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Tooth physiology constitutes the missing link in the education of the future dentist. In this paper, we outline some aspects of the tooth's physiology that will, to some extent, bring awareness to the processes that take place within the tooth during its periods of formation, eruption and during its activity as an organ in the oral cavity.

The exposed material is dedicated to specialists in the field, dental students, residents, doctorates and practitioners in continuing education.funcționării ca organ în cavitatea bucală.

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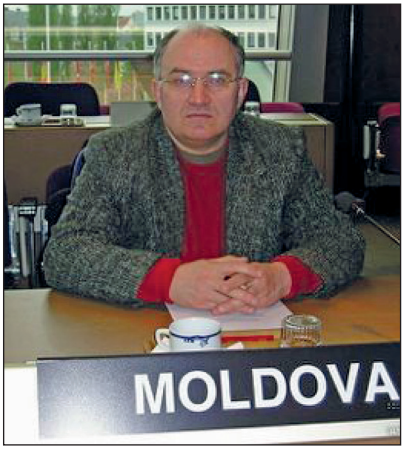

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* - Chapters written in collaboration with Rostislav Okushko

The Story of one "Dental Discovery" (as a preface)

"Theory without practice is of no value, practice is helpless without theory"- this Marxist statement, a theoretical one itself, is remembered by none in our days. Pragmatism, as a general principle, is completely governing the civilised contemporary world and the orientation of its vertiginous self-regulating vector is clear to all those who have asked themselves this question. We are not referring to an apocalypse, but to our modest sphere of dental interests and activities in the context of practice and theory.

These lines are addressed to a rather narrow and specific public. We would like to, once again, invite the readers to reflect upon the "society, theory and dental practice" triangle.

In the distant Soviet past, a tentative to join "practice" and "theory" into a single administrative institution was made. Back then, there was an institution called "The State Committee of Discoveries and Innovation", which confirmed one's ownership over the description of an unknown phenomenon and over a practical or theoretical innovation. Using this possibility, the researchers could count on the state to support their researches, and this mattered a lot. Thereby, in 1977, I, together with my PhD students- V.V. Baranov, E.I. Donskoi, A.V. Melehin, submitted a request to register our latest discovery: "The phenomenon of self-regulation of the enamel's hardness and solubility" (No. 32-01 from 12.05.77). Today, after four decades, we can afford ourselves to be more critical towards the title, because the enamel, being isolated from the rest of the organism, cannot exercise the act of self-regulation.

However, the actual content of the paper and the scientific facts described in it have not been challenged so far. The research

stipulated the variability of the enamel's physical parameters, which are controlled by the endogenous environment through the dental pulp. This scientific phenomenon fits perfectly into the general biological concept about the homeostatic reactions of an organism. At the same time, it absolutely refuted the belief (that persists even to this day) stating that the enamel is nurtured and mineralised by saliva.

My correspondence with the State Committee lasted over 10 years and my stubbornness was based on the idea that only by this insistent correspondence could we get a clear answer: yes, or no. A refusal could be based solely on the lack of factual proofs (the detection of methodical or logical errors that we could have made) or on the fact that someone else had preceded me with similar data. In either of these cases, we would have received the information that could have allowed me to go on. Regardless of their response, we would have received an additional impetus to work harder. Despite all these, during my lengthy correspondence with the State Committee, we came to the conclusion that there was a third option, unforeseen by me before. My "adversaries" wrote that my proofs were inconsistent, that those were only my suppositions, but would never mention WHAT was inconclusive and what my mistake was.

The biggest paradox was their contradictory commentaries, stating that this phenomenon had been known for a very long time, proven by someone else. At the same time, no references were made to the researcher who "had already discovered" this phenomenon.

The absurdity of their position was obvious: they would simultaneously affirm that this phenomenon had long been documented and that it was only an unbased supposition of mine. Later, I have decided to request an explication from the administration of the above-mentioned institution.

The historical meeting (for me) took place on Cherkassky street. I had been kindly invited by comrade N.N., who had

confidentially shown me the originals of the negative references I had received, with the signatures of the respective authors that had been hidden from me prior to that.

Among those signatures were some of the most notorious names in dentistry of the respective period. Delicately, so as to not humiliate my status as a professor, he explained to me some elementary things, obvious to all of us. I simply didn't fit into the general situation. Our first mistake was to include the names of my PhD students as co-authors. Instead, we should have invited some of the previously mentioned notorious people. Then, everything would have clicked into place. Everything would have been clear and the researchers wouldn't have had to sign ridiculous and contradictory negative references and the "priority of Soviet science" would have once again proven its notoriety from a judicial standpoint. Both science and practice would have gained something, etc, etc... That was it. I should add that N.N. listened to my arguments, and revising the letters, he explained to me that as an individual, he was completely on my side and he was baffled at the despicable way in which conflicts were settled in dentistry, where such absurd declarations and resolutions came out. However, his opinion is private, the rules require only the resolutions of the "deities" to be taken into consideration, and each department of science has its own "deities".

As we know, history does not accept the term "I would have...", and looking back, I am glad I did not listen to comrade's N.N. hints. Today, it is obvious that the problem did not solely lie in the human weaknesses of the pinnacle of the dentistry community of that time, the actual situation was much more serious and fundamental.

The responsible professionals could not and still cannot recognise the veracity and the importance of my arguments, and surely they cannot become supporters of the "dead" theory overnight. These colleagues, being loyal supporters of the

concept of upright self-sufficient practice, cannot betray it. The current practice, cannot help defending itself from different inappropriate theories, regardless of how logical they may sound. Millions of dentists are preoccupied with real, actual work, not theory. Dentistry protects man's gentle masticatory apparatus from microbial decay. The multibillion financial contributions maintain the respective social mechanism that guarantees the population a satisfactory denture.

I am being opposed not only by egoistical individuals, but rather by the whole, immense system called dental "practice". It is important to note that global dentistry, is NOT only helpless, but rather the opposite: it is extremely powerful even though it relies on an unintelligible pragmatic surrogate.

Here, medical practice is tied to a large logistics network, which includes a large variety of specialists and highly skilled workers. All of them are preoccupied by an important and serious practical issue that has no need of theories exploring its origin, which is seen as an axiom. In any case, this issue is none of their concern. They are satisfied with having a constant flow of difficult practical problems that require their skills, energy and devotion.

Fundamental theory is a completely different field of work that attempts to understand the essence of all the phenomenona occuring in the world (including the dental problems of the civilisation). This belongs to fundamentally different professionals who possess a different type of logic, conceptual apparatus, maybe even different brains and consequently they are driven by a different set of values. The result of their work comprises smaller or more significant scientific facts, models and phenomena that were unknown before, concepts that are abstract by nature - they cannot be touched, nor sold or bought, because they have no price. The importance of the said results is judged by whether or not they correspond to reality and in no case by their practical applications. Theory is dead, in the

sense that it remains enclosed in information storage mediums for an undefined period of time.

Modern dental "practice" only exists (and flourishes even) thanks to the current epidemiological situation. It sees "prophylaxis" as an important source of income through the distribution of services and facilities, but not as a mean to eliminate epidemics, an idea in which society so hopelessly believes.

The "theory" suggested by my adepts is unfit for modern "practice". Perhaps some of its details could prove some worth, albeit not conceptually. This "theory" is vital to an entirely different type of "practice", one which will meet the society's expectations involving dentistry's triumph over diseases, akin to how medicine has triumphed over numerous infectious or metabolic diseases. A "pragmatic" practice devoid of "theory" is incapable of such deeds. In order to convince ourselves that such is the case, we have to look no further than the last decade's multimillion dollar "natural experiments". As it turned out, fluoride, albeit theoretically unjustified, is the only veritable method of caries prophylaxis. However, it is well known that efficient doses of fluoride also act as teratogens, being a threat to overall health. Dental fluorosis is nothing but a marker for fluoride poisoning. The theoretical "insolvency" of scientific concepts is the underlying cause behind dentistry's lack of epidemiological control over its biggest diseases. The public has not discovered it yet, but their discontent and confusion with dentistry's powerlessness is rising. In the era of some of the most ambitious scientific discoveries, dentistry's incapacity to stop a pandemic and microbial-dependent disease in modern, educated humans, could do nothing but make them smile sarcastically. In such context, the emergence of new and successful approaches and concepts is inevitable. At this very moment, we can say no more about these new unexpected ideas that after a certain confrontation with the old status quo,

will eventually shape the practice that will transform today's dental conditions into some rare, banal diseases.

We could safely say that this bright future (for patients) will be inevitably based upon scientific knowledge, upon the "theory" describing the normal processes occurring within the normal human odonton (the functional complex encompassing the tooth, the periodontium and the corresponding segment of the alveolar bone). Once a certain fact is discovered and becomes part of science, it will go on to form the theoretical base of preventive dentistry, the knowledge of this fact being impossible to erase. Today, this knowledge is scarce, but it will persist forever. Not a single Commission or Mount Olympus of the scientific community have the power to erase it.

The laws behind these processes are absolute and require a respectful attitude from the professionals in the field who were tasked by society to preserve them. It would also be absurd to think of a future in which the future dentist would learn the aforementioned secrets from third parties such as the Internet or popular literature. If we are tasked with protecting the safety of teeth (i.e. the processes taking place within it), it could only happen by understanding and inserting the relevant concepts into the collective consciousness of the professional community.

In the following paragraphs, I would like to outline what can already be inserted into this consciousness without leaving room for doubt.

1. The dental apparatus (in human's masticatory apparatus) is formed by odontons, which serve as its functional units. The odonton includes three functional elements: the tooth (the element subjected to wear out), the alveolar bone (supporting structure and recipient of mechanical pressure) and the periodontium (the ligament, a hydraulic damper). The odonton develops within the crypt of the tooth germ.

2. The main biological process during the tooth's entire ontogenesis is the centrifugal transport of tissular fluid through the hard dental tissues. The said process, a result of the pulp's "pumping" function, ensures the delivery of mineralisation ingredients through the tissues subjected to mechanical stress, abrasion and microbial invasion to the location where these ingredients are being utilised. This process is coordinated by the organism's neurohormonal regulatory mechanisms.

3. A disturbance in the activity of the said mechanism, caused by various systemic "plurietiological" circumstances, drastically reduces the resistance of the transport pores found within the enamel and the periodontium, initiating the corresponding microbial diseases. Contrarily, the ensemble of conditions contributing to the development of an efficient functional resistance of the odonton, bestows it with good health and longevity on it, traits shared by all the species living in the wilderness.

4. The scientific stances outlined above clearly indicate towards the fact that the modern specialist should learn the answers to the following questions from the very beginning, i.e. from the simulation courses:

- Why should we strive to maintain the vitality of the pulp?
- Why is it necessary to forget about the "Black Boxes" that block the transport ways of the enamel and dentine and rather take into consideration the topography of the dental tissues during the preparation of the carious cavity?
- Why sometimes, in order to prevent various diseases of the odonton, the devitalisation of teeth becomes acceptable, adequate even?
- Why is it possible to stimulate the remineralisation of the enamel (in hyperplasia and white spot lesion) through the usage of hypertonic substances that do not contain calcium, phosphorus and fluoride?

Most importantly, even in its shortened form, this information on the clinical physiology of the tooth could be useful in preparing the future specialist to take an active part in controlling the dental diseases. Such monitoring, based on actual scientific facts, will inevitably transform into some type of predictive, personalised prevention. This is the task of the coming decades. In such manner, the data provided by the now defunct theory will be transposed into the daily practice, representing an inevitable and long awaited symbiosis between the interests of the dentists and their patients. Let us be frank: nothing is more practical than a good theoretical framework.

V. Okushko

1. The Biological Role of Dental Tissues

1.1. The Tooth and Its Skeleton

In anatomy classes or during simulation courses, what we hold in our hands is merely the skeleton of the tooth. At first sight, it differs very little from the actual organ. However, the crucial difference lies within the tooth itself, in its soft tissues, and namely in the dental pulp (in the bone marrow, if we were to make an analogy). Besides the pulp, which also stands out in a living tooth, are its radicular layers - the periodontium. It is something analogical to the periosteum, although they are obviously different. The basic function of the periodontium is to ensure the link between the tooth and the dental alveolus. First and foremost, we have the dento-alveolar ligament, a locking mechanism, through which, akin to the periosteum, pass various blood vessels and nerves, penetrating the dental tissues through special orifices and thus feeding them. The said fascicles are the most prominent at the tips of the roots- the apical foramina. In their immediate proximity lie smaller links - the deltoid ramifications. Another region in which one can find numerous orifices is the furcation region in pluriradicular teeth.

All of the above are transporting structures through which the arterial blood is delivered, the venous blood is evacuated and the nervous signals go back and forth. Thanks to this transport of energetic and construction resources, the tooth continues its formation even after the eruption and its tissues are continuously developing. The main consumers of the said resources are the specific/distinctive dental cells- the odontoblasts. Comparing the tooth to the bone as an organ, we should note that the marrow of

the latter is the hematopoietic organ of the body. Three groups of cells ensure the viability of the bone: constructive - osteoblasts, killer cells - osteoclasts and the supporting cells - the osteocytes. Their permanent functional "confrontation" allows the bone to reconstruct itself during the entire lifetime.

One can encounter this life giving "confrontation" in any osseous region of the body.

Within the morphology of bony tissues, these three elements look like some sort of puzzle, crystallising, at the same time, the image of an aging and decaying region, and the image of a young and developing region of the bone. Thanks to them, in case of a fracture, the bone consolidates itself perfectly and if, by a happy occurrence, the periosteum is unharmed, the bone regenerates itself completely. The former statements refer to any bony organ besides the tooth. Here, a single layer of cells carries out two functions: of formation and support. Teeth cannot, by any means, destroy an already existent tissue, being, from an evolutionary standpoint, inferior to the other osseous tissues, lacking that **perfect "Trinity".**

Within the tooth, the confrontation between formation and annihilation manifests itself rather plainly. The external environment damages the dental tissues, and the internal environment, represented by the pulp and partially by the periodont, builds the new tissues from within, or fills them up.

We all know that the vital centre, or the heart of the tooth, is nothing else but the pulp. It should be emphasized that the analogy between the pulp and the heart is much more than a metaphor, everything coming down to the energy flux both of these use in order to function properly. The energy used by the pulp (the dental pump) ensures the viability of this small mineralized organ, ensuring the hydraulic support needed by the hard tissues.

The enamel and the dentine are penetrated by transport structures filled with tissular liquid. The latter, being under

pressure, moves centrifugally and comes out in the oral cavity through pores. The said liquid makes up 20% of the dentine's volume. It moves through the dentinal tubules up to the enamel-dentine junction. These ducts are radially distributed from the pulp up to the peripheries. Starting from the said junction, the liquid (making up 10% of the enamel's volume) moves through a series of fissures (meridians) and ducts. The liquid found in the enamel is a solution of different salts characteristic of the intercellular liquid with a wide spectrum of amino acids, trails of fatty acids and saccharides. It moves at a speed of around 1 mm per second.

We have already outlined that inside the tooth, it is the periodontium that plays the role of the periosteum, thus becoming a dento alveolar ligament. Just like the periosteum, it does not only have fascicles of blood vessels and nerves which penetrate the tooth, but it is also capabile of enhancing the length of a tooth by stratification of cement layers. The secondary cellular cement is formed by it overlapping the primary, acellular cement, being produced by cementocites- some cells that remind us of osteocites. The cementocites form layers similar to osseous layers, inside of which they then remain engulfed. The growth of secondary cement layers is frequent near furcations or in the apical zones. As a result, the radicular part gets longer, thus compensating for the loss of dental layers through abrasion. Thus, the wear of the external dental layers is foreseen by genetic programming and is accompanied by a compensatory increase in volume of the dental tissues through the formation of secondary dentine inside the pulp's chamber and of secondary cement on the radicular surface.

Everything mentioned above is indicative of the fact that the tooth as an organ and all its tissues represent a unitary biological system that performs specific functions, necessary to the organism. Therefore, we have absolutely no reason to classify it as something else, as a "skeleton organ".

Something entirely different is the difference between the tooth and the bone when both are seen as organs. The skeleton-tooth, described in osteology as part of the human skeleton, has very little to do with it. Obviously, they resemble each other by their chemical composition and the character of its crystals similar to hydroxylapatite. However, from a biological standpoint, according to the formation history (phylogenesis) and the development history (ontogenesis), these parts of the skeleton are completely different. Moreover, it would be correct to say that vertebrates have two skeletons- an external and an internal one.

The internal one, used for support, is rather large and represented by numerous bones tied together by through joints. ***The external one***, used for protection, is represented by a modest number of small formations that are not tied to each other- the teeth. The internal skeleton is much younger and more progressive, being significantly younger from an evolutionary standpoint, having a wide array of possibilities to adapt and restructure depending on the constantly changing conditions of the environment. The external one is extremely old, primitive, simple and unable to change. It cannot adapt or reform, the only thing it can do is to add new layers on top of each other if needed and upon finishing its mission it simply leaves the organism.

The internal skeleton develops from mezenchyme, the external one- solely with the implication of epithelial layers- the ameloblasts. The bone building cells (osteoblasts) keep their viability inside the bone tissue. The enamel building cells (ameloblasts) die and the dentine building cells (odontoblasts) leave as soon as they have formed a new tissue.

The enamel, dentine and the primary cement, in comparison to other bones, do not contain any cells. The tissular microstructure is unchanged and is directed from the centre, which contains cells, to the acellular periphery.

In case of fractures, the components of the internal skeleton are able to regenerate completely. Dental tissues do not regenerate and in the created fissures such processes as adhesion and restitution take place, trying to repair the damage.

The internal skeleton, from the very moment of its formation and during its entire lifetime, undergoes countless changes, adjusts to the environmental conditions. The external one, except when factors of brutal nature are involved, develops strictly according to a genetic programme, recreating the smallest details of the relief and channels, which are characteristic only of the respective individual and their univitelin twin. Thanks to this unique relief and network of channels, it is fairly easy to determine family bonds, appurtenance to a certain race, etc. Odontoglyphy, akin to dermatoglyphy, is a science that enables us to identify a preson by the relief of their dental crown. This relief does not change, does not regenerate, nor can it be destroyed.

All the elements of the internal skeleton comply with systemic effects. For example, the excess of paratireocrine (the hormone of the parotid gland) leads to a dangerous disease which manifests itself through the demineralization of all the bones of the internal skeleton. On the other hand, the external skeleton always preserves its outlines, being immune to such diseases.

2. The Tooth as a Biological System

The overloading of tissues that support the tooth is prevented through the creation and maintenance of the hardness gradient inside the hard tissues. Thus, the biggest mechanical resistance belongs to the enamel, which is capable tofo withstanding pressures of up to hundreds of kilograms per square millimetre, whereas the periodontium has the smallest resistance, being essentially a hydraulic damper.

2.1. The Regulatory Mechanisms of the Tooth

The tooth and its ligament (periodontium) are capable (thanks to the respective receptors) of perceiving different irritations and transform them into centripetal nerve impulses. The enamel and the dentine are "penetrable" for these signals. Within them, there are informational channels capable of sending signals, which are then received by receptors located near the odontoblasts. These signals, upon getting to the central analysers within the cortex, are processed and transformed into subjective sensations: pain, roughness, discomfort, etc. Besides this, in these respective centres imperative efferent signals also form, especially vegetative ones, capable of leading to vascular reactions, which, in turn, change the degree to which dental tissues are provided with energetic and plastic resources, necessary to sustain the life of dental tissues. Both these fundamental functions of the odontoblasts (construction and pumping) are hormonally regulated. It is well known that parotin (the secretion of the parotid glands), which is secreted as a response to the releasing-factor of the hypothalamus, activates the work of the dental pump and increases the speed at which liquid moves through dental tissues.

2.2. The Gradient of Intertissular Hardness

The gradient of mechanical hardness within the structure of hard tissues appears during the process of their development and formation. In accordance with its biological mission, the tooth, like any other destructive weapon, has a respective functional unit. Comparable to the edge of a sword, nature has created the sharp edge of an incisive out of the most durable material, highly capable of destruction while remaining almost intact. Other zones of the tooth are not nearly as hard. The gradient of hardness is the essential rule of tooth formation. If we were to ignore anatomy and sort the tissues in a descending order by their perception of mechanical force, the order would be: enamel, dentine, cement.

Dentine – the main dental component by mass and volume is the natural support that takes upon itself all the pressure exercised upon the enamel and distributes it evenly on the whole surface oriented towards the alveolar walls. This tissue, although not as hard as the enamel, is more elastic. Its hardness is less than 100kg/ $mm^{2.}$

The periodontium works as a hydraulic damper thanks to the liquid component. In the cement, the fibres stay, they ensure the tooth's stabilization in the alveoli and constitute the base of this tissue. According to Pascal's law, the masticatory force decreases considerably in the periodontal tissue and is evenly distributed upon the bone and its medullar spaces.

This way, the mechanical properties of hard dental tissues, thanks to the hardness gradient ensures a considerable decrease of the force coming from the **enamel** (the main destructive tissue) all the way to the periodontium (the hydraulic damper). Anatomically, these three tissues surround the core of the tooth – the pulp, which does not have a substantial mechanical role, creating the impression that it is being left out, but at the same time it has an essential role in ensuring the viability of this organ.

The intertissullar hardness gradient is physico-morhological feature of the tooth. These mechanical characteristics are genetically predetermined and are possessed by the said tissues from the very moment of their formation. However, this gradient is typical of every tissue in part as well. Even more so, it is variable. In a normal tooth, alive and functional, the indices grow. In the absence of the pulp, however, they diminish. This variability of tissues is possible due to the processes taking place in the transporting structures of the tooth. They ensure the shortest path to deliver the nutrients necessary to improve the tissues from the odontoblastic layer found in any zone of the enamel or dentine.

The dentinal canaliculi penetrate the layers of the dentine and they end in the enamel-dentine junction. From these main paths and along their entire length, come out almost perpendicular ramifications that penetrate the entire volume of the tissue.

From the enamel-dentine junction all the way to the surface of the enamel, throughout its entire thickness, there are organic plates, the totality of which forms a meridian and microcanaliculi, the point of their highest concentration being located on the summit of the cusps. Depending on the direction of these sections, these formations take the form of either blades or fascicles. Through them, towards the external pores of the enamel, the liquid moves ensuring the variability of the hard structures.

3. The Enamel – an Endogenously Controlled Specific Dental Tissue

The enamel's primordial biological mission, as a functional tissue of the tooth, is to exercise a destructive mechanical action upon objects of the external environment. Moreover, it also aims at appreciating the mechanical characteristics of the said objects.

The enamel possesses these qualities due to its mineral component, represented primarily by crystals similar to hydroxylapatite, comprising 96-97% of live weight.

On the surface, the tissues are permanently mineralised, having a more homogenous structure. They are less porous and, respectively, harder. Conversely, the deeper layers, near the enamel-dentine junction, are more porous and softer.

Being an acellular tissue, the enamel is incapable of using or generating energy by itself, and neither does it have the capacity to make such decisions as energy management. All of these are

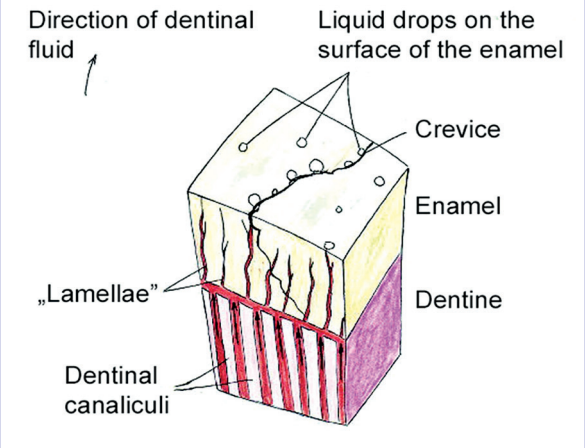

Fig. 1. *The volumetric deplascement of the dental fluid. (by G. Bergman)*

taken care of by the pulp – the tooth's core. The gathering of information and the utilisation of energy take place in the enamel itself; however, these processes are managed by the pulp. Now, let us explain thoroughly these two processes.

The enamel obviously has the capacity to perceive external irritations, be they distinguishable or not (below the distinction threshold). The possibility to perceive these irritations is owed to the presence of water in the enamel. It represents a filtrate of the intercellular liquid of the odontoblasts, which contains a whole package of ions, organic components (mainly amino acids). Besides these, it also contains polysaccharides and fatty acids. The mass of the liquid makes up 1.5-3.0 % , but its volume makes up to 15%. Ergo, we can affirm that the adamantial liquid is the second component of the enamel by volume (after the minerals).

Through a simple experiment, we can prove the veracity of the previous statements. If we dry up a living tooth under a stream of hot air, trying to eliminate by evaporation all the water found inside the enamel, the subject will complain about a sensation of swelling. The enamel in the dried up zone will lose its usual colour and take up a gray hue. In this dehydrated state, isolated from their natural transport pathways, the tissues lose the ability to perceive external irritations. They pick up neither the mechanical irritants nor the electrical or chemical ones. The sensations will not return even if the surface of the enamel will be slightly damped. The enamel will be temporarily excluded from the sphere of biological control. Only after a couple of minutes, when the integrity of the liquid column and the centrifugal flow will be re-established, will the informational link between the enamel's surface and the pulp be restored, i.e. everything will turn back to normal.

Thus, the enamel undoubtedly has the property of perceiving external irritants. This exchange of information takes place through the inter tissullar liquid, incorporated in the walls of the

transport pathways. As mentioned above, inside the enamel and the dentine a centripetal movement of the liquid column takes place. This proves the existence of a pressure difference between the internal and external layers of hard tissues. The integrity and continuity of the said process proves the existence of a permanent energy flow which maintains the pressure difference.

The experimental study concerning the enamel's microhardness under functional load was conducted by V. R. Okushko and A. V. Melehin (1971-1981). The authors have determined that the enamel's surface subjected to constant abrasion has the highest hardness of the whole structure. The analysed data confirms that the previously listed regions can withstand pressures of up to 5000 atmospheres more than the unaffected symmetrical regions. In addition, the functional regions have a more defined microhardness gradient, i.e. the difference between the external and internal layers, the latter being softer than the external layers. The results of measuring the volume of radial organic microstructures of the enamel and dentine (A. V. Melehin, 1978) have determined a significant expansion of the microvascular bed under the influence of functional load. Therefore, the quoted work mentions that the volume occupied by dentinal canaliculi grows up to 1.5 times larger on average, but the morphometric appreciation of the component of fascicles, plates and fusiform formations becomes several times larger.

The enamel's fascicles can be compared to the "empty" prisms of marsupials. Transforming into leaf or strip shaped ramifications, these structures slowly "get lost" into the interprismal regions, moving distally. (Schumacher, 1972).

The admantial plates and blades differ from the fascicles by their bigger length, sometimes reaching the external layers of the enamel. The limits of these structures are much clearer near the enamel-dentine junction. Akin to the fascicles, they form ramifications, but the latter have a well defined outline.

The elements of the described system manifest themselves in six different morphological landscapes. First- the scalloped enamel-dentine junction. Second- slit shaped parallel formation, visible on the internal surface of the enamel. Third- adamantial fascicles. Fourth- adamantial blades. Fifth- the linear trajectories of moving liquid outside the visible structures. The sixth is the terminal submicroscopic branching, through which the liquid penetrates the enamel's whole thickness.

The human tooth enamel has a strictly regulated transporting system which penetrates its whole thickness from the enamel-dentine junction all the way to the superficial layers of the enamel.

The enamel is divided into three parts:

- The superficial layer – considered the densest among all the layers of the masticatory surface
- The middle layer – has ultraporous properties, the nodular fibrous chains forming "arcades"
- The basal layer – has microporous properties thanks to fibres made of connective tissue.

4. The Tooth's Functional Structure

It is well known that different types of connective tissues serve as support axes for all the organs and systems in the human body. The tooth's functional structure - the enamel, is supported by two types of tissue: mineralised (dentine) and lax fibrous tissue (the pulp). The delimitation between these two structures is conditional, as the tooth's specific cells (the odontoblasts) belong to the pulp, but their apophysis are found in the dentine, tending towards the enamel. These apophyses occupy less than half the distance between the pulp and the enamel-dentine junction. For the most part, dentine is an acellular tissue. Moreover, the dentinal canaliculi formed as a result of the odontoblasts' activity, and the dentinal liquid are, de facto, their intercellular environment. Thus, the demarcation line between the pulp and the dentine can only be made anatomically, at a macrostrucutural level. From a histological standpoint (micro structural level), such a delimitation cannot be made.

The "pulp-dentine" complex is a unique one, made of connective tissue (the enamel's infrastructure), in which the functions of central and peripheral layers are clearly separated. These are included into the tooth's main tissue- the dentine. It is a mineralised tissue and performs the crucial mission of providing mechanical support. Its layers, as it had been stated earlier, are penetrated by canalicular tubes- transporting structures that connect all the acellular mineralised tissues of the tooth into one single mechanism. The liquid filling the said transporting network (dentinal and adamantial liquid) is produced by the pulp- the central trophic component of the fibrous tissue complex.

The chemical, physico-chemical, hydrodynamic and rheological parameters of the liquid ensure the viability of hard tissues, determining their possibilities of suitable biological

variability. In this case, the pulp generates a life-supporting flow and it is up to the dentine to guide it to the enamel. Therefore, the dentine is an intermediary multifunctional link, ensuring support and transport. The dentine lets the information flow, it also lets the energetic and plastic resources pass through itself on their way to the enamel, while the enamel sends back mechanical forces. The mechanical pressure is transposed upon the dentine's mineral frame, whose mechanical properties depend upon its mineral components, akin to the bones. The dentine's mineral components make up rougly 70% of its mass, the other 30% being organic matter and water.

The dentinal liquid, found preponderantly in the respective canaliculi, represents, in fact, the intercellular environment of the odontoblastic layer. From a biomechanical point of view, it ensures the elasticity of tissues, just like adamantial liquid.

We should note that the enamel is a massif, its thickness reaching figures of up to 1.5-2.0 mm, whereas the radicular cementum is only a couple of hundredths of a millimetre thin. Respectively, the canaliculi are more common and more tightly packed in the coronary region, especially in around the projections of cusps. Here, their numbers reach hundreds of thousands per square millimetre of section. In the radicualr regions, their number is considerably smaller, 3-4 times smaller (25-30 thousands on the same surface area). The dentinal canaliculi are really some canaliculi with an own wall- the Neumann membrane. They penetrate without any discontinuance the whole thickness of the tissue. Their diameter is measured in microns and their length in thousands of microns. These are extremely elongated extracellular formations.

The dentinal canaliculi, for the most part, are isolated from the dentine's mineral massif and thanks to this fact they can transport their contents all the way to the enamel-dentine junction. The said content leaves there its large molecules and heads to the enamel through the ducts. As to the cementum,

neither the existence nor the function of cementar liquid have been proven. However, taking into consideration the structural identity of odontoblasts in the canaliculi, we can consider that the situation there is similar. The tube isolated by dentine, which opens up into the Tomes granular layer fulfils the same role of transportation.

Thus, the physiological attributes of canaliculi are obvious. These long and thin formations tie every odontoblast with a topographically distanced region, and yet those regions have unique physiological responsibilities within the acellular tissues (enamel or dentine). As mentioned earlier, the cellular layer of odondoblasts with its apophyses, penetrates the dentine through the dentinal canaliculi. However, the depth of the said penetration constitutes about one third of an apophysis' length. This way, dentine is still mostly made up of acellular mass. Nevertheless, the presence of odontontoblastic apophyses and intracellular liquid in the dentinal canaliculi enables the dentine to transform. The dentine is topographically and functionally closer to the pulp and, therefore, in comparison with the enamel, this tissue is much more variable, this statement referring to both its composition and its biophysical parameters.

The dentine is constantly restructuring and this occurs, first of all, thanks to the odondoblasts' ability to produce predentine- a weakly mineralised organic base that will become the future, well mineralised tissue called secondary dentine.

In interglobular dentine, the globules- the epitaxic crystallisation centres are represented by mucopolysaccharides of obvious odontoblastic origin. The speed at which dentine is forming and the centripetal movement of the mineralisation front are influenced by systemic and local stimuli, including those coming from the enamel. In case of different changes in the formation of dentine, both the previously mentioned phenomena change parallels. (Baume 1970, Jenkins 1978, Hansson 1978).

Another type of dentine variability, caused by the functional wear of its tissues, is ensured by its ability to form a so-called transparent dentine. Whether it happens under normal or pathological circumstances, the loss of enamel will eventually uncover its foundation, the enamel-dentine junction. The enamel disappears in these regions. The space that it previously occupied is now empty, being "seized" by the oral environment. Thus, the subject of the odontoblasts' control no longer exists, rendering the biological mission of apophyses and dentinal canaliculi, which ensured the link between the odontoblasts and the mineral massif, non-existent. Both the canaliculi, with their strict orientation towards the previously controlled subject and the Nasmyth membrane disappear. They are utterly obliterated, being filled up with a mineralised mass.

The dentine in the said region turns into a dense mass, homogenously solidified and transparent, in which the concentration of osteotropic elements grows considerably, rendering it twice as hard. If initially its microhardness was about 40-50 kg/mm^2 ,then in the transparent tissue, the same index grows to about 70 kg/mm^2 or even more. It is as though this new tissue is trying to compensate for the lost functionality of the consumed enamel. It is important to note that this remodelling of dentine, accompanied by the abolition of the old ductal structure, does in no way affect the tissue's property to grow and enhance its volume towards the pulp' chamber.

The process continues just as actively and productively, except the fact that the newly formed and cumulated tertiary dentine acquires a completely new form. Due to it not being needed anymore, the ductal system oriented towards the enamel is absent, after all, the enamel does not exist anymore. The number of canaliculi is insignificant, as they get smaller, narrower and take the form of a clew. From this fact originates another name for the said tissue – "irregular dentine". Inside this replacement tissue, one can often find blood vessels, hence its other name "vascular dentine".

5. The Pulp as a Life Support Centre for the Dental Tissues

It is doubtless that the pulp's biological role to ensure that the hard dental tissues have everything they need and to increase their resistance to the strong mechanical forces generated during certain activities of vital importance.

It is within the pulp (the soft dental tissue) that the nerve impulses are perceived or generated, energy is released, the pressure gradient in the hard tissues' hydrodynamic system is maintained and finally, it is here that the conditions needed for the "dental pump" to become operational are created. Initially, the latter provides the respective structures with products necessary to their formation (building material) and afterwards with products needed to maintain them, i.e. recovering the properties of hard tissues. In other words, we can affirm that if the enamel and its properties are a generalisation of a tooth's biological predestination, then the pulp is a generalisation of everything that makes the former possible. It is from here that the organ draws its informational, energetic, structural and plastic resources.

Morphologically, the pulp is a lax, fibrous connective tissue that fills up the tooth's cavity. It consists of:

- Main substance (fundamental)
- Cellular zone
- Vascular zone
- Fibrous zone
- Nervous zone

The fundamental substance of the pulpar organ is the connecting link between all of its components. Mucopolysaccharides, mucoproteids, hexosamines, glycoproteins, chondroitin

sulphate and hyaluronic acid have played a great role in its formation. If its volume increases, the dental tissues become more susceptible to attacks from microorganisms.

The cellular zone is equipped with different types of cells: odontoblasts, fibroblasts, histiocytes, undifferentiated mesenchymal cells and stem cells.

The vascular zone encompasses lymphatic vessels, veins, arteries and arterioles, all of which penetrate the depths of the pulp through the apical orifice.

The fibrous zone is comprised of collagen, reticular and argirophile fibres. Anatomically, a great deal of these fibres are situated in the apical zone and less in the coronary zone, where they form fascicles.

The nervous zone – through the apical orifice, nervous fibres penetrate the pulp all the way to the crown, forming a network. The main role is played by the myelinic nerve cells, which form the Rashkov plexus that is responsible of pain perception.

The odontoblasts' role in the formation of dentine causes the permanent displacement of their cell bodies towards the pulp's centre and the elongation of canaliculi. As a result of this displacement, the odontoblasts become more tightly packed which leads to the physiological atrophy of some cells.

The odontoblasts' spontaneous activity undeniably has a cyclical character. They are cells of secretory activity, whereas their apophyses perform a transport function.

The information from literary sources concerning the functions of odontoblasts and their organs is basically limited to their plastic function of dentine formation, referring to the synthesis of collagen and the respective fibres, the formation of dentine and its mineralisation (Carneiro, Leblond, 1959).

The multilayered positioning of odontoblasts in the coronary part must be seen as an important characteristic of the said cells (approximately 6 layers in the pulpar horns and a single

layer at the roots).. In this particular zone, the volume of the cells pressed to the pulpar walls is proportional to that of the enamel projected on a certain sector. This fact, undoubtedly can be considered as another proof of the existence of a functional link between the odontoblasts and the enamel. The formation of a multicellular layer of odontoblats is biologically relevant, because it is logically associated with massive layers of enamel subjected to functional abrasion.

The dental pulp (or rather its predecessor- the dental papilla) commences its biological mission before the emergence of the tooth itself. From an energetic and plastic point of view, the tooth starts out from here. As for the epithelial layers of the enamel organ, they keep their coating properties upon encapsulating themselves in the mesenchymal layers. This temporary sack "organ" does not allow the implementation of other cells throughout its whole lifespan. And the fact that by the end this sack is filled with the most mineralised tissue in the entire organism is thanks to the dental papilla, the pulp being non-existent at this point. The so called "pulp" of the enamel organ is something opposite to a true soft dental tissue. It is merely an epithelial padding, an extension of buccal epithelial layer.

There are neither arterial nor venous vessels in it.

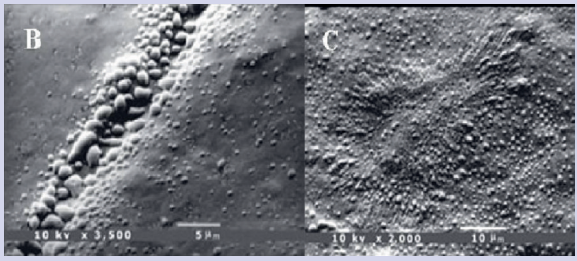

Fig. 2. Drops of liquid on the enamel's surface (A. Bertacci).
B – the fissure at the surface of the enamel, surrounded by drops of liquid;
C – drops of liquid on the enamel's surface

Only in the papilla do the blood vessels actively ramify, forming a compact ball. The fenestration (microperforation) of capillaries indicates upon the main direction in which the necessary objects are transported by the blood, that direction being the first generation of odontoblasts. Everything the tooth will be built from is brought by the blood from the vascular clew that grows together with the organ. Moreover, all the energy consumed to build, sustain and maintain in biological balance all the cellular components and intercellular substances, comes from the respective blood network through a constant flow. If we were to interrupt this flow, deteriorating the odontoblast layer and the vascular system that feeds it, it would have immediate consequences upon the function of enameloblats- the enamel builders. The enamel from the respective place will irremediably change its regularity and will get deformed, which will lead to a local form of severe hypoplasia. Under normal circumstances, the interstitial fluid is transported through the whole thickness of the dentine and enamel, later appearing on the enamel's surface as drops of liquid (A. Bertacci, 2009).

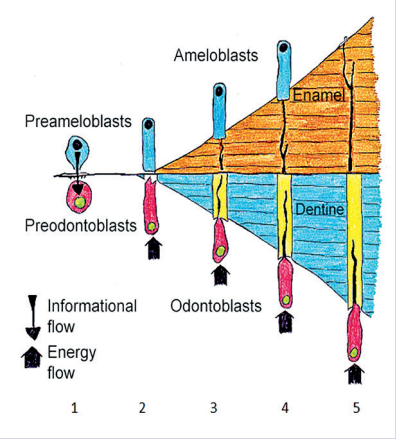

Fig. 3. ***Functional stages of the formation of dental tissues:***

1 ***– informational contact;***

2 ***– the emergence of builder cells (ameloblasts and odontoblasts);***

3 și 4 ***– the formation of enamel and dentine and their stratified structures***

5 ***– the completion of amelogenesis.***

The answer to the question concerning the possibility of controlling the movement of the fluid from the pulp has been discovered after a series of studies in which the authors used the phenomenon known as electroosmosis as a condition to increase the centrifugal liquid flow. If the enamel is connected to a DC network, after a couple of seconds some visually noticeable drops of liquid will emerge on its surface (at the cathode). It had been concluded that in devitalized teeth, the enamel remains unchanged for about 15 minutes, whereas in a living tooth, the current's intensity increases after 2-3 minutes and reaches its peak after 7-10 minutes, when drops of water emerge on the enamel's surface, at the cathode, proving that the changes produced by denervation have an immediate and considerable effect upon the enamel's permeability. This tells us about the increase of centripetal permeability within the hard dental tissues when the pulp is stimulated with an osmotically active substance.

The sectioning of the inferior alveolar nerve, just like the sectioning of the neuro-vascular plexus or the disarticulation of the mandible, leads to an immediate increase in electrical bioactivity, gradually reaching its peak in 1-2 minutes. During the slow fluctuations of electric potential, with a frequency of 10-12 fluctuations per minute and an amplitude of 30-40 mkV, there occur fluctuations of considerable amplitude (up to 100 mkV) with a duration of 0.5-0.8 seconds. This way, the conditions leading to the enamel's centrifugal permeability (Novocaine blockade, the sectioning of the inferior alveolar nerve and the isolation of the mandible) also enhance the pulp's electric activity.

The stimulation of the inferior alveolar nerve with electrical impulses reduces the enamel's permeability and the amplitude of the slow fluctuations of the spontaneous electric activity and it also reduces the amplitude of the rapid potentials.

The pulp's resting potential, its electrical activity and the centrifugal permeability of hard dental tissues are closely tied. The lower its resting potential is, the higher its electrical activity, the electroconductibility of hard dental tissues and the enamel's centrifugal permeability are.

The fact that the change in the electrical activity of the odontoblastic layer and the change of the resting potential take place immediately after exposure, anticipating the change in the enamel's centrifugal permeability, tells us that in this complex, the electrical activity of the pulp's cells is of primary importance.

6. Tooth Development and Eruption

The development and formation of the tooth is divided schematically into three phases: primordial, differentiation of cells (histo-differentiation) and formation of dental tissues (histogenesis). These stages are well described in any histology textbook.

A tooth begins to form at the end of the embryonic period, corresponding to the seven-week age of embryonic life. During this period, on the edges of the primary buccal cavity, the buccal epithelium stratifies, forming a fold that sinks into the thickness of the mesenchymal tissue. The first stage of tooth formation is determined by the separation of the future primary tooth from the vestibular side of the epithelial plate, to which it is connected through an epithelial cord called "*gubernaculum dentis*".

At this stage, we have three morphological characteristics: the enamel epithelial organ (epithelial cells), the dental papilla and the dental sac (mesenchymal cells).

The sinking of the epithelial cells into the thickness of the mesenchyme implies the employment of the main energy resources. Once embedded under the epithelium layer, the future shield builders begin a completely different life. Every guest cell - the ameloblasts (the enamel constructor) - finds a pair of nourishing cells from the „local" ones. The couple exchanges chemical signals - growth factors and start the creative work uncharacteristic for their parents. The histogenesis starting signal (formation of tissues) comes from the epithelial cells in the form of a chemical compound belonging to the tooth growth factors group - TGF (tooth growth factor). This imperative information is perceived by the preodontoblasts which are forced to transform into odontoblasts - mature post-mitotic tooth cells (which do not divide).

From that moment onwards, the formation of predentine and dentine starts. At the same time, the dental canals start to evolve and initially provide communication between the forming pulp and the enamel. In addition to the construction activity, the odontoblasts facilitate the creation and preservation of the pressure difference, thanks to which the necessary energetic and plastic ingredients are brought to the ameloblasts through the liquid in a centrifugal motion. Ameloblasts – flow consumers also lose their natural ability to replicate. Instead, these ones have a longer lifespan and use the energy and materials brought to build the enamel.

The fatal encounter point of epithelial and mesenchymal cells is the moment when the first „foundation stones" of the hard tissues (enamel and dentine) are laid. After that, the epithelial cells (ameloblasts) move from the meeting point to the sister cells, to the „roof". The odontoblast nurse-cell (the tooth builder) works synchronously and symmetrically with the ameloblasts. It also withdraws, but in the opposite direction, towards the future pulp. Then the process is repeated at one day intervals. After each cycle (daily), a new calcified is laid, and the construction cell goes one step backwards, moving further away from one another, from the meeting place - the enamel-dentine junction.

The builder-cells work rhythmically and for a long time, hundreds of days, until they build the two layers of the mineral shield. To do this, they need energy, which implies eating and breathing, because while moving away from the calcified layer, every builder-cell not only carries out the construction work, but also offers an organic part of itself along with the minerals. The base composite material is similar to reinforced concrete. Here, the base material – the strictly oriented mineral crystals (hydroxyapatites) – alternates with the organic „reinforcement"- the collagen fibers. The way the cell has to go through and the trace it leaves is a „perpetual trace" in the form of a single, exceedingly long enamel prism. It is equal to several hundreds

of its diameters. The amount of encapsulated „reinforcement" is also correspondingly high. An excellent construction work is done, which is ensured by an appropriate energy flow. In order to understand what happens in the follicle, and then in the enamel shield, we will briefly recall the laws of the energy flow.

In order to ensure its vitality, the body needs a constant supply of chemical energy, supplied by food products. The body provides the organs with the necessary energy and substances to form these organs and to maintain their functional utility. The specific cells of these organs (in the tooth - odontoblasts) are designed to provide biological properties relevant to the working portion (enamel). The non-specific cells, inherent to all organs, ensure the neurovascular infrastructure (dental pulp) required by the specific cells. Initially, from the organ's primordiun stage to the end of its life, there is always a universal rule of centrifugal displacement of matter and energy followed: from the connective tissue area with an abundance of vessels, to specialized cells and from them – to the working structures. In other words, from the transformation of energy to its use and to ensuring the delivery of the necessary components to the destination. The tooth pulp works for the odontoblasts, and the odontoblasts – for the enamel.

Within the dental follicle, we distinguish two basic formations – „epithelial enamel organ" and „dental papilla". The enamel epithelial organ, through its name, suggests the idea of being the enamel constructor, and the dental papilla – the one which constructs the dental tissue called "dentine". Not incidentally, these definitions are written in the Subjunctive Mood. Despite the fact that they work together to build the dental tissues, their roles are different. The enamel organ is not an organ, it is rather the place of storage of construction waste and a rubbish collector. The construction of enamel and dentine, in regards to the transport of building materials, the provision with energy and, to a great extent, with information are provided by the dental papilla.

Indeed, the enamel organ is morphologically a sac, woven from the skin cells directed towards the interior of its own cavity. It is „tied", and within it, the descendants of the embedded epithelial cells live, or rather finalize their existence. They take the form of a star – as they have been crushed from all sides by a fluid – and do not float freely in it just because they are linked to each other, as well as the half-dead „stellate" cells. Practically, no blood vessels penetrate the sac – neither do they bring, nor do they take out. It has no lymphatic vessels or nerves. It is, in fact, in some way disconnected from all the „public services" and links, it is a „hospice" for dying skin cells.

The dental papilla is the exact the antipode - the future dental pulp. Moreover, it comes into existence when the skin cells of the follicle enter a „special relationship" with the nourishing cells. Then begins the formation of the enamel organ's sac and as though the dental papilla is pressed into it. Nevertheless, the dental papilla is in fact an organ, a dense conglomerate of pulsating blood vessels and nerves, which is also the exoskeleton constructor. It is not sealed, and it is open to the internal environment of the body. Blood vessels supplying building materials, energy sources, oxidants lead to it. The blood that comes out of it which gave away all its nutrients to the energy-releasing cells – odontoblasts. Here the terminations of the nerve cells with their multiple ramifications are also oriented. The papilla itself – is a tight node with an intense infrastructure maintenance of the existing functional units of the building cells – the odontoblasts, which have processes that influence the forming enamel.

Namely, this overall landscape pops up under the microscope. On the one hand, a temporary „empty" sac – the enamel organ, and on the other hand - another, full of energy, materials and information, pulsating, full of life, organ calculated for a long life of the whole organism – the dental papilla. Between these two structures is the product of their activity: the enamel and dental tissue layer (dentine). Thus, the answer to the question of where

the „energy furnace" driving the entire construction machine is, seems rather obvious obvious.

From the early stages of tooth development, all the processes unfold into a well-separated area within the maxillary bone surrounded by a dense osseous structure - the crypt wall. In addition to the fact of being isolated from the surrounding tissue where there are no less turbulent changes (i.e. the growth and development of the bone tissues), the crypt also ensures the movement of the fluid, the accumulation and redistribution of the minerals dissolved in it, which are necessary to the developing tissues.

Just in case, let us assess the situation quantitatively. We can record the intensity of the tissue staining to assess the presence and activity of enzymes or perform a morphometric measurement of blood vessel diameters and obtain an objective digital representation of the intensity of the energetic processes. Thus, the vascular flow speed here is 4 times higher than in the muscles at rest. The result is unambiguous. The „oven" burns in the papilla. Everything is always uneventful in the „sac", only a part of its cells that adhere to the developing tooth - the ameloblasts (the enamel constructors) is alive and actively building the enamel, the hardest tissue in the body.

However, in order to live, these cells need to burn the corresponding fuel using oxygen. In fact, not only live, but also create and build; they need many materials, including minerals, for these processes. The enamel builders receive these materials through the formed transport system. The flow of required materials, guided by the organism into the papilla is on the other side of the still forming shield, and the distance between the supplying cells (odontoblasts) and the consuming ones (ameloblasts) is large and steadily increasing. Towards the end of the work, these cells are at a great distance from one another, translating into whole millimeters. Despite all these, the chemical energy and the necessary materials dissolved in the liquid reach the ameloblasts. In order to understand this mystery sacrament,

we do not need any further explanation. It is enough to remember, that the tooth from the beginning of its development, eruption, and all the way is to its petrification, continuously pushes tissular fluid through its tissues. There exists the transport network for the volumetric movement of fluid - the tooth canals penetrate the dental tissue and the special transport system „meridians" into the enamel. By the way, marsupials (and obviously only they) also have a tooth canal network in the enamel. The relationship between these dental features and the sac is one of the mysteries of the animal world on the fifth continent.

The dental papilla is capable of storing large amounts of minerals and then redistribute them to the tissues in the process of mineralization. It was proven by fermentative-photocolorimetric chemical studies during which the concentration of free calcium and phosphorus from dental papilla fluid from the enamel organ and follicular fluid, was quantitatively determined and subsequently compared to their content in blood.

Their average values are shown in the fig. 4. The figures show that the pulp of the dental follicle is able to cumulate these elements because their level is higher than in the circulatory bed. The concentration of ions is lower in the free fluid of the enamel epithelial organ and negligible in the follicular fluid. This

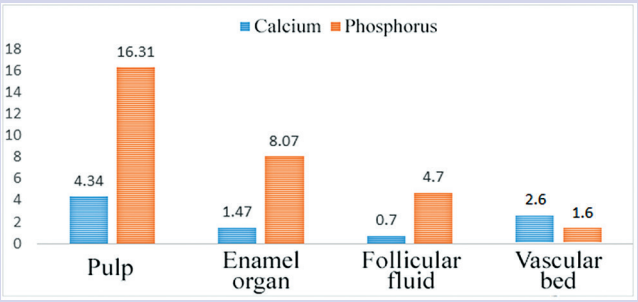

Fig. 4. The concentration of calcium and phosphorus in the dental follicle, follicular fluid and vascular bed, mmol/l

confirms once again that the supply of mineral components of the hard dental tissues is carried out through the papilla of the dental follicle and never from the outside.

As for the volumetric circulation of the tissular fluid from the energetic „oven" - the papilla - to the ameloblasts, it is the cornerstone of everything that happens within the tooth. The suppliers - odontoblasts (tooth builders) and the transport routes operate at a daily, uninterrupted rhythm. This also happens during the life of the enamel builders and after their departure from the „stage". Dental tissue cells (builders) continue to supervise the cell-free enamel. In addition, the thicker the protected enamel layer is, the thicker the layer of dental supervising cells is. If it is thicker, in the case of „pulp horns", the layer consists of 4-5 rows of cells, ready to help another member in carrying out their duties. If it is thinner, then there are fewer layers. If the enamel is missing completely (the root), there is only a thin layer of cement and just a layer of building dental cells. This arithmetic is absolutely logical.

Moreover, we have another pattern. When enamel wear or a disease leads to its disappearance in a particular area, its functional responsibilities are entrusted to the exposed dentine. The tooth canals are filled with minerals and the newly formed layers lose any regularity: the tooth canals become narrow tangling into a node. The odontoblasts are embedded. In addition, since the tooth canals that lead to the enamel are no longer necessary, they cease to exist. The new tissue is tough, but does not look like the original dentine, designed to provide enamel efficiency.

Thus, the quantity of odontoblasts in each sector always corresponds to the volume of enamel it supports. The dentine with a regular canal form exists as long as there is a supervised enamel area. From its beginnings to the end of its existence, the enamel is kept alive by the energetic support, which is carried out directly by the dental cells we refer to as odontoblasts.

I. G. Lukomsky obtained faithful experimental evidence of these phenomena in the 1930s. The experiment is simple and

clear. It has been shown that the destruction of the dental papilla without touching the enamel organ reiterates what would happen in the case of the disappearance of the energy centre. It stops the development not only of dentine but also of the enamel: the cells (the builders) remain alive, but they are no longer able to form the enamel.

With its embryonic development mimicking the evolution of an exoskeleton, the tooth, as well as its ancestors, is formed to the detriment of the energy flow that comes from the center of the follicle - the dental papilla. The completely acellular life of the enamel reiterates the same general feature characteristic of any mineralized external skeleton. All the enamel's biological processes are dictated and secured by the pulp, and only by it. As to saliva, in a certain sense, it is not mandatorily needed and in a certain sense, it is merely an occasional partner in the life of the enamel. Anything can take its place: sea and river water, mud and sludge, and sand, and air. The energy flow that forms and maintains this tissue is born in the internal environment of the body and is directed from the inside to the outside. This is a veritable scientific fact.

Thus, the absolute importance of the pulp's viability as a mandatory condition for a normal functioning tooth becomes clear. The destruction of the pulp is justified only if its preservation is not feasible.

The dentist's supposition that the enamel might be fed through saliva, especially in the absence of the pulp, is unfounded. Extending the existence of a half-dead tooth and its functional viability can be achieved by „preserving" the dental hard tissues that have remained outside the control area of the energy flow in the body. Here the strategy is to seal the enamel and dentine transport paths, which have already become useless.

In connection with the above mentioned, it must be remembered that in the healthy tooth the amount of liquid moving from the center to the periphery is very high - in the

enamel of the erupted tooth it amounts to 15%. Thus, about one sixth of the enamel's volume previously occupied by water is required in a certain way to be filled with an insoluble sealing substance. It is not easy to implement, but it is possible, and the inventors and manufacturers have a lot of work to do here. Preserved, these hard tissues acquire a strong mechanical and physico-chemical stability that meets the expectations related to the lasting existence of a half-dead tooth.

Of course, something that builds up and maintains the viability of the enamel refers to the bioenergetic flow. This flow keeps the entire mass of dental hard tissue away from the natural aspiration to dissimulation (to entropy, balance, degradation, and ultimately always to the minimum of free energy).

With regard to the guidance of odontogenesis, it should be stressed that besides the substances - signals mentioned as paracrine and autocrine that control this process, there is a pattern among these well known factors to which we should pay attention. It is no secret that a diet excessive in easily assimilated carbohydrates (the so-called cariogenic diet), which suppresses the movement of the liquid in the tooth canals, also suppresses dentinogenesis. Researchers have identified the mechanism of this effect. It has been found that an excess of carbohydrates in the diet is perceived by the hypothalamus, which decreases the secretion of hormones which normally activate the parotid gland. This gland then reduces its hormone release - the parotin, which controls the physiological activity of the odontoblasts.

Each ring in this chain of hormonal signals is studied under different conditions of special researches, leaving no doubt that the stimulation or suppression of the activity of the odontoblasts influences both functions – both plastic and „pumping". Modern cloning techniques will allow the creation of a genetic line of parotid gland cells that produce parotine to control the function of odontoblasts. It has also been found that urea compounds, which are introduced in various ways into the internal

environment, provide a stimulatory parotine effect, which is clearly reflected on the activity of the odontoblasts. In particular, the uric phosphate is able to eliminate the negative impact the cariogenic diet has on dentinogenesis.

The hypothesis stating the leading role of the hypothalamus as a pacemaker, which, through the hormonal circuit described above, determines the formation of diurnal rhythmic variation and other rhythmic structures is justified.

6.1. Eruption and Occlusion Formation.

Among the many differences between teeth and other organs, there are also the characteristics of their development. The organogenesis of other systems is complete before birth. At birth, the teeth are not morphologically developed yet. Moreover, most of the „late" ones are in the early stages of development, relevantly embryonic. Thus, teeth formation begins at the embryonic stage and is complete towards the 20th year of life. In addition, it should be noted that unlike other organs, which until birth are in their permanent place, the teeth erupt and position themselves next to other neighbors for up to two decades. Both before and after the eruption they are in a spatial motion (mechanical displacement) associated with the restructuring of the alveolar bone. This extremely critical process for the clinic exceeds the limits of the tooth physiology. The physiology of maxillo-facial development as part of human body physiology is a special chapter of our specialty.

Here we will limit ourselves to just a few important facts for us that relate to this subject. We note that the alveolar bone in humans predominantly consists of spongy bone. It is a very flexible, transformable skeleton part that exists, as long as the teeth do. Let us briefly look at how developing teeth find their natural place.

Before it dissipates, the energy released by the muscles of the masticatory apparatus is transformed into mechanical forces that produce the work necessary to the body (suck, bite, grind food). The maxillary bones at the same time become a point of intersection of the stress lines caused by the muscular effort and resistance of objects in the environment. This causes the bone, which also has its own active internal pressure and which is subjected to waves of pulsating pressure, to actively transform the areas of internal mobile multidirectional mechanical tension. In this turbulent and unstable field of alternating lines of mechanical force the eruption of teeth takes place.

As mentioned above, the tooth develops within a closed space - in the dental follicle crypt. The delivery and accumulation of mineral components dictate the passage of large amounts of liquid through the tissues in the mineralization process. The flow of liquid is definitely centrifugal - from the dental papilla through the tissues and to the exterior. From the crypt, this liquid is evacuated into the oral cavity through a drainage canal (former gubernaculum dentis).

As the rate of mineralization increases, the flow of liquid also increases, which in it turn leads to the widening of the drainage canal.

When the enamel organ mineralizes, the formation of the tooth's root and the periodontium begins. The main fluid flow is then also redirected, thus promoting the tooth movement on its axis and transforming the drainage canal into the tooth eruption canal. In permanent teeth, the drainage canal merges with the alveoli of primary teeth, which leads to the root resorption of the latter and facilitates the eruption of the permanent tooth.

These stages of eruption are clearly visible on radiological images, in the changes of the crypt's shapes. Initially, the crypt has a spherical shape, then it forms an oval shape and at the end, it turns by segmentation into the tooth alveola.

The hydrodynamic shape of the tooth is genetically predetermined (rationalized) which, through purely physical

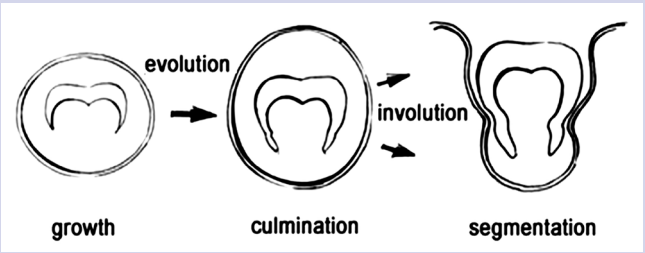

Fig. 5. Changes in the growth of the crypt of the dental follicle

laws, allows it to concentrate the tensions of turbulent waves, turning them into a strictly outward pointed tooth movement force, i.e. the „expulsion" of the tooth. The resultant vector of these forces passes through the root of the tooth, always having the bell shape at the tips of cusps, which always have rounded, rational shapes. The tooth constantly pushes with its cusps on the bone, which dissolves in these areas, paving the way for the tooth to move in a genetically predetermined direction. This is how the teeth of all vertebrates move.

The same process is observed by the physicians in all cases of normal and abnormal tooth movement. Once the guiding canal is fully opened and corresponds to the diameter of the crown of the permanent tooth and the enamel organ in its path finishes its involution, the pressure difference increases sharply, and the tooth crown no longer faces resistance. The rate of tooth eruption at this point suddenly accelerates due to the flow of dental fluid. We note that the „expulsion" of the tooth from the bone, due to well-understood causes, always goes occurs faster in areas with intensive mastication and not so fast in areas found outside this process. Thus, the process of tooth movement observed in clinics and their eruption are included into the elementary models of basic biomechanical and hydraulic laws (due to dental fluid).

7. The Enamel's Functional Acid Resistance and the Electrical Activity of Odontoblasts

By changing the dental's fluid speed of movement, we can modify the tooth's sensibility to caries.

The degree of acid resistance of a pulpless tooth characterizes its solubility that depends solely on the structure and composition of the enamel. This is the enamel's structural (passive) acid resistance. The acid resistance of a living tooth is determined by both its active and passive components that depend on the pulp's vital activity. The structural acid resistance is determined by examining the enamel's organic and inorganic components, whereas the functional acid resistance- by examining the state of the dental fluid, controlled by the pulp and its pump. Dental fluid has a basic medium. Its migration, even when the pulp is at rest, is fairly active. In 24 hours, the fluid manages to renew its composition tens of times.

The conditions activating the pulp's bioelectrical functions also increase the centrifugal permeability, the electrical transmissibility and the acid resistance. The conditions that diminish the pulp's bioelectrical activity do the opposite.

The coordination of metabolic processes in the odontoblastic layers is carried out by subordinating the small outline (the tooth) and the big one (the entire organism).

8. The Abrasion of Dental Tissues

When speaking of biological objects (including the tooth), destruction should not be seen as straight up annihilation, but rather as a compulsory phenomenon similar to dissimilation, a normal part of the vital processes. For these reasons, a decrease in the "tempo" of abrasion should be considered an undesired even abnormal state.

The existence of teeth and periodontia in the oral cavities of ancient and contemporary man is always accompanied by significant abrasion of hard dental tissues. Abrasion, a passive wear of tissues, is a necessary condition for a tooth's health. A real, healthy tooth is an organ subjected to constant wear and has a certain degree of abrasion that tells us about the "implication" of informational-regulatory mechanisms, i.e. the biological control. A complete set of teeth (the bite) of a dentally healthy adult cannot be represented by juvenile, nonabrased teeth. The lack of abrasion is an unequivocal symptom of a bad dento-occlusal system. If a young man has all of his 28 teeth intact but with nonabrased cusps, we cannot promise him high

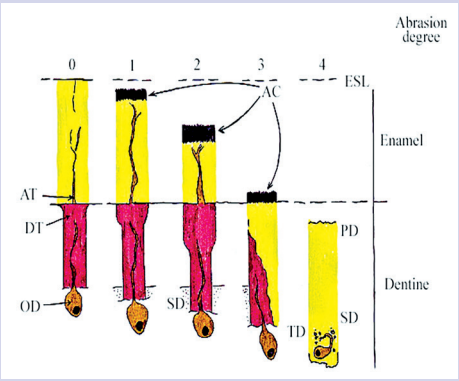

Fig. 6. The abrasion of hard dental tissues and the role of odontoblasts

TA – adamantial transport pathways;
DT – dentinal tubules;
OD – odontoblast;
CA – adamantial callus;
PD – primary dentine;
SD – secondary dentine;
TD – tertiary dentine;
ESL – enamel surface level.

resistance to dental diseases. On the contrary, we should warn him that he might have a rather unpleasant eruption of his wisdom teeth or other problems. However, if we were to make a denture/dental prosthesis for an old person, considering the previous statements, we wouldn't need to reconstruct a juvenile denture with cusps, crests and fissures. On the contrary, if such formations are intact, we will have to remove them.

According to A. Brock, one of the founders of anthropology, using data from the 19th century, it is recommended to split the indices of abrasion into six stages:

0 – lack of abrasion
1 – minimal abrasion of the enamel
2 – the abrasion of cusps with pencil openings of dentine
3 – dentine is uncovered on a significant surface
4 – the substitution dentine is uncovered
5 – the abrasion of teeth all the way to the equator
6 – the abrasion of teeth all the way to the roots (only a small portion of the enamel surrounding the roots may be still left) .

These correspond to: 1-children, 2-teens, 3-adults, 4-fully grown-ups, 5-elderly, 6- old people.

Yielding to the realities of civilization, we have to admit the existence of a number of exceptions from the optimal abrasion indices, each age group having the index corresponding to the previous (younger) age group. However, we have a number of solid proofs, confirming that a proper natural and artificial abrasion will have positive effects.

Now, we must also take into account that the abrasion of a healthy tooth leads to the compaction of the remaining layers of enamel and dentine, a process identical to the callusing of soft tissues. The area subjected to abrasion becomes harder and more compact, thus minimizing ulterior abrasion.

The only condition needed for the development of a callusing process or the acceleration of maturation is to keep the biological control intact, i.e. to protect the pulp's viability and its link to the external sector. The mechanism of this counter expedient (the increase in hardness of the deteriorated sector) works thanks to the centrifugal flow of dental fluid that contains all the necessary ingredients. The controlled flow of dental fluid is always headed for the damaged zone, restoring and consolidating what remains.

A light abrasion is only one of the shortcomings of the structural-functional complex of the respective anatomical zone. Others include the thinning of the facial skeleton, the predisposition to an incorrect consecutiveness in tooth eruption dyschronosis, to occlusal anomalies, to caries and periodontitis.

This peculliar state is characterized by the following factors (except the case when the tooth's initial anatomical shape is unchanged):

- More pronounced articular tubercles
- A minimal amplitude of the mandible's lateral excursion
- Predisposition to periodontitis
- A difficult eruption of the third molars
- Arthritis of the temporo-mandibular joint

The questions concerned with this syndrome are relevant in clinical practice and need a special and separate discussion. It is important for us to understand that the insufficient abrasion of teeth is a typical characteristic of modern man.

9. The Passive and Active Components of the Interaction between the Surface of Hard Dental Tissues and the External Environment

The tooth's crown, sunk in oral fluid for an entire lifetime, is subjected to the action of chemical, physical and microbial factors. What happens during this constant interaction upon the dental tissues of both living and nonliving teeth? Let us begin with the oral environment's acidity level. Obviously, demineralization will dominate in case of decreasing pH, the tissues losing ions to saliva. A contrary result- mineralization- appears when the pH increases and the saliva is oversaturated with mineral salts.

The living tooth will organize a self defence as an answer to different actions of various intensity of the environment, be they disintegration or mechanical wear, microbial invasion or acid demineralization. In comparison to other tissues of the organisms, the enamel, being acellular and devoid of blood vessels, cannot respond with an inflammatory or productive reaction. There is only one move in its arsenal- to initiate a wave of hyper mineralization.

The most important point in this situation is that the depth of calcium's penetration and its inclusion into the tissues of living teeth are much weaker than in nonliving teeth. This tells us that something impedes the penetration and fixation of ions into the enamel.

The vitality of teeth is opposed to any process that leads to the minimization of free energy, even against passive divergent processes and even against the movement of substances in or out of the enamel.

10. Resistance against Destructive Environmental Factors

The microflora of the oral cavity, through its biochemical activity, always leads to the decrease of mechanical resistance in tissues, the previously regular masticatory forces becoming too much to handle. The "safety margin" of the enamel and dentine is in need of a constant resupply and it is only the dental pump that can make up for the loss of provisions through its activity. It is solely the pump that can ensure a tooth's correct and safe operation throughout the whole life, till its end. The presence or absence of life force within a tooth in no way affects the destructive potential of the microbial environment or the functional mechanical forces.

The mechanical wear of the enamel's surface, i.e. the loss of its particles is a vital component necessary to the tooth and represents the cumulative result of multiple short timed "episodes". Every "episode" of wear is essentially a purely mechanical process the result of which depends upon the initial physical indices- the force and the abrasiveness of the interacting objects.

In nonliving tissues the micro destructions add up and the separate microscopic trajectories merge into large scratches that cause abrasion and loss of tissue.

The mechanical deterioration of the enamel's surface determines the occurrence of biological changes favourable to the formation of dense and highly stable mineralized zones deep within the tissues. A functional, invisible, mineralized nucleus of the sort can be found in the enamel of a living tooth, under any zone of mechanical abrasion.

The microscopic deteriorations in the enamel add up, thus appearing the signs of its weariness (fatigue), which leads to the

appearance of large fissures and loss of integrity. Therefore, the cumulation of weariness is, in fact, the inevitable end of every cycle.

The portion of tissue subjected to microtraumas finds itself in the centrifugal fluid flow's area of action and eventually obtains additional mechanical resistance.

The microbial lesions of the enamel are essentially of physico-chemical origin. The prolonged presence of dental plaque on the enamel's surface and its boundaries do not coincide with its real destructive effect.

Within the enamel, under conditions that exclude biological control, the complex of destructive factors leads, at different rates, to the destruction of tissues, a process that takes place simultaneously in all the listed directions. The cumulation of microdestructions of various origins by the "weariness of the material" mechanism, ultimately leads to the destruction of tissues.

With aging, the dental tissues "petrify" more and more. The space previously occupied by water and organic components has been replaced by mineral components. During the reduction of the said volumes, the links of hydraulic transport to the pulp also weaken, i.e. the possibility of physically controlling the enamel's state is vanishing. The enamel tends more and more to be a nonliving tissue (abiotic). This process manifests itself most in the zones of intensive functional load, where the possibility of biological control is the smallest. Simplifying the situation a bit, we can assume that the callused sectors of the enamel gain a higher structural (passive) physical resistance upon losing its adequate variability.

This is why, the loss of biological control (after depuplation, for example) does not lead to any dramatic result for an old tooth, because the cumulative effect of destructive changes would be slow. The opposite happens to the teeth of young people. The tissues of such a tooth a much more dependent on biological

control, this is why the cumulation of microdestructions after the depulpation of a recently erupted tooth will lead to an absolute obliteration of the crown.

Ever since the emergence in literature of data referring to the finding of marked atoms belonging to the saliva in the superficial layers of the enamel, the illusion that these tissues are nourished by saliva appeared. If a pulpless extracted tooth, or fragments of it will be put into contact with a fluid (saliva, oral fluid, physiological solution) containing the radioactive elements P 38 and Ca 45, then the listed elements will bind to the superficial surface of the tissues. For obvious reasons, the denser and more mineralized a tissue is, the less likely are the elements to bind are; and the more dematerialized the tissue is, the more actively will the listed ions bind to the surface.

In a normal state, when the pulp is linked to a living pulp, the latter will impede the penetration of radioisotopes from the exterior, from the saliva. The pulp, through its biological activity exerts its bioactive designation, that being to limit the "unauthorized" penetration of substances from saliva in the enamel. In some cases, for example with radioactive iodine or with some poisonous substances, the protection is futile, whereas in the case of Ca45, the penetration in the enamel is completely stopped.

As a result, the isotopic studies confirm that the pulp controls the processes taking place in the superficial layers of the enamel and determines their general direction- the resistance to passive penetration (abiotic, physico-chemical) of ions from the oral fluid into the enamel.

Therefore, the passive abiotic processes (physical and chemical) within the enamel are the basis of every primary interaction between its surface and the active substances of the environment. At the same time, the signal sent from the scene of the event, will naturally lead to the corresponding changes within the enamel and its surface, preparing it for the possible harmful effects.

11. The Dental Pump

The word combination that serves as a title for this chapter is odd from both a linguistic and dental point of view, these words not collocating. The term was proposed by American endocrinologists to refer to the main component of a tooth's hydraulic system. According to their data, the surplus of sugar in the bloodstream, whether or not it is caused by insulin deficiency, leads to a decrease in the efficiency of the dental pump.

Inside the tooth, there are transport structures- the dentinal canaliculi, filled with liquid and directed towards the enamel. Obviously, this liquid can only move along the course of the canaliculi.

As it is well known, the dental fluid inside the dentine is being kept at a constant pressure and flow. This liquid changes its composition ten times a day and is similar to that which ensures the moistening of all the epithelial tissues, including the enamel. Thanks to it, the enamel can change its properties. The tissular fluid emerges as small drops on the enamel's surface. Its rate of formation can either raise or drop. Its speed of movement is in the region of tens of millimetres per hour and its pressure- a couple of

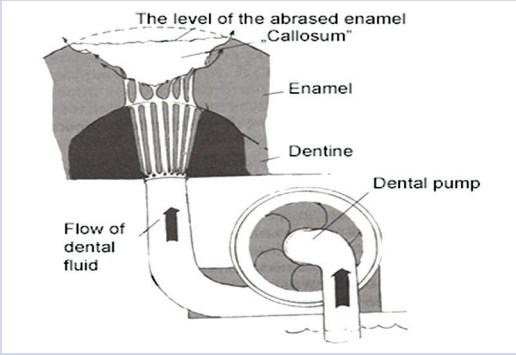

Fig. 7. The dental pump and the formation of the enamel's finite mineralization zone- „the callosum"

tens of mmHg. All the electrolytes of the intercellular environment and all the amino acids are present in the enamel liquid.

The automoistening of tissues (the pump's activity) considerably worsens the adhesion of the filling masses. We are talking about a decrease (or increase) of this important characteristic by an order of magnitude.

The pump's activity is aimed at the fluid overcoming the resistance of the "molecular filter" of the enamel-dentine junction and of the capillaries of the enamel's ultramiscroscopic spaces. We can increase the pressure gradient (difference) and thus increase the movement speed by ensuring osmotic pressure on the enamel's surface or through a local reduction of atmospheric pressure.

The pump's primordial function is the hydraulic pressure between the physiological limits. It is thanks to it that within the tissue the rational physiological changes of the immediate or delayed variety take place.

The simultaneous changes refer to the enamel's superficial layers and their resistance to chemical agents and their electrical properties. The decisive and convincing experiment concerning the assessment of simultaneous changes was performed on lab rats and consisted of the following phases: an incisive was devitalized under anesthesia (the other, symmetrical tooth was

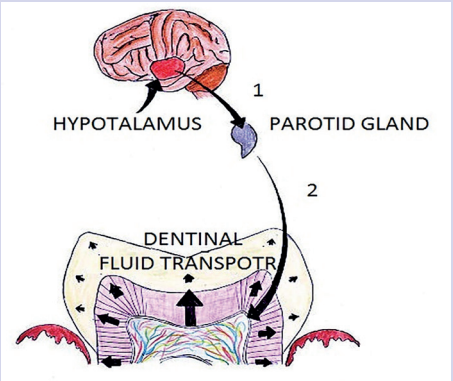

Fig.8."Hypothalamic-parotid" endocrine axis, the control of the dental fluid flow (J. Leonora)

1 – releasing-factor;
2 – parotin
(the image was published with the consent of the author)

used as a reference), and immediately after that the procedure of dosed etching of the enamel began. The immediate proximity of incisives in the case of rats allowed to simultaneously etch both the devitalized and the reference tooth.

Upon neutralizing the remaining acid in both teeth, the depth of the destructive effect can finally be assessed. The technical resources (polygrapf-profilometer) allowed the attainment of qualitative and quantitative characteristics of the superficial enamel's degree of destruction. This way was documented the primordial effect of the discussed process. The tooth's devitalisation leads to an immediate drop in the acid resistance of the superficial enamel. In other words, the cessation of the pump's activity instantaneously reflects upon the chemical properties of the teguments.

The slow changes linked to the dental pump's operation reflect upon the enamel's base functional quality- its hardness.

Being disconnected from the dental pump, the enamel-dentine block of a living tooth undergoes a series of changes that lead to its degradation. This occurs only if the neighbouring dental tissues are not subjected to negative changes.

Over time, the enamel does not accumulate resources anymore, but rather loses its own mineral resources. It "softens" substantially (according to the microhardness data), becomes more soluble, more electroconductive and absorbs the radioactive calcium isotopes more actively, exhibiting all the signs of "loosening".

Thus, the proper operation of the dental pump, which ensures the existence of pressure in the tooth's hydraulic system, is the necessary condition to proper mineralization of the enamel throughout the entire life.

Besides this, ensuing from the general vision, we understand that even the fluid's composition, including its viscosity, cannot be the same in two different people and at two different points in time. For the reason outlined above, the efficiency of the hydraulic mechanism should also vary by definition. We should also note

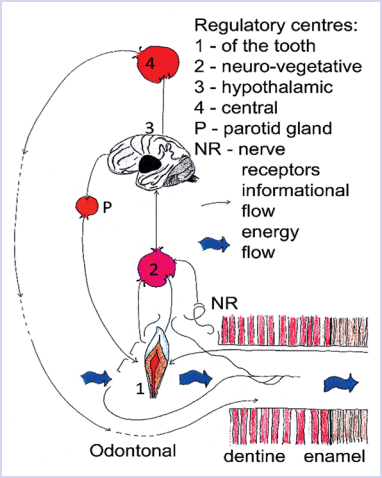

Fig. 9. The main diagram of the control mechanism of the"dental pump".

that the fluid's movement speed and its quality are tied to a less concrete but largely significant index- the general state of the organism.

Particularly important and interesting is the experimental data concerning the role of hormones in adjusting the speed of the centrifugal movement of dental fluid. It is calculated quantitatively, by the movement speed of different colorants through the lumen of dentinal canaliculi. The "hormonal axis" hypothalamus-parotid gland-odontoblastic layer was described in many experiments conducted on different objects. It has been proved that the hypothalamus secretes a parotid-hormone releasing factor. The stimulation of the said function was obtained by injecting carbamide into the bloodstream – the oppression of a diet rich in carbohydrates. In conformity with the stimulation or the slowdown of the releasing factor's secretion, the movement speed of the marked substance through the dentinal canaliculi changed from a halt to maximal speed. If the experiment is performed in the absence of a parotid gland, the outlined effect will be absent. However, if we were to administer parotid hormone, the effect would show up. Considering the hypothalamus' obvious role in managing and organizing biorhythms, we have all the reasons to assume that the described hormonal chain carries out its activity in accordance with the respective biorhythm, determining the rhythmical apparition and structure of dentine and enamel and also the periodic biorhythmic fluctuations of the enamel's acid resistance.

The daily and annual (seasonal) rhythmical fluctuations of the parameters have been linked to a pattern, akin to the ones concerning pregnancy and menstrual cycles. Besides the periodic rhythmical fluctuations, the acid resistance also depends upon accompanying factors such as physical and emotional stress, the administration of certain drugs (especially hormonal drugs), various diseases, sleep disorders, etc.

Upon subjecting the tooth to different actions with a localized effect, its acid resistance changes significantly. The experiments carried out on animals and volunteers proved that exciting the tooth with factors of various physical nature leads to an increase in the pulp's activity which in turn manifests itself through the enamel's increased acid resistance. The respective response mechanism includes at least three levels: organic (self-regulation), nervous and hormonal. It is necessary to assume the existence of an even higher level that determines the quality of the dental fluid- the organism.

Particularly interesting is the increase in acid resistance during the blockade of whole tooth. By eliminating the innervation and blocking the link with the central nervous system, the pulp's physiological activity is no longer inhibited by the CNS. This type of regulation is based upon the inhibition of local processes, typical of multiple systems of the organisms, proving their considerable autonomy.

While maintaining the centrifugal flow of dental fluid, the dental pump stops the "unauthorized" penetration into the dental tissue of not only harmful and destructive agents, but also of neutral (different pigments) and benefic ones, such as Ca and P that constitute a great part of the enamel's crystal structure. On the other hand, the dental pump's biological mission is to also ensure the enamel's general and localized mineralization. Thus, the tooth's main specific cells- the odontoblasts continue throughout their whole life their activity that started in the embryonic phase.

12. The Biological Rhythms of a Tooth's Activity

The tooth's skeleton changes with age. Given its genetic predestination, together with all the other parts of the organism, its evolution is headed towards the only possible scenario- aging. The main processes are not a local phenomenon of the dental skeleton, they are the same for the whole macroorganism.

We should accept and consider life's biological hard-coding not only for deciduous teeth, but for permanent ones as well. These have a lifespan comparable to the lifespan of a living person. In this case, this organ's main stage, its wave of vitality is asymmetrical: the growth and assimilation processes occur till the eruption, after which a drastic change follows, the mechanical decay of tissues caused by the abrasion and mineralization of the remaining tissues becoming the dominant process.

The enamel's mineralization throughout life is not ensured by the systemic mechanisms with their biological clocks, but rather by the physico-chemical properties of the oral fluid in which the crown is covered, an external environment that is foreign from its body.

As an example, there are three types of basic specific cells: the ones creating the bone (osteoblasts), the ones that destroy it (osteoclasts) and the ones that maintain the balance between creation and destruction (osteocytes). Thanks to them, the bone is constantly renewed throughout the entire life. When it comes to the tooth, only the odontoblasts play the role of both "creators" and "maintainers". Ergo, a continuous process of creation, maintaining and destruction take place within the bone, whereas in the tooth there is only creation and maintaining. Additionally, the tooth's base , the dentine, is surrounded by acellular formations (enamel and cement) that are incapable of any internal reconstructions.

The structural organisation of this biological system and the rhythmical processes that occur within it seem like a complex of scientific analysis, a complex which is unified in both time and space. However, the multitude of information concerning this field still has a character of ascertainment.

Up until this day, there is no information about any variable parameter that is not involved in a continuous cyclical variability. However, we can admit that there are no such parameters and nature complies only to rotational laws- the universal principle of creation.

We must specify that chronobiology and its sub-branch-chronomedicine (or medical biorhythmology) do not have a unified conceptual model yet. Each of these disciplines only makes the first steps towards the common goal. Chronodiagnostics, chronotherapy, chronopharmacology, etc are rather a sign post than an actual predominant doctrine. Chronodontology is more of a terminological notion, this branch having no valuable accomplishments.

Next we will refer to well known facts regarding odontological biorhythms. We will refer to three biorhythms: daily ones (circadian), lunar and annual. For these, we will have to take into account at least the basic information concerning the dynamics of their underlying mechanisms.

The circadian rhythm is without a doubt one of the most important "invisible" features of every living thing, referring to the first manifestations of life on Earth and being inherent to any living being. This rhythm subdues all the events in the organism, including the ones in the tooth. The centrifugal flow originating from the vascular glomerulus and containing all the components necessary to the formation of dental tissues is the main coordinator of this rhythm. In 24 hours, the creation front of every calcified structure shifts its position around 2-6 mm. Not only the circadian rhythms, but also the lunar and seasonal ones are of endogenous origin and form during the stage phylogenetic de-

velopment. In this case, the synchronization of their frequencies is influenced by astrogeophysical cycles. It was experimentally established that among all of the external factors, photoperiodicity, temperature changes and fluctuations in Earth's magnetic field play a particularly substantial role. Light is the strongest synchronising factor. The light signal, through the visual analyzer and the hypothalamus, acts upon the pituitary gland and induces rhythmical hormonal fluctuations in the bloodstream. A specific circadian rhythm concerning the suprarenal cortex and the pituitary gland's adrenocorticotropic function as influenced by photoperiodicity has been even discovered.

The enamel's acid resistance is a parameter governed by a distinct circadian variation. A higher acid resistance was observed during the night in every person's enamel, reaching its peak at about 03:00 and its minimum at 15:00.

The result of this observation was compared with the data obtained from the previous experiment, the one revealing the effect that the mediators of the sympathetic and parasympathetic nervous system have upon the enamel's acid resistance. During the chronic and acute experiments, it has been concluded that catecholamines inhibit the parameters of the studied enamel and the ganglioblocants activate its acid resistance. A completely opposite effect is achieved by the mediators and blockers of the parasympathetic nervous system. By comparing the two, we were able to conclude that the alternation of the two components of the autonomous nervous system may be a possible explanation to the circadian rhythm of the enamel's acid resistance.

A monthly rhythm was also discovered by analyzing the enamel's stability in women of childbearing age. In this case, an individual rhythm was identified in each of them. Although they did not coincide, there was a clear periodicity of about 28 days. When these numbers were compared to each woman's individual menstrual cycle, their close coincidence couldn't remain unnoticed. In women whose menstrual cycle lasted 28-

32 days, the acid resistance was minimal on the 12th-16th day. Regardless of the period's length, the minimum coincides with the days of ovulation.

The dependence of the enamel's acid resistance upon the activity of female sexual hormones was studied by observing the acid resistance's dynamics during hormonal treatment. Oral contraceptives have kept the fluctuations of the acid resistance curb to a minimum. These observations were only estimative. However, this is not about the role of a particular hormone in changing the state of the enamel, although such a dependency might undoubtedly exists. This idea can be confirmed through other observations, regarding the time of the first menstruation and acid resistance (and respectively, dental caries). Girls with an accelerated puberty have a weaker enamel and a higher caries incidence, while the "late bloomers" have a significantly better dental health (ceteris paribus).

From a practical standpoint, the seasonal fluctuations of the enamel's state are the most important. This conclusion was reached by conducting long-term observations upon several groups of children (7-8 years old) and teenagers (14-15 years old). Every month, on the same date, each of them had their oral cavity checked by a standardized method, thus monitoring the acid resistance during the same morning phase of the circadian rhythm.

As a result, a general pattern was discovered in several different groups- February and March were met with a constant drop in acid resistance, whereas the optimal parameters were encountered during the months of May and July. Throughout the rest of the year, the EART (enamel acid resistance test) index was close to the annual average.

As to the individual characteristics of the enamel's fluctuations in acid resistance, they are as follows: in some cases the seasonal fluctuations were camouflaged by oscillations of different genesis, in other cases an additional sudden leap was identified in the tooth's vulnerability to caries in the months of

November and December and while in some children this leap occurred annually, it was sporadic for the rest. Starting with puberty, the acid resistance's rhythmical fluctuations get more acute in the majority of pupils.

The occurrence of critical situations is mainly due to the internal mechanisms that periodically bring down the enamel's resistance to dangerously low levels. And when it happens for annual and monthly minimums of resistance to coincide, it results into a serious and prolonged opening in the shield, which is sufficient for the commencement of a microbial and acid invasion. The obtained data answers by itself the question concerning why and when the stage preceding the caries begins and later on the disease itself- it happens during a critical drop in the enamel's physiological acid resistance.

The enamel's stability is a physiological parameter that depends upon the activity of the dental pump, whose activity varies according to the periodical changes that occur within the organism. In practical terms, it is important to know that the most serious and prolonged decline of these numbers occurs annually, by the end of winter. Knowing this, there is a genuine perspective of devising a strategy involving the chronoprophylaxis of dental caries.

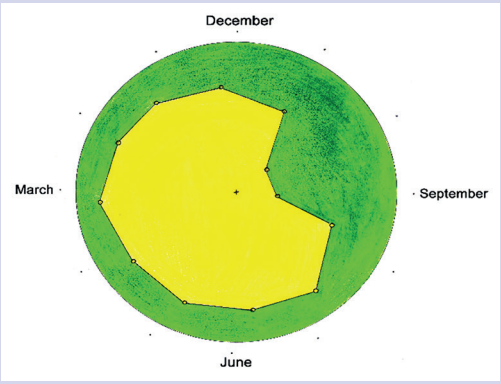

Fig. 10. Annual dynamics of acid resistance among schoolchildren (conventional units.)

13. The Dynamics of the Enamel's Hardness

The enamel's breaks the record for being the hardest living tissue. Its biological vocation is to be exceptionally and incomparably hard, harder than anything it would have to encounter. As we know, in mammals, the enamel's external surface withstands pressures of up to half a ton per square millimetre. For the human enamel, this number is 400 kg/mm^2. This resistance is due not as much to its chemical composition as to its permanently controlled special structure. The key to its hardness relies on its net-like construction which is strictly ordered and tightly packed, made of calcium needles similar to hydroxiapatite. The enamel is a specific composite material. The small crystals tied with an elastic organic substance, together with the intertissular fluid have transformed into a solid, yet flexible and changeable monolith that serves as building material for adamantine prisms , with a diametre of 3-6 mkm and a length equal to the enamel's thickness. Several prisms of the sort form adamantine fascicles that can be seen under a microscope.

The mechanical characteristics of the mineralized surfaces are different from the others not only by their large numerical values, but also by their distribution, and more importantly, their ability to acquire adaptation variability. There is a layer with intermediary properties between the extremely hard surface and its elastic support.

Not only does the hardness gradient exist between the layers, but within them as well. Therefore, the enamel's external layer is the hardest and most mineralized one. The dentine's structure is governed by a similar rule.

Unlike lifeless instruments, when the tooth inevitably loses its worn out layers, it immediately prepares the layers underneath to take their place, mineralizing the adjacent layers one by one,

deeper and deeper as time goes on. Regardless of how old a tooth is, as long as it still has its pulp, it keeps the hardness of its functional surface and the hardness gradient between its layers. This property is inherent to the tooth and is a result of the adaption changes undertaken by the dental pump.

Unfortunately, the pulp's destruction (depulping, devitalization) is a common therapeutic practice, this is why it can be easily recreated in an experiment. Upon doing this, the only thing left to do is to compare the way living and devitalized teeth will fare under load. It seems that the results are at opposite ends of the spectrum. The enamel of devitalized teeth becomes more fragile, the microhardness drops and due to a cumulation of microlesions, the destruction can be seen with the naked eye. At the same time, the external layers of a living tooth experience an increase in toughness, as a response to the requirements set by nature.

The lab data does nothing but confirm the observations made by dentists. Everyone knows this harsh truth- by eliminating the pulp from a young tooth we condemn it to destruction and physical load only quickens this process. The dentures attached to living teeth last twice or thrice as much as those attached to pulpless teeth. A devitalized tooth inevitably loses the strength of its hard tissues and their adaptation variability.

Let us remember the tooth's fluid transport system and its wetness. Ergo, the tissular fluid makes up about 2% of a tooth's mass and 10% of its volume. Water evaporates easily even at room temperature and it penetrates the tissues just as easy as it leaves them, which tells us about the presence of fluid in the communicating vessels, thus forming a moist transport system. Moreover, thanks to diffusion, all the soluble substances contained in the dental fluid move along the gradient line through the enamel's micropores filled with fluid. Thanks to the dental pump, these substances only move centrifugally: from the pulp towards the exterior. The fluid continuously flows towards the surface, where it finally emerges as microscopic drops.

Evolution has been quite ingenious solving the challenging task of combining the enamel's impressive hardness and its variability, including the ability to pinpoint its own lesions.

The initial stage of this mechanism modus operandi is the ultramicroscopic deterioration of the hydraulic network, followed by the signal (hydraulic shock) and finally the distribution of mineralized components to the right place at the right time.

The electrical resistance of tissues changes in two stages. Initially, the zone subjected to abrasion becomes a much better conductor, favoured by the extension of the tissue's transport paths, the water in which serves as a second degree conductor. As the wear increases in the central zone, the electrical conductivity in the said area decreases, while it remains high in the peripheral zones. When the abrasion reaches the enamel-dentine junction, the so called "transparent dentine" makes its entrance. The obliteration of dentinal canaliculi, in this case leads to the zone's mineralization, an increase in hardness, acid and electrical resistance, all of which are signs of the dentine's transition to a whole new quality.

The hypermineralization zone tends towards a conical shape, with its base towards the tooth's functional surface and the tip towards the pulp. This conical zone of dentinal "callosum" is constantly extending as the abrasion goes on. However, there are always zones of living dentine forming around it, feeding it. There is a physiological energization in these layers, starting from the odontoblasts and spreading throughout the hydraulic system of the whole tooth. The mission of these layers is to consolidate the cone and is dictated by specific conditions. In the end, when the tooth's abrasion is substantial, the cone occupies the whole crown. But even in this situation, the dentine keeps its odontoblasts and their functions as well as the transport structures controlled by them.

14. The Acid Resistance of the Enamel

Despite the similarities, the acid resistance of the enamel-substance and the enamel of a living tooth are two completely different phenomena. The essence of this difference could be explained with an easy example. If, hypothetically, 50% of all the minerals will get out of the enamel-substance into the solvent, such a substance will obviously cease to exist. Nevertheless, if the same thing occurs within the enamel-tissue, the result will be different. If the loss of minerals takes place at a moderate rate, it can be compensated by the tooth's or the whole organism's reserves. Thus, the release of such a large number of ions, does not in no way indicate towards the enamel's possibility to withstand destructive agents such as acid by itself.

The enamel's ability to defend itself was assessed following the interaction between acid and the tooth's surface, i.e. by examining the degree of destruction caused by the etching. This can be done in an easy biological experiment. A small drop of acid is applied to the enamel's surface for a couple of seconds. Following its removal from the surface, it will leave an opaque mark. At this moment, what we have to do is to measure the degree of opacity, or to be more exact "roughness".

The possibilities to assess the roughness left by the etching are fairly high. There are several techniques to do this: by comparing the surface's degree of disorganization by examining it under a microscope or electronically scanning it, or by measuring its optical properties. However, the most adequate technique in our case is "profilometry". The corresponding device (profimometer or profilograph), "palpates" the studied zone with a diamond sensor. All the irregularities on the sensor are perceived by an electronic device that, using the date, recreates the profile with

a magnification rate of hundreds of thousands. Thus, the device allows us to see the model as a cropped section, a hyperbolized image of the relief. The device's purpose is to evaluate how well the surface was polished and to eliminate the roughness. We have 14 types of roughness, including the subtypes.

The variation of the enamel's acid resistance

Modifying factors		Variation of the enamel's acid resistance	
		Increase	Decrease
Biorhythms	Annual (seasonal)	Summer	End of winter
	Monthly (menstrual cycle)	The follicle's growth stage	Ovulation phase
	Daily (circadian)	Night	Day
The organism's general condition	Systemic diseases	Obesity related diseases	Linked to acute inflamations
	Physical exercise	Moderate activity	Physical exhaustion
	Emotional stress	Absent, relaxed	High
Systemic actions	Vegetative substances	Cholinergic	Adrenergic
	Other substances	Fluoride, ascorbic acid, urea, tocopherol, eleuterococcus drugs, parotid hormone	Hydrogen peroxide, bradykinin, easily assimilatble carbohydrates
	Acupuncture	The inclusion of general stimulating mechanisms	-
Local actions	Actions upon the efferent nerve	Deafferentation	Nerve irritation
	Actions upon the tooth	Mechanical, physical, physico-chemical stimulation	Intrapulpar injection of cocaine
	Curative manipulations	Keeping the dental pulp	Depulping

To obtain comparable results, we need a strict procedure of dosing the etching process. A precise dosing can be done by manipulating the exposure time or the type and concentration of the acid. During the experiments on extracted teeth, the exposition and concentration were not limited, but were selected empirically, depending on the degree of information provided by the obtained image. Usually, considerable concentrations (10-15%) of lactic or hydrochloric acid were used and the exposure time was of about one minute. The clinical doses are smaller. Then follows the search for the optimal concentration of acid at an exposure time of five seconds. Therefore, the word "etching" when talking about mass screening studies on living subjects should not be interpreted literally. In the enamel of living teeth, an exposition of five seconds disorganizes only the film's superficial organic layer. After a couple of hours, the roughness is gone.

In order to quickly and easily measure the surface's degree of disorganization, we can colour the etched surface to the detriment of precision. The colorant resting on the rough surface will allow us to estimate the degree of etching. The more intense the colour is, the deeper the decalcification is, respectively the lower the acid resistance is. The colour reference scale allows us to obtain the needed index in conventional units.

The critical period was during the months of February and March. Namely during these months does the acid resistance suddenly drop, and by the way, it is during this time that the most serious dental problems lay their foundations.

The enamel's stability towards acids depends upon the solubility of the said structures and the protecting biological activity of the pores, which is a rather complicated and not fully understood activity. Solubility is governed by the strict laws of physics and chemistry. The reaction's result is determined by

the quantity and composition of substances and strictly depends upon the initial solvent and the substrate.

We can manipulate the acid resistance with the help of less traditional methods for western medicine. For example, by stimulating the biologically active points (the "Hegu" point) through acupuncture, we will get an increase in acid resistance even as the procedure is still in process. The same result can be reached by employing different acupuncture techniques. The episodic stimulation of the biologically active points have unique short term effects, but if done regularly, the procedure can yield long lasting effects.

After several experiments backed by solid proof, no one is surprised by the hypnotic effect. In a deep hypnosis state, the acid resistance increases and remains at the same level for several hours after the session.

The nerve's sectioning produces a violent reaction between cells and a spectacular increase in electric activity recorded by the oscillograph. This wave coincides with an increase in acid resistance.

The fact that the enamel has biological variability is a fundamental truth that was discovered through a usual, simplified evaluation process of the enamel's disorganization degree caused by a dosed acid microtrauma.

It was proved that during February and March there was a drop in acid resistance among all the children. Therefore, the children with the most prominent drop in acid resistance are the most prone to dental caries. Next, multiple drugs were sorted into two categories: those that increase acid resistance, and those that do the opposite. By administering the formers, it is possible to cut in half the number of new carious lesions. This effect was obtained by prescribing significant doses of vitamin C, Siberian Ginseng, etc to the group at risk during the critical time of the year.

Based on this fact, a preventive treatment program was organized for children of school and preschool age. A mass examination of children's acid resistance, allowed us to divide them in two groups according to their estimated potential caries incidence. The ones from the risk group received treatment and were examined 2-3 times a year, whereas the ones from the group with high resistance – much more rarely. Obviously, all the children were instructed how to properly take care of their oral cavity, and were practical lessons of oral hygiene also organized.

The major factors influencing the enamel's properties are highlighted in the table. These properties depend upon the pulp's functional state. At the same time, this dependency weakens with time due to the increasing mineralization of tissues.

15. The Endogenous Variability of the Enamel's Organic Teguments

The surface of the erupting tooth is covered with a thin organic layer (cuticle), made of dead cells which were once the enamel's builders - the ameloblasts (Nasmyth membrane). After the eruption, the cuticle remains intact only in a scarce number of places, untouched by abrasion. Meanwhile, it is replaced or covered by another layer - a biofilm, which is a deposition of organic matter coming from the oral fluids. The organic layer is always populated by microorganisms found in the oral cavity, being a "living roof" to the tooth.

The film's properties are highly unstable and vary by large margins.

The dental fluid is being continuously released from tissues and together with the oral fluid is goes on to imbue the oral cavity's surfaces with an aqueous solution, thus influencing the populations of microorganisms.

The osmotic forces of physical and chemical nature allow the fluid to be released through the protecting layer of film. By focusing these forces on the enamel's surface, we can generate negative pressure and thus speed up the process. Similarly, the fluid's release rate can also be influenced by electric current (electroosmosis), though this process is better documented and studied.

Deep fissures are always full of dental plaque. Therefore it would be wrong to assume that the calcium and phosphorus salts go all the way through the whole fissure in order to ***selectively*** reach a place that seems highly inaccessible in the first place. The precipitation spot for the salts is a place covered in unwiped deposits, where dental fluid trickles from almost every direction. The only direction from which nothing comes forth is the outside,

where the fissure's opening is. The stratification vector is pointing outside, furthering the outside layers and their microflora. This is a genuine manifestation of the natural sealing of fissures, one of the most important adaptive processes happening in the tooth and on its surface. They undoubtedly target the exclusion of microbial flora and dental plaque. The sealing is a slow process of counteracting the efforts made by microorganisms to get inside the enamel.

We should note that the majority of these depositions is made up of 80% microbial mass, colonies of various microorganisms, mainly streptococcus. The water found in the microbial cells and in the moisturizing membrane of protein matrices and glycoproteins, accounts for about 90% of the mass of the said structure. The latter contains the metabolic byproducts of microorganisms and precipitates (deposits) of organic salivary components. It consists of salivary proteins, glycoproteins, synthesized with the help of sialic acids. A significant part of those consists of extracellular bacterial polysaccharides (dextrans, levans, mutans).

Taking into consideration the usual concentration of microbial cells in a millilitre of fluid and that the fluid's movement speed is around 1 ml/min, we can say that the environment's microbial output is in the region of a couple of billion microorganisms per minute.

The dental pump's anti-plaque effect can be explained, more or less, through as simple microbiological investigation. The filter paper disks soaked in dental fluid coming from the electroosmotic fraction near the anod inhibited the development of microbial colonies in all the experiments, without any exceptions. When the same disks were soaked in other solutions, even in oral fluid, there was no noticeable inhibition in the development of microbial colonies.

This data clearly point that the dental fluid's is able to oppose the formation of dental plaque. The pump's activity is without a doubt, one of the most important mechanisms making up the enamel's antimicrobial resistance. A decrease in its activity leads to the stagnation of the hydraulic system in all of the tooth's hard tissues, which in turn causes the dental fluid to lose its bactericidal properties, even if it still has amino acids in it. Moreover, if the liquid is stationary, it cannot perform the action of mechanical cleaning anymore, leaving the enamel's micropores and microlesions "unwashed". This explains the plaque's tendency to colonise the enamel and penetrate the subsuperficial layers.

16. The Tooth's Functional Activity and its Pathology

If we admit that the hard dental tissues are regular tissues of the organism, there is no reasons for us to give up a basic medical principle, that of being able to distinguish the nosological forms that we are interested in. As we have already seen, the tooth is one of the human body's organs, constituting a whole structure. As a consequence, the dental tissues can also be affected by various diseases or anomalies, just like any other organ or tissue of any organism. The tooth's pathological state should be conceptually divided into three categories of fundamentally different origins: **"developmental defects", "lesions" and "diseases".** The first category consists of disorders (including mutations) caused by harmful events that took place in the past and imprinted their effect during organogenesis. The second category encompasses the immediate effects of harmful events that took place in the recent past or in the present. The last category refers to the current pathological process that influences the development of future events. (In this case, the harmful factors disappear, but the pathological processes continue by themselves, creating a vicious cycle, a positive feedback.)

Schematically, the relationship between these three categories can take the shape of a triangle, each tip representing a category. Clinical practice is much more diverse than this, each of the listed category being accompanied by intermediary, transitory subcategories, but all of them are contained between these three points.

Surely, the respective diagram does not refer solely to our dental problems, but to all the pathologies of the human body. This chance to simultaneously visualize all the clinical possibilities and their dynamics in authentic pathological circumstances is extremely important to us.

It should be noted that the first category of pathologies is associated with subtle disorders caused by informational errors, the second one- with a brutal energetic impact coming from the outside, and the third one- with specific internal processes, appearing in biological systems, tissues, organs or in the entire organism and creating the necessary conditions for the formation of vicious pathological cycles.

Now, we will add some observations concerning the first category, "developmental defects". It is important to realise that we are not facing just a simple event, but rather the consequence of numerous events that impeded the optimal development intended by genetics. However, it is clear that there is no clear limit between developmental defects and the normal, intended version, both being divided into hereditary (genetic) and congenital (non-genetic). The array of conditions described in here is rather wide- from severe structural and functional abnormalities such as systemic disorders to localized cellular disorders. These states are largely determined by chain of events occurring after the exposure to pathogens or other harmful factors.

Following this classification principle, automatically implies the need to make some adjustments in dentistry, especially when it comes to our view of "developmental defects". It becomes clear that it is wrong to analyse the forms of dental fluorosis separately from hypoplasia, as both of them are disorders belonging to a group of congenital malformation of hard tissues called "hypoplasia". In essence, fluorosis should be named "fluoric hypoplasia". Additionally, the isolation of fluorosis from the hypoplasia group confuses the new students who will think of them as two fundamentally different conditions.

If we admit that fluorosis is a typical hypoplasia caused by some specific circumstances, if we cease to see it as a disease, then it is certain that it is some special kind of disorder. It is indeed a form of dysplasia, i.e. a mark left by an event from the

past, but not a disease in any case. Such a perspective would allow us to adopt a correct approach towards the prophylaxis of dental fluorosis and other disorders using fluorine based substances.

The enamel's mechanical deterioration has a dialectic character. Moderate mechanical deterioration is actually favourable for the tooth and respectively for the entire organism, triggering a chain of the tooth's defensive reactions and strengthening its structure as a result.

Indeed, by examining the exterior, it is difficult to differentiate the defects caused by mechanical actions from the ones caused by diseases. The reason is obvious. Compared to other tissues, the dental tissues are more rigid, thus all the pathologies are limited to a single phenomenon- the visible or invisible loss of tissues and the formation of defects. This is the only course a pathology can take in a tooth. The art of dentistry implies being able to differentiate between these very similar malformations, be they "developmental defects" (dysplasia), lesions (abrasion) or diseases (dystrophic defects, caries).

17. The Beginnings of a Tooth's Pathophysiology

By definition, inflammatory, neoplastic or degenerative pathological processes cannot occur in a tooth's hard tissues. There is only one known type of pathological processes typical of mineralized tissues, and it can be described as "acid-microbial degradation". Consequently, there is also a defensive reaction that is known to us: the formation of a dental pump in the hypermineralization zone. Thus, when discussing the beginnings of a tooth's pathophysiology, we should refer to the interaction between two biological systems: ***the tooth-organ and the microbial colonies surrounding the crown.***

It should be noted that the organism's self defence should not be interpreted solely as an attempt to keep its internal environment clean. It is much more complicated than this. Not only do the microorganisms try to penetrate the organisms internal environment, but they also constantly succeed in it. The smallest damage, such as the removal of a deciduous tooth or intensive brushing can open the gates to an invasion, which leads to a transitory bacteraemia lasting a couple of days. Some systemic factors such as: stress, shock, hemodynamic instability, etc. also contribute to this event. The organism needs this migration of microorganisms that ensures the proper operation of a special intercellular informational network regulated by cytokines. This network feeds upon "bacterial modulines", small molecules originating from microbes, the byproducts of a microorganism's disintegration.

For now, we willnot consider it, as there is something else that interests us. It is well known that the penetration of microbes into the organism's internal environment is a perfectly normal phenomenon. This process is continuous and takes place

even if there are no local lesions to allow it. It is impossible for the teguments equipped with pores to remain uninfected while surrounded by microorganisms. It is a natural, normal physiological occurrence. The penetration transforms into a pathology only if the flora finds a way to auto-reproduce and quickly multiply. The enamel, with its significant porosity (10% of its volume) and a multitude of fissures of various sizes, cannot be sterile, not even under optimal circumstances.

As it has been mentioned earlier, there cannot be inflammatory, degenerative, allergic or neoplastic processes in the enamel, as it does not posses cellular elements. The only way it can interact and be affected by the environment is through different types of confrontation with the microflora along the contact surface. The microflora's strategy is to head for the nutrients while getting as far as possible from the antimicrobial compounds found in the oral fluid, such as enzymes, lysozyme, antibodies, leucocytes and competitive species. The superficial pores found in the enamel host pure nutrients in the form of a saline solution of amino acids. Therefore, the purpose behind the migration of microflora is to get away from the oral fluid and closer to the adamantine fluid.

The enamel's "strategy" to combat the described phenomenon as part of the macro organism, is more than obvious. It ensures that the internal environment stays relatively clean and prevents a mass invasion of micro organisms from happening by alienating and rejecting them. All the interactions occurring at the contact surface are determined by the biological abilities of both parties.

In the absolute majority of cases, the teeth of mammals withstand being in the immediate proximity of aggressive microflora and despite the latter penetrating the enamel's superficial layers, they can preserve their integrity and functionality.

In the case of some mammals, the microflora is moved away as a result of the cavities being filled up with mineral precipitates. Thus, the demarcation line moves towards the exterior and the microbial communities are pushed as far as possible.

A similar phenomenon occurs in human teeth, as well. Over time, layers upon layers of mineral depositions fill up all the fissures and dents on the tooth's surface, continuously growing towards the exterior. However, the signs of this rather interesting phenomenon have no clinical significance and are occasionally found on the polished surface of healthy extracted teeth.

For dental specialists the most important one is obviously the third scenario of interaction between the microbial community and the dental tissues, when the latter are overwhelmed by the flora's constant aggressiveness and begin their decay. This happens, for example, in the case of a "smooth" cuneiform defect or even in the case of a dental caries- one of the most spread out contemporary diseases. In the following paragraphs, based on the acquired knowledge, we will briefly analyse each of these nosological forms.

17.1. Smooth Defects of Hard Dental Tissues

The opinions concerning cuneiform defect and those similar to it are quite varied. Nevertheless, most of them agree upon the fact of them being a result of multiple pathogenic factors. The adepts of this theory mainly begin their lists of harmful factors with terms such as "the brush's harshness", "the patient's exceeding force", "the composition of hygienic products". As a second leading harmful factor, they usually list the abrasion caused by the said hygienic products, especially the dental powders, which, by the way, have been out of daily use for a very long time.

Indeed, these lesions closely resemble a polished zone. Interestingly enough, the same defect can be found among people who are not exactly best friends with the toothbrush, it can be found even among some animals- rodents, predators.

The cuneiform defect is indeed similar to a mark left by some polishing instrument, injuring an entire group of teeth. The localisa-

tion, shape and depth of the defect is identical among all the neighbouring teeth. We have too little proof to affirm that these are the result of a mechanical action. No one has found scratch marks characteristic of grinding. Therefore, this theory should be excluded.

In order to reconcile the opposing opinions, a new theory has emerged as a compromise, albeit a more unrealistic one: the mechanical abrasion of teeth caused by the mucosa of the oral cavity!

Finally, there is yet another argument against the hypothesis of mechanical abrasion, this one being backed up by experimental data obtained by mechanically grinding extracted teeth with a toothbrush. It took a great deal of time and effort, and in the end, they obtained a groove shaped defect with rounded edges. Most importantly, there were lots of longitudinal scratches on the defect's surface.

The second most popular theory by number of supporters would be the chemical theory. This one explain neither the localisation nor the distinctive shape of the defect. The main counter argument against the mechano-chemical theories is the presence of a constant deposits on the affected surfaces. Akin to bacterial plaque, these are not easily removed with a mere stroke of the toothbrush. Undeniably, the chemical or mechanical agent has to pass through this layer of deposits before damaging the subjacent hard dental tissues. In reality, however, it is the subjacent tissue that is subjected to damage. At the same time, the relatively prominent neighboring surfaces remain clean of any deposits. It is namely these surfaces that remain intact, even though they are subjected to more mechanical traumatisms.

It seems beyond any doubt that the deposits we are talking about are not here to protect the tooth from developing this defect, but rather to favourise it. The presence of bacterial plaque on the defect's surface has the same pathogenetic value as its presence in a carious cavity. The difference lies within the microscopic scale of the process.

The primary smooth defects are characterized by the emergence of some superficial linear defects. As opposed to the carious process where we have a subsuperficial demineralization while the superficial layers keep their integrity intact, here we have the what the former does not: the stratified destruction of superficial layers. The progressive penetration of acidic microorganisms into the enamel, layer by layer, lead to the acidic dissolution of mineral components and the fermentation of the organic ones.

The cause of either scenario is quite evident. The still immature enamel, being more lax and at the same time protected by the compact external layer, allows for the development of the subsuperficial process, whereas the superficial process develops in the mature, homogenous enamel.

An increasing number of people adhere to the theory of abfraction in the etiology of the cuneiform defect. This theory succeeds in convincingly explaining how the deterioration of superficial enamel in the neck's region takes place under the influence of an occlusal trauma. But how does the defect itself take shape ?

How the cuneiform defect develops, with its proper geometrical shape, through the linkage of two surfaces in a straight line is fairly easy to explain. The line connecting these two surfaces (a groove of sorts) is the shortest path through which the cervicular fluid from the gingival papillae (distal and mesial) is being drained out. It is these areas that fill up with fluid when the defect's surface is dried out. The groove is always filled with cervicular fluid- a rather favorable environment for the development of microorganisms. It is a safe haven, where the colonies are protected from natural and artificial hygienic factors.

The perspiration of cervicular fluid in patients with a cuneiform defect is considerbaly larger. It gets particularly stronger (10 times stronger) in cases of mechanical, osmotic or electrical excitation of a living tooth, although it remains unchanged for devitalized teeth. (This proves that dental fluid makes up a portion of the cervicular fluid).

Regardless of how the above data is tackled, we have to finally eliminate the question concerning the essence of the cuneiform defect. The described phenomenon refers to diseases and not lesions.

This also applies to other dental dystrophies- erosions, vertical defects. The colouring of the surfaces affected by the said defects shows that they are covered with soft organic deposits, which, in turn, again points towards their microbial genesis. To conclude, if these defects are covered with soft organic deposits, regardless of their geometrical shape, they should be cataloged as non abrasive (non traumatic) pathologies and more precisely, of microbial origin. The defect's geometrical shape in this case is only a secondary concern. This kind of tissue loss cannot only have a flat, cuneiform or grooved shape, but also a hollow one.

Every practitioner has encountered such a defect. The process is localised on the masticatory surface of molars and premolars subjected to abrasion. As it seems, these defects do not even have an official nomenclature or name, but it obviously does not reflect upon their prevalence. Steep walls, round or oval floor- this is the clinical and morphological picture of this "bucket" shaped defect. The tentative to explain its development as a result of occlusal forces is unjustified. The defect's floor does not even come into contact with the antagonist teeth. At the same time, upon dyeing, the defect's surface gets well coloured, which proves that it is covered with soft deposits.

It should be remarked that the previously mentioned interpretation of smooth defects fits quite well into our knowledge of the relationship between the oral cavity's microorganisms and its organs. In the oral cavity, on the external surface of the oral mucosa, in the gingival grooves and on the enamel's surface, there is an ever present struggle between two forces: the body's own forces and those of the microbial flora. Eventually, after a certain amount of functional abrasion, the tooth's dentine and cement will have also joined this confrontation. Just like in any case of violent opposition, the equilibrium can either remain unchanged for an un-

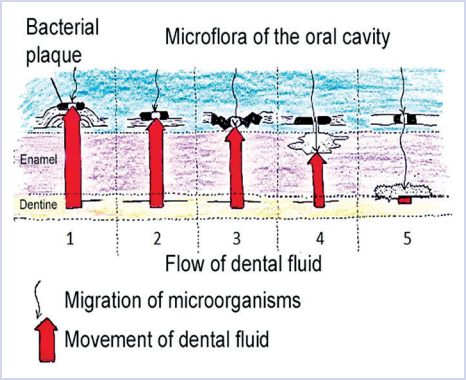

Fig 11. Types of interaction between the microflora of the oral cavity and dental fluid:

1 – the distancing of microflora in the sealing of fissures;
2 – biological balance;
3 – "smooth" defect;
4 – subsuperficial demineralisation;
5 – penetrant caries.

limited amount of time or it can shift in favour of one of the belligerent parties. In such situations, the state of balance, hard coded into the genetic program of every organism can be observed in the majority of cases. The confrontation is essentially at halt, the only variance being tied to functional wear. (Fig. 11)

Another type of interaction is associated with the tooth's capacity to extend its initial anatomical boundaries. This is what occurs during the so-called "natural sealing" of fissures- the formation of mineralized structures upon the floor. In this particular situation, the frontline moves towards the tooth's functional surface. The last form of interaction with the microbial environment is the one referring to diseases. Generally, this is a process in which the frontline moves inwards, i.e. inside the dental tissues. There are two possible scenarios: subsuperficial destruction (subversive) and surface destruction (progressive). On the one hand, we have different types of dental caries and on the other hand- various defects with smooth walls- the subject of our discussion.

All the smooth defects outlined above should certainly be cataloged as "diseases" and be clearly differentiated from the similarly looking abrasions- "lesions".

17.2. Dental caries

In order to describe this main disorder of dentistry (dental caries in its preclinical stage) in a more theatrical manner, we will tackle its most expressive clinical version.

We are talking about the "penetrating" caries.

This version of dental caries is well known and thoroughly described by patients who claim that the dentist aimlessly "drilled" their absolutely healthy tooth.

In clinics, we genuinely have cases when upon touching a seemingly healthy tooth or with slight hints of incipient caries located deep into the fissures, where the dental probe can barely even fit, the drill would collapse into a hollow space, reaching hard tissues located by the neck's level.

From an anatomo-pathological point of view, we get a rather intriguing picture. One, or more frequently two canals pass through the whole thickness of the enamel- the access paths of pathogens. These canals reach the enamel-dentine junction, or rather the place where it used to be before the disease. The dentine and the enamel are separated by a macroscopic fissure. This fissure reaches widths of hundredths of microns and is extending in all directions from the connections formed by the mentioned canals all the way to the areas near the tooth's neck.

This enormous strand is filled with soft detritus, damp and swarming with actively multiplying microorganisms. The prepulpar dentine transforms into transparent dentine and a noticeable layer of tertiary dentine can be observed at the border with the pulpar chamber. The enamel is represented by remains of prismatic structures that sometimes have mineralization zones.

This picture is invisible to dentists but beautifully described by morphologists. Being in possession of the said information, it is fairly easy to reconstruct the chain of events that led to such

damage and subsequently restore the lost tissues. The distinctive phenomenon is obviously related to these canals tying the fissure to the enamel-dentine junction.

It goes without saying that these canals, reaching depths of about 2 millimetres and diameters of hundreths of millimetres will be the main actors of our stage. The classic mechanism of subsuperficial demineralization, frequently demonstrated on molds cannot occur in this case. As it is well known, the acid is produced by microorganism using carbohydrates that come from the oral environment. The microbes cannot exert a targeted action upon a fixed point, far away from the oral environment with its energy resources. However, this is what actually happens. They target a single point, moving in a single direction and relentlessly obliterating the structures in their way. Through chemical interactions, they drill a long, narrow canal, striving to reach the enamel-dentine junction. Such a scenario, in which the acids would "drill" a canal for the microorganism and then for the carbohydrates to follow suit, seems absolutely incredible.

What exactly happens in such caries? The answer is quite simple and logical: as a response to the weakening control of the tooth pump, the microorganisms get the opportunity to grow towards the source of amino acidic aliments, migrating and colonising the orifices of the canals. Having got there, they secrete acids and widen the pores, moving ever forward in accordance with what their genetic programming tells them to do. In comparison to other forms of caries, there is no adequate defensive reaction involved, thus favourising a rapid colonisation of the enamel-dentine junction.

Reaching the abundant source of fluid coming from the dental pulp, the microorganisms quickly populate the zone tangentially to the junction's outline. As the process goes on, more and more enamel is isolated from the dental fluid. The pulp mobilises its own protection mechanisms, thus obliterating the dentinal canaliculi and forming a layer of tertiary dentine. Simultaneously,

the microorganisms release acids and ferments that travel along the concentration gradient towards the tooth's exterior, dissolving the enamel's mineral components. In the peripheral zones of the destruction, the dental fluid would continue to flow through the dentinal canaliculi for some time, consolidating the viability of the microorganisms as a result.

This would be the only logical explanation tackling the pathogenesis of "penetrating" caries. However, a new question arises: is it different from a banal, regular caries, i.e. are their mechanisms cardinally different ? Clinical experience proves to us that they are not. We have several intermediate versions of dental caries, but we cannot speak of different nosologies. We are forced to mention that regular and penetrating caries develop following the same scenario. The idea of the primary lesion being sterile is merely a hypothesis without sufficient proof. De facto, microorganisms do not exert their action remotely and do not destroy the enamel's organic and mineral components. The primary process is the colonisation of the enamel's hollow points, such as the superficial pores, the spaces between rhythmic structures, i.e. the spaces occupied by dental fluid (the subject of chemotaxis, the chemical attraction of microorganisms).

It is namely by this modus operandi- the initial penetration and the ulterior spreading through the stratified tissues- that the process always evolves. The differences in various clinical pictures depend upon the depth reached by the microorganisms before the tooth belatedly activates its systems of organised mineralisation. If the germination is stopped early, when the microorganisms merely reach the pores, the caries will not develop at all, if it happens by the time they reach the subsuperficial zones, we will get a mineralisation of the carious defect. If the penetration is stopped upon reaching even deeper layers, a superficial caries will develop that will subsequently open into the environment of the oral cavity.

From the point of view of pathological anatomy, we can always see the traces of demineralisation following the outlines of rhythmical structures. The currently analyzed form of penetrating caries occurs only when the microbial flora encounters an organized riposte solely in the deep layers of the enamel, including the enamel-dentine junction and the adjacent superficial dentine. The ulterior tangential spreading of the destructive process will then follow along this demarcation line.

Therefore, in all versions of carious destruction of the enamel, the penetration and spreading of microflora takes place through the enamel's transporting structures that have a radial placement and a centrifugal direction. The basic destructive activity of microflora has a tangential orientation, almost perpendicular to that of the access paths. What this section represents is rather clear to us.

It is fairly well known that the enamel is stratified. The stratification reflects the daily and monthly cycles of tissue formation. Among relatively well mineralised layers, there are also softer ones- forming the enamel's porosity. These layers clearly manifest themselves during the carious demineralisation, whereas during the chemical demineralisation in laboratories, their role is virtually unseen.

These results can be easily explained through the fact that caries imply the primary colonisation of the enamel's transporting structures by the microflora. Thus, it is impossible to attribute to it the traditional explanation of "layered" demineralisation, as it involves a sterile destruction caused by acid. Following this scenario, only one single subsuperficial demineralization centre can occur, situated immediately underneath the superficial compact layer and parallel to it . However, the occurrence of multiple parallel "storeyed" layers of demineralisation, substantially far apart from each other and the bacterial plaque can only be explained by the spreading of the destructive process from a primary outbreak zone that appeared at some point in the enamel's layers.

In other words, in order to penetrate the deep lax layers, the pathogenic agent needs access paths. This is possible only through a vertical tunnel, "a pipe" that goes through the well mineralised layers. The possibility to find them in a microscopic preparation of a carious outbreak zone is quite difficult and possible only when they coincide with the axis of the micro section.

The idea outlined above merges into a single picture all the known scenarios of interaction between the enamel and the microbial flora populating its surface and therein lies its fundamental advantage. This approach gives us the possibility to nontraditionally prevent the tooth's destruction by means of countering the microflora's tendency to colonise the depths of the tooth. The technical principles of solving problems of this nature are obvious and perhaps will lead to exceptional practical results in the foreseeable future: the affected zones are dried and then drenched in a strong antiseptic of prolonged action. We are emphasizing upon the fact that such techniques are not complicated from a technical standpoint. The problem lies in the economical cooperation between the patient and the doctor while solving the issue of stopping the destructive process within the tooth. This technique of preventing the carious process will only be used when the dentists will deem convenient to prevent the caries rather than eliminate its consequences.

It is worth noting that this position is shared by few researchers. More popular is the theory stating that the beginning of the carious process coincides with the beginning of the subsuperficial demineralisation and it is thought that the cause lies in the activity of microbial communities residing in the dental plaque. Ergo, it is considered that the enamel is primarily destroyed by acids over a long period of time, all the while remaining sterile.

To fully realise how great the difference between these two points of view is, we have to remember the three pathological processes described at the beginning of this chapter. Caries is

obviously not an innate defect, yet the question persists whether it can be considered an actual disease or merely a chronic condition. Herein lies the query which will help us perceive the difference between these two theories. However, considering the incontestable arguments pointing towards the existence of the tooth pump as well as the fact that the liquid fraction drips through the enamel and it renews several times throughout the day and seeing the tooth as an open biological system, then the question of passive acid demineralisation will disappear by itself.

Caries is an unusual disease that develops as a result of disturbed balance between the antagonistic biological forces: the micro and macroorganisms.

Another, more prevalent theory is the perception of caries that discards the information outlined above about the tooth's physiology. In this case, the subsuperficial demineralisation of the tooth is carried out by the prolonged influence of microbial acids upon tissues. Initially, it is no more than a chemical trauma, which leads to the formation of a sterile subsuperficial defect. The microorganisms remain outside the tissues and the defect is a secondary effect of their remote activity (the tissues simply find themselves in the radius of their activity), meaning this secondary effect is not tied to the biological purpose of their activity. In this case, the colonisation of the hotbed begins only after its opening. It is then that the microbial invasion of the enamel and dentine commences- when the organism responds with a defensive reaction by creating a defensive hypermineralisation zone around the hotbed. It is at this point that we can talk about the onset of the disease itself with its vicious circle, in certain unfavourable even being able to lead to the death of the individual. And it all started with a chemical lesion, not different from a corrosive process found in other materials. It is understood by itself that these different approaches to caries call upon different preventive measures. In one case, we have the prophylaxis of a disease, in another one- the prophylaxis of chemical corrosion.

Let us remember that any condition that has a progressive dynamic, initially asymptomatic, is characterised by the staging of processes. Each stage requires progressively more radical and complicated treatment. Firstly, we have the pre morbid stage, which merely requires corrective actions, mainly with no drugs involved. Then we have the disease itself, therapeutically treated and lastly, the stage of irreversible changes requiring surgical treatment.

Being a chronic process, dental caries is different because we diagnose it only when it has reacned the stage where it needs surgical treatment. This treatment consists in removing all the irreversibly changed tissues from the carious hotbed. The detection and especially the conservative treatment of caries in its macula stage is fairly rare. Concerning the pre morbid stage, not only is it not diagnosed, but many authors do not acknowledge it at all. In the absence of a suitable correction, this state will usually progress into an incipient and/or superficial caries.

Besides the question of how caries start out, it is equally important to ask ourselves WHEN it begins. Indeed, the common vision of caries explains how it occurs with a fair number of arguments.

It is supposed that the microorganisms from the enamel's surface cause an acid attack . However, it is still unclear what determines the time of initiation.

If there is an attack, what serves as an initiation signal? The experts are prone to believe that there are no changes in the tooth or the enamel before the initiation point, and neither can anything happen due to the fact that the properties of the materials are unchanged. There are certain reasons why they have come to such conclusions. The maturation of enamel, mineralisation, the accumulation of fluorapatite, these are all well known processes, being slow and most importantly unidirectional. They span across years and decades and are

always directed towards the accumulation of mineral mass. Unlike the bone, a sudden endogenous demineralisation of the enamel is something completely unrealistic, i.e. an occurrence never seen before. Therefore, it is logical to assume that in such circumstances, the beginning of the process is determined by an event taking place outside the tooth. It is thought that this event is triggered by the activation of the plaque's microorganisms, the ones found directly on the surface of the enamel. The question of when this peculliar triggering event occurs in the oral cavity remains unanswered.

The hygienic habits and addiction to sweets are individual habits that usually remain unchanged throughout the whole life. These cannot suddenly change, thus the question of when the carious process begins is still open. We received the answer to the question of WHAT can happen at any stage of life. However, classical cariology is still unable to answer WHEN. It also cannot explain why sometimes this WHEN refers to a single tooth, sometimes to multiple teeth (carious disease) and sometimes to none at all. The waters are unclear even for the adepts of the theory concerning endogenous events that lead to pathogenic situations. Researchers have tried to detect some signs indicating towards the organism's "struggle", which may be associated with the occurrence of dental caries. Previously, it was widely thought that caries begin with this so called "general struggle" of the entire organism, but the attempts to explain the nature of this problem failed. No general diseases capable of triggering the events leading to caries were discovered. Certain connections between caries and systemic disorders do not arouse any doubts. For example, we have actinic caries, xerostomia caries, yet these do not bring any more clarity to our questions. Up to this moment, no one has managed to prove that caries are preceded by certain events taking place in the entire organism.

Obviously, dental caries, just like any other disease is connected through a myriad of threads to a network of causes

and consequences. This once again confirms the banal truth about the dependence between everything that happens inside and around us. However, the situation did not become clearer. For us, the practitioners and our patients alike, these different hypotheses, however attractive and beautiful they may be, are ultimately useless.

Regardless of whether the dentist acknowledges the ideas outlined above, he will always perform the same sequence of steps: he will detect the affected zone, remove the affected tissue and restore the said zone. The dentist's equipment and the materials he uses are in no way related to the successes of cariology. We should humbly admit that everything the dentists use to carry out their activity is the product of a chemist's or an engineer's work. The medical discipline merely approves and implements their results. In dentistry, the power belongs to technologies.

The clinician's return to actual medicine and the increase of their possibilities would be attainable by introducing into their line of thought all the available knowledge regarding a tooth's normal and pathological physiology. Please note that the volume of dental fluid make up 1/10! The subsuperficial layer, where the volume of nutritive fluid is maximal, becomes the biological target of microorganisms. It is here that all the primary events occur, i.e.the metabolisation of the enamel's liquid components.

The opinion concerning the primary destruction of minerals is not biologically justified: the cariogenic microorganisms are not lithotrophs. Before everything else, it is the dental fluid that should be metabolised- it is easy to use and its energetic resources (from the aggressor's point of view) are unlimited. The sequence in which the enamel is metabolised is rather obvious: the fluid first and then the organic and inorganic structures. Thus, the fluid has a double role in the pathogenesis of caries. As a chemical compound, it serves as a subject of microbial trophotaxis; as a centrifugal flow it serves as a defensive mechanism source for

the enamel's pores and fissures. It becomes clear that the tooth's devitalisation deprives the enamel of its taxis component and makes the tooth resistant to caries ("the mummy cannot get sick"). Conversely, the presence of wide pores and/or a slow flow of dental fluid turns the deep layers of the enamel into something easily accessible. The narrowing of the pores and the acceleration of the flow's rate could lead to an increased resistance to acids and microbes.

By accepting this point of view, we become aware that the moment we were looking for- the debut of caries as a pre-disease, is the moment when the functional state of the enamel starts to deteriorate, the latter being caused by physiological conditions, including biorhythms. Clearly, such an approach towards the pre-morbid stage is merely the **path** to solving the issue of dental caries and not the ultimate solution to the underlying problem. The questions concerning the prophylactic methods and how to control the occurrence of this state should be tied to pre-caries.

Now, some words about the prophylaxis of pre-caries. We should note that caries is, without a doubt the result of a perturbation in the natural balance between the activity of microorganisms and the enamel's possibilities to withstand the said activity. Everyone regards this as an axiomatic statement. The main strategy of repelling the carious activity encompasses two different approaches: to reduce the aggressiveness of microorganisms or to increase the enamel's resistance.

Thanks to our sufficient understanding of the various processes revolving around the activity of microorganisms, we can almost fully exploit the first approach.

The second approach presents a much narrower spectrum of uses. They are usually limited to increasing the enamel's resistance, reducing its porosity and supplying the mineral components it lacks. It has been known for a long time that the enamel, being a tissue of a living organism, is not a passive object and cannot be changed by merely injecting it with "useful"

components. As we have convinced ourselves, it possesses a wide spectrum of various mechanisms, each of them implying a certain array of parameters, among which the most important one is the superficial acid resistance. This could fundamentally change the whole ideology behind the prophylaxis of caries. The new approach presents itself with substantial possibilities, because it allows us to seize control of the dynamics of the fluctuations in acid resistance. These aspects are extremely important when dealing with young individuals, prone to caries and whose enamel has a high porosity index.

Regardless of what prophylactic element we may refer to, they all contribute to changing a certain parameter, either in the oral environment or in the hard dental tissues. This is exactly how the specialists in different fields of engineering work, studying the materials and fighting off the corrosion in the built structures. In both cases, the hope relies on the fact that through certain methods, the aggressiveness of the environment will get to match the resistance of the material.

It goes without saying that the physiological (biological) aproach described by us and based on the tooth's physiology, should not and cannot exclude today's methods practised in the prophylaxis of dental caries. Proper oral hygiene, a reasonable intake of easily digestible carbohydrates and last but not least assuring the developing child with optimal conditions, retain their value as general nonclinical recommendations suggested to any patient. However, if we are talking about prophylaxis in the case of a particular, real person, of a certain age and gender, with an enormous array of genetic and physiological parameters, both inherited and acquired, then the more specific recommendations can be made only by a clinician specialised in prophylactics.

In theory, only the general practitioner that knows everything about his patient could appreciate when and which measure to undertake in order to assure the patient's optimal health trajectory. This trajectory, above everything else, is based upon

informational control. Clearly, the contemporary medical theory is still unable to provide doctors with recommendations that will help them detect and correct individual dysfunctions. In cariology, however, such a possibility has arisen thanks to the discovery of the enamel's functional variability. Even the first versions of the EART method, based on assessing the lesions caused by dosed acid exposure, allow us to fundamentally change our scheme of preventive measures, thus enabling us to enact concrete actions directed towards concrete patients. Our actual understanding of the essence of pathological processes paves the way for new strategies and perspectives that imply a so called "informational" approach.

These principles do not exclude material (physical) interventions, but here they have an entirely different nature. The problem is no longer confined to providing the enamel with something it lacks or removing the harmful components. The action mechanism of the employed substances is also different. For example, carbamide proved to be useless at complementing various deficits. Conversely, from a technical standpoint, they might even prove "harmful" (carbamides being good solvents for minerals). They are used solely as remedies capable of generating hydraulic signals that will in turn activate the tooth pump.

This informational action mechanism could employ many other "non material" effects to do its work, such as electrical impulses, mechanical oscillations aimed directly at the tooth or indirect stimulations, aimed at the organism's regulatory systems (for example the stimulation of biologically active zones, the short term action of large doses of ascorbic acid, etc.). In both clinical and experimental environments, the preventive effect aimed at stagnating the development of caries is not achieved through the substance's physical properties or the procedure's intensity. The effect is guaranteed by the informational action of the stimuli, how well their signal is perceived and its conformity in relation to the concrete state of the organism.

If the said local or systemic procedures prove to be adequate, the tooth pump will start its activity and we will be rewarded with an increase in the enamel's acid resistance. In such conditions, a definite clinical effect is achieved – a reduction in the estimated prevalence of dental caries.

It is important to note that this kind of preventive treatment leaves within the tooth (and the organism) a certain trace in the form of a collection of information, necessary for the subsequent homeostatic reactions. Following this, the tooth knows better where to direct its energy (hydraulic) flow first. Thus, a physiological maturity of sorts is developed, which greatly enhances the organism's defensive possibilities.

The main idea behind these schemes of preventive treatment is represented by informational interactions. Within the chains of the organism's ever functioning self-regulatory mechanisms, a new decision making link emerges – the doctor. By summarising all the known information on the current state of the patient's organism and on the enamel's active resistance mechanism (for example through the EART), the doctor makes a decision and gets involved into the informational flow. It induces additional extra biological impulses that will in turn activate the systemic and (or) local energy flows, selectively increasing the enamel's acid resistance at the necessary moment. This is the essence of the physiological prevention of caries.

However primitive the current technology used for these methods may be, it proves to be remarkably efficient even in its infantile state. That is because the poorly developed technology plays an insignificant role here, it is all thanks to an adequate approach.

18. Tooth Health and the Human Health *

The development of science, the vertiginous increase in the amount of scientific knowledge will inevitably lead to narrower specialisations. The more thorough the information possessed by a discipline is, the less its followers know about the other disciplines, which increases the risk of a professional isolation. This divergence could also be attributed to medical disciplines. The intention to oppose this phenomenon is just as useless as opposing oneself to the technical-scientific progress, but neither can we completely accept it, because that would mean losing our integrative vision and overall practical approach.

Odontophysiology – the tooth's physiology that we are currently discussing, being a fundamental discipline, may and should serve as a link between the dentist's specialised knowledge and the general problems of medicine, including those of other medical disciplines.

Dentistry, as a medical discipline, standing on equal footing with the others, acquires new qualities and possibilities in which it can "guide" people's health. Such model of integration should, to a certain degree, influence the whole practical and fundamental medicine.

All the information on tooth physiology presented in the previous chapters, convinces us that the tooth is not a semi-living formation, but an ordinary organ characterised by functional mobility. Upon being fully acknowledged, this simple circumstance will force our specialty to return to the field of medicine. Clearly, the process will not be a simple one, it will be painful, slow, which is why we are unable to foresee how it will turn out. Additionally, we cannot overlook the most difficult and perhaps most important question of it all – **what contribution could dentistry make to medicine?**

The beginning of this millennium was met with an unexpected rush of information tackling the dependence between dental conditions and vascular disorders. If we take into consideration that it is namely these that make up the leading causes of death, then the value of this information is incontestable for the future of humanity.

The impact of dental health upon general health is not a new discovery. Many decades ago, it was proved that chronic inflammation hotbeds from the dento-alveolar zone could initiate and maintain septic states. Based on this information, a strict directive (that unfortunately is mainly forgotten nowadays) emerged that required the sanitation of the oral cavity before planned surgeries, even though at the time it was still believed that dental health influenced only a small number of general conditions. The data we have in the 21st century tells us about much more serious conditions.

It is already known that certain signs, such as periodontal inflammations, caries and its complications, hindered dental eruption or the number of extracted teeth are closely related to the state of the cerebral and cardiac blood vessels. In a still inexplicable way, the occurrence and development of fatal atherosclerotic plaques depends more on the state of an individual's oral cavity than it depends on his diet! Besides the obvious, the incidence of strokes and fatal heart attacks is undoubtedly tied to dental health as well. These data are not presented by some superficial and insubstantial publications. The studies were carried out by reputable authors who took decades to observe thousands of patients. The latter confirms the scientific veracity behind the interdependences we have discussed.

In short, the factual material states the following: the more affected are the organs of the oral cavity are, the more likely and more fatal are the ensuring vascular catastrophes (strokes and heart attacks). Currently, the interdependence between atherosclerotic processes and inflammations is considered to

be a proven fact. The only thing we lack is a unanimous opinion concerning the etiology, severity and localization: the dysfunction of the endothelium, associated with an inflammatory process, subclinical inflammation, the inflammation of the blood vessel walls near the atherosclerotic plaque.

Now, we can finally tackle the most interesting aspect: how can we perceive this apparently mysterious connection? There are two diametrically opposed answers to this questions. The first one ties periodontitis and dental caries to an improper oral hygiene. These will in turn create chronic inflammation hotbeds, which will eventually favour the development of atherosclerotic plaques, ultimately leading to a fatal and most unfortunate result. We can read about this in "The Oral Health Bible". Perhaps, the advertisement companies will begin using phrases such as "This toothbrush will not only reward you with a sexy smile, but it will also grant you a life without strokes and heart attacks, prolonging it by a whole 10 years!" (people with a relatively good oral health have indeed a lifespan longer by about 10 years). These will perhaps convince more people to visit the dentists.

The other answer is at the opposite side of the spectrum. To better understand the problem, we will analyze other universal patterns. Within a strictly scientific approach to medicine, based on concrete proof, the proposed scheme tying oral hygiene to other diseases is merely a hypothesis with little arguments to back it up. As we have previously seen, the occurrence of caries is not even remotely close to being a simple liner process. We are now convinced that the state of the tooth depends, above everything else, upon the state of the tooth pump, which, in turn, is dependant upon the central nervous system, the hypothalamus, the endocrine activity of the parotid gland and last but not least – the vast conglomerate of mutually reciprocal processes taking place within the organism.

Undoubtedly, **periodontitis**, another condition of the contemporary man, is also fairly close to the scheme outlined

above, although not nearly as clearly defined. The endogenous factors: the physiological and pathophysiology processes play an equally important role in its development. However, the relationship between the state of the periodontium and that of the blood vessels is fundamentally different. It is fairly clear that this relationship is mediated by the organism as a whole and the predisposition to dental conditions and vascular disorders have a common origin, taking its roots from the biology of contemporary men. To document this position, we have to refer to literature tackling not the physiology of the tooth and the periodontium, but rather the physiology of individuals developing in different populational environments. We do not have this possibility yet, which is why, when discussing the subject of "Teeth and general health", we will refer only to the information confined within the limits of our specialty. Thereby, we will briefly revise the physiology of the periodontium.

The periodontium is a complex of tissues whose biological goal is to maintain the tooth in its position and evenly distribute the masticatory pressure. The functional tissue of the periodontium is the dentoalveolar ligament that predominantly works as a hydraulic shock absorber, evenly distributing the force exercised upon the tooth across the whole alveolar wall. The periodontium's hydraulic system is connected to the tooth's - the dental fluid is part of the shock absorbing fluid. Through the gingival sulcus, the tissular fluid rhythmically passes into the oral cavity, just like the enamel fluid, thusly protecting the periodontium against the infiltration of oral microflora.

Periodontal conditions are much more varied than those involving hard dental tissues. These conditions are not limited to the pathology of its acellular component- the cementum, which is only a part of the whole complex. The other tissues are ordinary cellular components, equipped with a standard complex of defensive mechanisms: blood vessels, lymphatic vessels, nerve fibres, receptors, etc. The periodontium's functional tissue- the

dentoalveolar ligament- carries out its biological purpose of a hydraulic shock absorber, being functionally and anatomically tied to other structures: the alveolar wall, cementum. On the outside, the whole ensemble is protected by the gum and its part that is stretched on the tooth- the bottom of the gingival sulcus.

The fate of the periodontal tissues and their functional state are closely related to that of the tooth. In some dental conditions, for example pulpitis, the process gradually extends into the periodontal tissues. In the case of tooth extraction, all the periodontal tissues, with the sole exception of the gum, gradually atrophy and disappear. Functionally, the tooth and its supporting structures are a whole, thus ensuring the tooth's destructive power.

The diversity of periodontal tissues, the many differences between its structures and cellular components are met with a considerable diversity of pathological conditions. Unlike hard dental tissues, these can host inflammatory reactions (including allergic reactions), tumors, dystrophic and atrophic processes. The multiple conditions are studied by a specialty called **periodontology**. For now, we will examine only one nosological form- periodontitis, that is almost as prevalent as caries. The geographical and historical patterns of these two conditions are similar. Both are diseases of affluence (lifestyle diseases). Akin to caries, periodontitis is considered to be a disease caused by poor oral hygiene, the direct cause of profound marginal periodontitis being the accumulation of tartar that traumatises the soft tissues and causes their inflammation.

There is indeed a connection between the pathology of the enamel and that of the periodontium. The tooth's devitalisation, i.e. interrupting the flow of fluid, leads to an increased resistance to caries and periodontitis. There is also a connection between the flows of dental and crevicular fluid. The most interesting of all are the distinctive relationships between the predispositions to these conditions.

The patients prone to caries are resistant to periodontitis and vice-versa. In medical literature, this phenomenon is frequently referred to as the "Dubois antagonism", after E. Dubois, who described this pattern in the 19th century. Contemporary dentistry comes to confirm this, there being groups who behave entirely according to this pattern. There is also an intermediary group, in which both conditions are found.

Not having a satisfactory explanation and being of little clinical importance, this antagonism is merely a random fact. Conceptually, such an approach is unjust, especially since it is one of dentistry's few scientifically incontestable facts.

The Dubois phenomenon indicates that the circumstances favouring the development of either condition hinder the development of the other .In other words, it is clear that there are divisive, antagonistic factors that exclude each other. Discovering these factors would help us understand their interaction and their influence upon the pathological process. Their dynamics within the confines of the current conditions would pave a new way for analysing the occurrence of various conditions in the contemporary man.

The main cause behind caries and periodontitis is considered the poor horal hygiene. To explain the antagonism in this context, we should admit the existence of two types of dental debris that would be incompatible with one another, excluding one another. Moreover, because the poor oral hygiene of prehistoric men was accompanied by a healthy periodontium and healthy teeth, we can deduce the existence of yet another type of non-aggressive debris. In other words, we should have three types of dental debris, something that does not exist in the real world.

The microbial flora is undoubtedly the active component that causes both conditions. Then, there should be different kinds of antagonistic microbial communities, incompatible with each other. Competing, they should logically cancel one another with only one condition occurring as a result (or none at all). Despite the

great number of scientific studies, such microbial communities could not be identified. As a result, we should conclude that none of these different kinds of debris and microbial communities, each of these responsible for either caries or periodontitis exist. This factual data confirms once again that the microflora is an absolutely necessary condition, but it is not responsible for causing **either** caries or periodontitis, let alone for the "antagonism".

The whole list of arguments could be repeated, this time replacing "microbial flora" with "excess of carbohydrates". This factor plays in equal role in the occurrence of both caries and periodontitis. A carbohydrate causing specifically caries or periodontitis is unreal.

We can name yet another pathogenic factor- the fluoride deficit. We do not need arguments stressing upon the great value of fluoride in the prophylaxis of caries. Some less known, but veracious data confirm its influence in the occurrence of periodontitis. It would be naïve to look out for two antagonistic components of fluoride, the deficit of which causes either caries or periodontitis. Therefore, both the excess of carbohydrates and the deficit of fluoride are important, yet non-mandatory circumstances. Moreover, they are not specific for either of these two conditions.

Lastly, there is also the undisputable role played by the decrease of functional forces exercised upon the tooth, yet not even here can we talk about different types of antagonistic forces, favouring the occurrence of one condition over the other. However, in regard to this subject we can say a bit more.

Akin to how the occurrence of these antagonistic conditions in sterile environments is impossible, so is their occurrence in circumstances lacking functional masticatory forces. Thus, we can affirm that one cannot attain health without experiencing optimal functional forces and one cannot attain disease in the absence of microorganisms. Despite all these, the causes of the analyzed antagonism remain unclear.

Only the anthropological materials can shed a bit of light upon this issue. Historically speaking, the incidence of caries, periodontitis and bite anomalies have increased simultaneously. The agrarian revolution led to the occurrence of these conditions in relatively large numbers. The industrial revolution was met with a dramatic spike. As we can see today, the postindustrial era led to a decrease in their prevalence. Because the dynamics of morbidity involved both bite anomalies with their skeletal complex and microflora dependent conditions, the questions around the changes in the viability of the microorganisms throughout these epochal changes could be dismissed.

According to the collected factual data spanning across thousands of years, the incidences of both caries and periodontitis tend to increase simultaneously (Fig. 12).

The group of people manifesting the Dubois antagonism is present in both the Neolithic and contemporary populations, but the percentage of people suffering from both conditions has considerably increased. The explanation to this lies in the

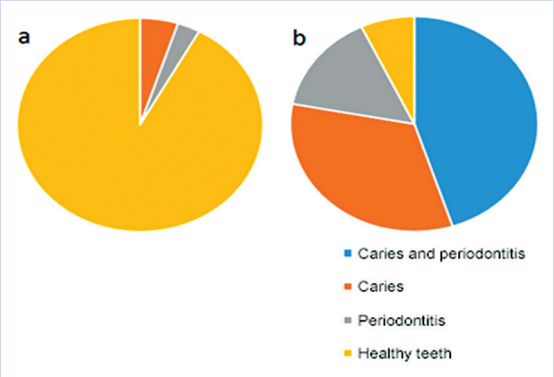

Fig. 12. The dynamics of the prevalence of caries and periodontitis

a) Neolithic era
b) Contemporary era

increased diversity of contemporary men. Their interaction with the changing conditions of the environment as a result of anthropogenic activity, led to significant changes in their reactivity and resistance and also to a considerable diversification of the individual characteristics possessed by the members of species *Homo sapiens*.

Another pattern shift speaks in favour of this explanation: in the preindustrial era, dental caries was practically absent among children. Just like periodontitis, it was the disease of the adults and the elderly. The situation varies dramatically from what we have today, when caries has become undoubtedly the disease of the children and the young.

The fact that the Dubois antagonism has persisted for thousands of years, indicates toward its immunity to the environmental conditions that have been constantly changing since the dawn of civilisation all the way to the postindustrial era. The occurrence circumstances and the nature of the discussed conditions have changed along with the lifestyle and social style of the population. Meanwhile, the prevalence of both diseases has increased tenfold. The number of healthy people was brought to a minimum, while the population group that is simultaneously affected by these both condition has all but decreased. However, the continuous persistence of people manifesting the Dubois antagonism. The latter allows us to think that the roots of this antagonism lie in our genetic predisposition. This opinion is reinforced by the great influence of heredity upon the prevalence and severity of caries, the Holzinger index beign as high as 85%. The data on periodontitis, albeit not as obvious, also indicates towards the importance of genetic factors.

Therefore, it would be safe to assume that the bipolar genetic predisposition to dental disorders is tied to the enormous genetic polymorphism of the human race (or at least the ethnic European population). We can also suppose that the "caries-positive" and "periodontitis-positive" constitutional types should have different predispositions to other diseases as well.

The path to preventive medicine is difficult and full of obstacles. The main obstacle is the deep rooted common belief that diseases always come from the outside. In this case, prophylaxis would be equivalent to the optimisation of the environmental conditions and carried out by ecologists. In reality, the environmental conditions are not enough, although one cannot deny their considerable importance.

Contemporary genetics, anthropology, immunology, developmental physiology and other medical sciences are actively gathering information on the ever growing diversity of the human race, the latter having a direct effect on our medical issues. The large number of diseases affecting humans is a consequence of humans being more and more diverse. Consequently, the human "constitutiology" will inevitably become the cornerstone of prophylactologists (prophylaxis practicing clinicians). Such a specialist will differ from other doctors through his knowledge and approach. His professional interest will not lie in the diversity of human conditions, but in the dissimilarities between humans. In the near future, his activity will imply individualised prognosis and active prevention of diseases.

Clearly, such an activity should be remunerated in accordance to the ancient Chinese principle: the honorarium shall be proportional to the number of healthy patients form the "attached contingent". Instead of chaffering about the price of various medical services (diagnostic and treatment procedures with different drugs and surgeries), it is the final result- health and the assurance of health- that will be purchased en gross. Legally, this right can be granted through "subscriptions" or healthcare plans.

Today medicine is more successful at transplanting a heart than treating rhinitis. Indeed, the small deviations from the normal and the myriad of its variants present little interest to

the clinicians and theoreticians. However, it is the study of slight, pre-morbid symptoms that could grant us most information about the biological and psychological diversity of humans.

Only a small fraction of the tens of thousands of diseases (including the most severe ones) are acknowledged and guided. An enormous number of problems remains unsolved. This is mainly due to their lack of economic gain, prophylaxis not being profitable yet. Currently, only the diagnosis and treatment of the disease is possible. Only when the specialists will be able to derive profits from health will the situation change. The necessity for such specialists will arise only when society acknowledges the problem. A new branch of practical medicine will emerge that will task the theoreticians with solving the respective problems. This will grant us the scientific, technical and organizational possibilities to counterattack the conditions affecting humanity, including the dental conditions.

19. Dental Health and the General State of the Organism*

The idea of caries and periodontitis being the result of an internal disorder could be traced back to the times of Hippocrates. The constitutional particularities of both conditions were considered even by the founder of scientific cariology- W. Miller in 1882. The role played by systemic changes in the development of these conditions was assessed by numerous reputable researchers. During the years between 1930 and 1950, this subjected was intensely studied by Soviet scientists. At the time, it was common to connect caries to a certain general disease or a harmful state of the organism.

Through the efforts of numerous researchers, a connection was discovered between caries and the systemic disorders of the central nervous system, the vitamin balance, etc. This was not merely a speculation, but a conclusion based on extensive experimental observations. They determined the metabolic disorders emerging from carbohydrate rich (cariogenic) diets and actinic lesions. Unfortunately, the attempt to raise caries and periodontitis to the rank of general conditions, i.e. "disease", has failed. The connection between dental conditions and systemic disorders was deemed too weak, ephemeral even. This allowed those who oppose this idea to discard even the concept of forecasting the occurrence of these conditions based on general systemic disorders. They say that caries and periodontitis are not diseases, our patients are not sick and dentistry is not medicine.

Gradually, the so-called "modern" theories that emerged during the congress of the International Association of Dental Research in 1940 became dominant. Their essence is well known, which is why we will not analyse them. We will merely recall their core principle: only the microorganisms of the oral

cavity are capable of causing caries and periodontitis. There are no doubts to it. At the same time, every practitioner could find among the many cases stored in his memory during his activity, an impressive number of cases that apparently oppose this idea. There are frequent cases in which the patient's oral health does not correspond to his hygiene. Sometimes we can observe paradoxical relations in some people or even entire populations. There are cases in which the patient does not even know what oral hygiene is, but neither has he ever suffered from caries. Do these occurrences contradict the microbial concept of cariogenesis? The answer is no. Microorganisms are the main and absolute requisite for cariogenesis, but the mere presence of microorganisms is not enough. The latter tells us about the "missing link" in our understanding of the disease. Herein lies our problem.

This is how the situation is assessed by the few adepts of the medical concept, including the authors of this chapters. They do not doubt the veracity of the facts provided by "contemporary" cariology. We only negate the idea of microorganisms and environmental influence being the one and only, absolute requisite for caries. It is impossible to explain the main patterns of the disease using this theory. Respectively, an actual control over the epidemics of caries and the perspective of victory remain unfeasible.

The discovery of the enamel's acid resistance phenomenon and its connection to the prediction of caries morbidity has fundamentally changed the situation, for the decrease in the activity of the tooth pump is closely tied to the "missing link". Currently, if we want to assess the connection between caries and systemic disorders, we have to give up on the stereotype concerning the link between the destruction of teeth and certain health disorders. The possibility of identifying and studying caries at its premorbid stage emerges, in the context of purely functional states that albeit are close to being systemic disorders, are not actual diseases yet.

The existence of fluctuations in the enamel's stability index allows us to see the initial stages of the process in a new light. By acknowledging its value, we receive an answer to the question of "when" caries begins: it begins when stability decreases. If the decrease is noticeable and long lasting, caries will occur in multiple teeth at once. A systemic pathology occurs – ***the carious disease***. The decrease in the enamel's stability is a common denominator in different cariogenic situations in both experimental and clinical environments. If the enamel's stability decreases during a certain disease or state, it will favour the development of caries, if the stability increases, the opposite will be true. The idea tying caries to general pathological states does not exactly hold water. Therefore, it is not the "bad" or "good" events taking place in the organism that lead to a "good" or "bad" state of the teeth. The occurrence of dental caries depends upon the pump's functional state and not upon the general health.

The connection to general state in different diseases proved to be rather ambiguous and sometimes even diametrically opposed. For example, children suffering from obesity have significantly a stronger enamel and healthier teeth than their peers. On the other hand, people affected by scoliosis have a less resistant enamel and are more prone to caries. A connection between the prognostic of myocardial infarction and the enamel's functional resistance was also discovered. A decrease in the said index could show an increased probability of an unfavourable resolve of a heart attack. It seems that the majority of general diseases are weakly or ambiguously tied to the enamel's acid and caries resistance respectively. Undeniably, there are countless unidentified links between the enamel's functional state and different diseases and their various stages.

The "caries-state of the organism" relationship remained unclear as long as the common denominator was unknown: the enamel's active resistance. Now we can assert that only the diseases affecting this index lead to caries. The underlying causes

behind the decrease in the enamel's resistance need further research. It is clear that this process is a result of some rhythmic internal biological circumstances . This is confirmed by the data found in scientific literature on the genetics of caries, especially in literature focusing on the study of twins.

The high value of the Holzinger inheritance index (up to 85%) for caries, clearly indicates towards its heredity, i.e. its endogenous predisposition. There is also some data on the inheritance of certain enamel stability index.

At the same time, it is clear that the endogenous predisposition of the disease could be tied not only to an individual's genetic program. One cannot doubt the role of seasonal biorhythms. Initiating in the hypothalamus, the hormonal signals called "releasing-factors" coordinate the activity of the parotid gland. The latter controls the activity of the odontoblasts and the tooth pump respectively through its hormone called "parotine". The seasonal drop in the activity of this endocrine regulated chain causes a decrease in acid resistance and eventually increases the sensibility to caries.

Caries and periodontitis, as a mass condition, are indeed the creation of society. The closer a population is to the western socio-economic lifestyle, the worse their dentitions are.

This pattern could be equally applied to the people living in different geographical zones over different historical periods. Which factors of developed civilisations favour the occurrence of caries are still a subject of dispute. Conventional wisdom blames the changes in our alimentary behaviour, especially the use of sugar and other easily digestible carbohydrates. However, such an explanation is contradicted by historical facts. The massive spread of caries in certain populations precedes the spread of carbohydrates and white flour by thousands of years.

It would seem that the gap between these two events naturally refuses the simplistic explanation offered by conventional wisdom. Much more credible is the fact that the precarious

state of the dentition of the civilised man is caused by general biological factors that influence the structure of many conditions as a whole, including the mass spreading of caries, periodontitis and bite anomalies. It is about a certain uncontrolled biological degradation of man, tied to his departure from his natural habitat (biological pressing). The latter is one of the main mechanisms of natural selection. It is also the biological pressing that engages an individual's complete array of functional systems to their highest degree. The elimination of biological pressing and the survival of every individual leads to the genetic destabilisation of the population. Thus, almost every subject growing up in an environment that does not require physical effort, becomes weak and "functionally immature".

The cause of "civilised" morbidity (including lifestyle diseases and especially dental diseases) is clear- the elimination of biological pressing. However, it is fairly obvious that a tentative to correct the current situation by bringing it back is absolutely unacceptable. We need a much more humane strategy. This strategy, while not being officially declared a pursuable research path, is continuously self-organising, being found in multiple "crystallisation centres". All the healing (paramedical) practices should be attributed to this strategy. Their existence and the fact that they are spreading by themselves, represent a scientific fact observed throughout the word.

Para medicine is not scientific, but it is often efficient. This situation should be understood. In order to do that, we could use a fairly effective instrument: the assumption that science has two poles- a physical and an informational one. Clearly, from this point of view, the brutal physical conditions that evolve into lesions occur as a result of the disparity between the organism's own physical capabilities and the intensity of the environment's phenomena. The occurrence of the disease in such conditions is always dictated by an almost linear dependence upon the intensity of the acting factors. The prevention of these

diseases is conceptually simple- optimise the environment. Its implementation, on the other hand, is very difficult. The same could be said about the treatment of the said conditions- it is clear what needs to be done, it is clear that it needs to be done separately in each case, but **how** to do it is often an unanswered question.

We have an absolutely opposite situation at the other pole. The occurrence of these diseases does not depend upon the physical nature of the information carrier, its physical intensity and the quantity of the pathogen. The only thing that matters in this case is the information itself, the meaning of the signal. We have no statistical dependence between the "physical" and the result. Therefore, by merely altering the environment's parameters, we will not prevent the pathological process. We have a similar situation in the case of treatment and prophylaxis: what should we correct if the etiology is unidentified, unclear or questionable. By accepting the platonic dichotomy- "fine and harsh" diseases- as an actual concept, we can overcome the actual limitations.

Firstly, we should admit that our conscience is dominated by the ideology of the physical, harsh pole. However, this does not coincide with the actual situation: the majority of our patients carry the "bad" factors within themselves, and not even the perfect environment will grant them ideal health. This also refers to dental diseases.

The "phantom of the fracture", tied to an archaic concept has deep roots in official medicine. It is firmly believed and affirmed that if there is a clinical case, then we will undeniably have some sort of hidden defect, a "hard" lesion, hidden at an organic, cellular or molecular level. Traditionally, the lesion is seen as the primary cause of any disorders, especially in the context of teeth, that are chemically destroyed by the products of microbial activity. The detection and mending of lesions is the hidden paradigm of medicine. In reality, the matters are not so

simple, there can be no lesions. We should agree with Plato, but at the same time we should know that not only "hard" defects could lead to pathology, but "fine" ones as well- the signals, i.e. the information that does not injure or bend anything, safe for its normal function. (A relevant example would be the modus operandi of a computer virus.)

An enormous role is played by signals during both the development of the organism and functional disorders. They operate thanks to their encoded "meaning" and are related neither to the energetic resources nor to the organism's "living bricks" (i.e. tissues), because these signals are merely guiding the course of events. We should emphasize the idea that the value of these signals is not tied to the physical properties of the signal carriers, which can vary from the biochemical properties of the DNA to the soundwaves of speech.

If we start perceiving these concepts as a real possibility, we come to the conclusion that medicine should acknowledge the specifics of "fine" relationships. The role of pathogenic and sanogenous information per se should be acknowledged and studied independently from the carrier's material properties.

The correction of the environment's parameters should be considered as one of the methods of health control, but not the only one. Its usefulness lies in the area of "harsh" diseases and its immediate proximity. If the outset and the quality of the harsh disease is directly related to the dosage of the effect (a lack or a surplus thereof), then the fine disease depends on the content of the information received by the organism and/or the individual. Here we have once again a situation analogous to computer science: in order for someone to get sick or healthy, that someone needs to know the respective programming language. However, unlike computers belonging to the same batch, humans are highly individual. They are characterised by a constitutional diversity of biochemical, physiological and spiritual nature. Thus, the optimisation of modern man's quality

of life and his release from under the heel of "fine" diseases are closely related to the knowledge and understanding of his individual traits. However complicated this route may seem, there is no alternative. It is clear that civilisation itself is at fault for the existence and spread of the diseases of civilisation and the fact that some are predisposed to caries while others to periodontal disease depends on their constitution.

After taking a long stroll through the issues troubling medicine, we are back to the perception of dental diseases formulated more than a century ago by the founder of theoretical dentistry-W. Miller. A human's individual traits and parameters are among the leading causes of caries and periodontal disease. The more accurately we will learn to identify their characteristics and the patient's medial constitution, the more successful we will be in counteracting these diseases.

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