. beta-lactamase production: recent researches explain a series of failures of the beta-lactamine treatments by the fact that some anaerobic oral bacteria produce beta-lactamase thus protecting pathogenic bacteria normally sensible to these antimicrobial substances. Other germs may also benefit, such as: beta-hemolytic streptococci, which, in the presence of beta-lactamase producing bacteria, may tolerate penicillines in higher concentrations than *in vitro* by this mechanism;

c. the production of specific proteases which inactivate secretory IgA in the oral secretions leading to a certain decrease of the local antiinfectious resistance and thus favoring infections starting from this area;

d. the pathogenic cooperation between the influenza virus and *Staphylococcus aureus* on the nasal and oropharyngeal mucosa.

It is very important to delimitate the terms „specific infection“ and „nonspecific infection“.

„Specific infections“ are clinically, pathologically, etiologically, immunologically and epidemiologically individualized. Usually they are mono-microbial and caused by exogenous pathogens.

„Nonspecific infections“ are poli-microbial (mixed); the triggering and evolution of the infection are determined by synergical relations between groups of microorganisms, pathogenetically associated. Endodontic, periodontal infections and other infections of the oro-maxillo-facial area, infections of neighboring tissues (sinusitis, otitis, tonsillar and peritonsillar abscesses) as well as post-traumatic infections are produced by associations of anaerobic and aerobic bacteria, usually optionally pathogenic, belonging to the normal flora of the organism (mouth, skin) and, less frequently, by exogenous germs.

The term „focal infection“ designates the existence of an infectious site, more or less clinically detectable, which may be the origin of generalized infections (septicaemias) of septic, allergic, toxic lesions localized in various anatomical sites. At present, real infectious foci, which need curing, may be detected by clinical, radiological and biological tests.

In the pathogenesis of infections three elements must always be considered:

- persons at high risk;
- oral interventions at high risk;
- practical aspects of the preventive administration of antibiotics.

### Persons at high risk of infection

One of the major risks is represented by a cardiac impairment with an objective auscultatory sound, as well as a history of infectious endocarditis and any foreign material implanted into

the myocardial tissue. The Consensus of 1992 differentiates: major risk cardiac diseases (valvular prosthesis, congenital cyanogenic cardiac diseases and history of infectious endocarditis); cardiomyopathies at high risk (valvular diseases, congenital non-cyanogenic cardiac diseases, obstructive cardiomyopathies, etc).

### Oral interventions at high risk

In order to evaluate the risk of an oral intervention, one must keep in mind that during gingival and periodontal infections, spontaneous bacteriemias may occur, which may in turn lead to infectious endocarditis. The involvement of oral interventions in infectious endocarditis is extremely difficult to assess.

### Practical aspects of the preventive administration of antibiotics

Preventively administered antibiotics are chosen considering their antistreptococcal activity especially against the groups of *Streptococcus viridans* in the oral cavity.

For ambulatory dental treatments, which usually consist of dental extractions in persons without allergic symptoms to beta-lactamines, oral administration of Amoxyciline 3 g, an hour before the extraction. In persons allergic to beta-lactamines, Clindamycine 600 mg, an hour before the extraction is recommended.

For complex oral treatments performed in the hospital, with or without general anesthesia - Amoxyciline 2 g i.v. (30 minutes of i.v. perfusion) during the hour preceding the intervention and then, 6 hours later, 1 g. In persons with beta-lactamine allergy - Vancomycine 1 g i.v. (60 minutes of i.v. perfusion) or Teicoplanine 400 mg i.v.

Depending on resources, clinical situations, experience of the specialists, intraoperative facts and further evolutions, the above recommendations become orientative and every specialist will perform the necessary alterations of doses, rhythm and duration of administration.

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